123 Doc EDUCATION

FOUNDATION YEARS JOURNAL

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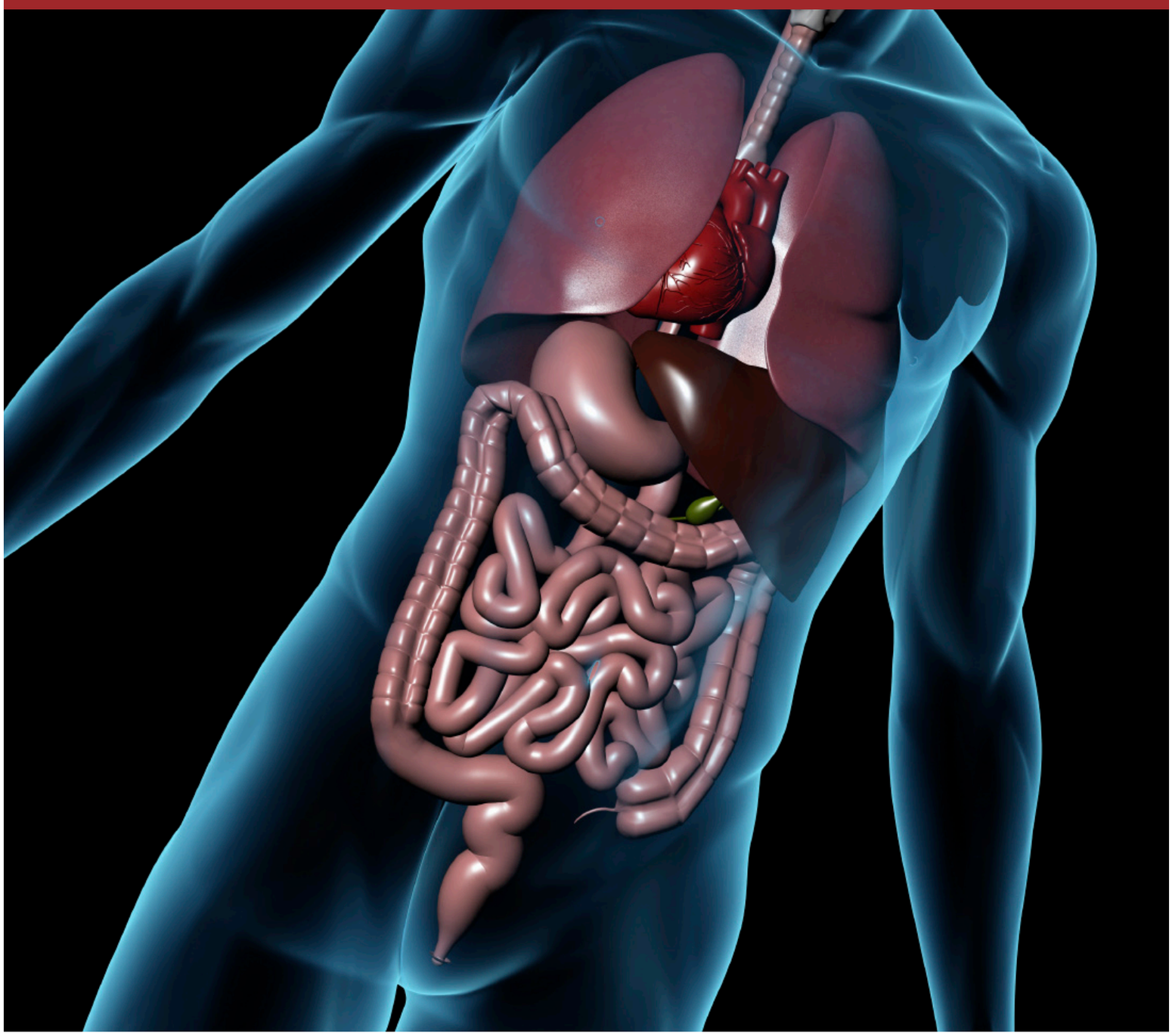
EDITOR IN CHIEF, MICHAEL VASSALLO

Volume 3, Issue 4: Gastroenterology



Contents

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FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 4

Foundation Years Journal

Foundation Years Journal is an international peer-viewed journal which seeks to be the pre-eminent journal in the field of patient safety and clinical practice for Foundation Years' doctors and educators. The Journal welcomes papers on any aspect of health care and medical education which will be of benefit to doctors in the Foundation training grade in the UK or international equivalents. The predominant emphasis in **Foundation Years Journal** is on work related to patient safety and in health care education.

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FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 4

Volume 3, Issue 4: Gastroenterology

Foundation Years Journal is the ONLY journal for Foundation Years doctors and educators, specifically written according to the MMC curriculum. It focuses on one or two medical specialties per month, each issue delivers practical and informative articles tailored to the needs of junior doctors. The Journal closely follows the Foundation Years syllabus to provide the best educational value for junior doctors. In addition to good clinical and acute care articles, assessment questions give junior doctors the chance to gauge their learning.The answers will be published in the next issue, but 123Doc will advance answers to clinical tutor subscribers so they can engage their students in the learning process. Each issue provides comprehensive clinical cases for trainees as well as practical teaching assessments for educators. Readers will benefit from:

- **MMC CURRICULAR-BASED CONTENT** to enhance understanding of the core competencies required from future leading doctors.
- **FOCUS ON SPECIALTY-SPECIFIC CLINICAL CASES** each month to form broad subject coverage.
- **ADDITIONAL IN-DEPTH** good clinical and acute care articles aligned with the case-based discussion assessments.
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Notice

FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 4

Aim and scope

The Foundation Years Journal is published by 123doc and is aimed at doctors in Foundation Training programmes, their educational and clinical supervisors, as well as medical students and other doctors (particularly international medical graduates) who intend to start Foundation training in the United Kingdom.

Journal sections

The Journal has been redesigned and various sections have been introduced to map the Journal more closely to the Foundation programme curriculum. You can view the curriculum from **http://www.foundationprogramme.nhs.uk/pages/home/training-and-assessment**.

The sections are the following:

1. Good Clinical Care (syllabus section 1)
This section deals with various aspects of patient management including history, examination, diagnosis, record keeping, safe prescribing and reflective practice. Articles could also refer to other aspects of care including time management, decision-making, patient safety, infection control, clinical governance, nutrition, health promotion, patient education, public health and ethical and legal issues.

2. Good Medical Practice (syllabus section 2)
Articles could be on learning, research, evidence-based guidelines and audit.

3. Training and Teaching (syllabus section 3)

4. Professionalism in Practice (syllabus sections 4,5 and 6)
This section includes relationship with patients, communication skills, working with colleagues, probity, professional behavior and personal health.

5. Patient Management (syllabus section 7)
Articles should be focused on the recognition and management of the acutely ill patients, core skills in relation to acute illness, resuscitation, management of the 'take', discharge planning, selection and interpretation of investigations.

6. Practical Procedures (syllabus section 8)

7. Test Yourself
The intention is to provide a vehicle whereby trainees and educational supervisors can present original and review articles mapped against the Foundation curriculum.

Submission of manuscript

All articles submitted to the Journal must comply with these instructions. Failure to do so will result in return of the manuscript and possible delay in publication.

Manuscripts must be submitted exclusively by email (see detailed instructions below). Manuscripts should be written in English of a sufficiently high standard that is intelligible to the professional reader who is not a specialist in the particular field. Where contributions are judged as acceptable for publication, the Editor or the Publisher reserve the right to modify the manuscripts to improve communication between author and reader. Authors whose native language is not English are strongly recommended to have their submissions checked by a person knowledgeable of the language. If extensive alterations are required, the manuscript will be returned to the author for revision.

Covering letter

The manuscript must be accompanied by a covering letter bearing the corresponding author's signature. Papers are accepted for publication in the Journal on the understanding that the content has not been published or is being considered for publication elsewhere. This must be stated in the covering letter. If authors submit manuscripts relating to original research in the field of education, the corresponding author must state that the protocol for the research project has been approved by a suitably constituted Ethics Committee and that it conforms to the provisions of the Declaration of Helsinki (as revised in Edinburgh 2000), available at **http://www.wma.net/e/policy/b3.htm**. All investigations involving human subjects must include a statement that the subject gave informed consent and patient anonymity should be preserved.

The covering letter must contain an acknowledgement that all authors have contributed significantly and that all authors are in agreement with the content of the manuscript.

Authors should declare any financial support or relationships that may give rise to a conflict of interest.

Submitting a manuscript

Manuscripts should be submitted by email to **(agnes@123doc.com)**. We do not accept manuscripts submitted by post. Corresponding authors must supply an email address as all correspondence will be by email. Authors should use double spacing when submitting their manuscript. Two files or documents should be supplied: the covering letter and manuscript. The covering letter should mention the title, authors, their contribution, provenance, journal section where their work is to be considered (see above) and any conflict of interests. Please supply the files in Word 2003 format.

Figures should be supplied as a separate file, with the figure number incorporated in the file name. High-resolution figures (at least 300 d.p.i.) saved as jpeg files should be submitted.

FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 4

Manuscript style

Unless otherwise stated manuscripts should follow the style of the Vancouver agreement detailed in the International Committee of Medical Journal Editors' revised "Uniform Requirements for Manuscripts Submitted to Biomedical Journals: Writing and Editing for Biomedical Publication", as presented at **http://www.ICMJE.org/**.

Abbreviations

Abbreviations should be used sparingly to facilitate reading the article by reducing repetition of long, technical terms. Initially you must use the word in full, followed by the abbreviation in parentheses. Thereafter use the abbreviation only.

Units

All measurements must be given in SI or SI-derived units.

Trade names

Drugs should be referred to by their generic names, rather than brand names.

References

All articles must be referenced appropriately. To reference the Journal please use the following abbreviation FYJ-123Doc. (The Vancouver system of referencing should be used and some examples are given below).

References should be cited using superscript Arabic numerals in the order in which they appear. If cited in tables or figure legends, number according to the first identification of the table or figure in the text.

In the reference list, the references should be numbered and listed in order of appearance in the text. Cite the names of all authors, when seven or more list the first three followed by et al. Names of journals should be abbreviated in the style used in Index Medicus, *and be in italic font*. Reference to unpublished data and personal communications should appear in the text only.

References should be listed in the following forms:

Journal article

Vassallo M, Vignaraja R, Sharma JC, et al. The Impact of Changing Practice on fall Prevention in a Rehabilitative Hospital. The Hospital Injury Prevention (HIP) Study. *J Am Geriatr Soc 2004,* 52:335-9. Book Azeem T, Vassallo M, SamaniNJ. *Rapid review of ECG interpretation.* London UK: Manson Publishing 2005.

Chapter in a book

Martin GM. Biological mechanisms of ageing. In: J Grimley Evans, T Franklin Williams (eds). *Oxford Textbook of Geriatric Medicine, 1st edn.* New York: Oxford University Press 1992, 41-48.

Journal article on the internet

British Geriatrics Society position paper. Dementia ethical issues **http://www.bgs.org.uk/Publications/Position%20Papers/psn_dementia_ethics.html**.

Tables

Tables should be self-contained and complement, but not duplicate, information contained in the text. Number tables consecutively in the text in Arabic numerals. Table should be double-spaced and vertical lines should not be used to separate columns. Column headings should be brief, with units of measurement in parentheses; all abbreviations must be defined in footnotes. Footnote symbols: †, ‡, §, should be used (in that order) and *, **, *** should be reserved for P-values. The table and its legend/footnotes should be understandable without reference to the text.

Line figures

Line figures should be sharp, black and white graphs or diagrams, drawn professionally or with a computer graphics package. Lettering must be included and should be sized to be no larger than the Journal text.

Colour figures

We encourage authors to submit colour figures and graphics that facilitate the comprehension of the article.

Figure Legends

Type figure legends on a separate page. Legends should be concise but comprehensive - the figure and its legend must be understandable without reference to the text. Include definitions of any symbols used and define/explain all abbreviations and units of measurement. The Journal accepts the following types of articles (as title please):

Case Based Discussion

These are mainly intended for inclusion in sections 1 and 5 as highlighted above and should be about 1000-1500 words long. The CBD can focus on various aspect of patient care such as presentation, treatment or prescribing. The articles should include areas that are evaluated in the case based discussion assessment tool of the foundation programme .

The manuscript should be set out in the following sections:

- Abstract: this should refer to salient points from the case being presented together with a mention of what aspects are being discussed.
- Case History: this relates to the initial presentation and should include the clinical setting, clinical problem, investigations and treatment. The history section should also include an ongoing update (e.g. 2 days later, a week later, etc.) of patient progress and management.
- Discussion: this section should include a critical analysis of patient management in relation to clinical assessment, investigations, differential diagnosis, treatment, follow-up, professionalism and clinical judgement. The discussion should also include a discussion about the ongoing management issues and decisions. It is important to note that the case based discussion is not a review of a particular condition.
- Two best of 5 MCQs to be included in the Test Yourself section, with answers and detailed teaching notes explaining the answers. The answers only are NOT sufficient and it should be kept in mind when writing the teaching notes that the reader may take the test questions independently from reading the article.

FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 4

Authors writing a case based discussion should not write a short history and then write an article about the condition that the patient presented with. Such information can easily be obtained from a text book and is not the scope of Journal. Case based discusions written in this style will be returned to the author without being published.

Practical Procedures

Manuscripts on practical procedures should be about 1000–1500 words long. They should be set out in the following sections:

- History: this should describe the presentation of the patient and mention why or how the patient ended up needing the procedure.
- The procedure itself.

This should include:

- indications and contraindications
- explaining the procedure to the patient (including possible complications) and gaining informed consent for procedures
- preparing the required equipment, including a sterile field
- position the patient and give pre-medication/sedation or local anaesthesia as required and involving the anaesthetist where appropriate
- safely disposing of equipment, including sharps
- documenting the procedure, including labelling samples and giving instructions for monitoring and aftercare
- recording complications and the emergency management of such complications when appropriate.

Adequate pictures and diagrams need to be supplied in order to make the procedure as clear as possible.

Two best of 5 MCQs for inclusion in the test yourself section, including answers and detailed teaching notes. The answers only are NOT sufficient and it should be kept in mind when writing the teaching notes that the reader may take the test questions independently from reading the article.

Audit

Manuscripts, 1500–2000 words long, on audit are encouraged. The Journal will only publish high quality audit i.e. completed audit cycles or audits that have led to guideline development. Part 1 audits or surveys will not be accepted for publication.

Review Articles

We are interested in review articles on any aspect of the curriculum that is of relevance to our readership. They should be a maximum 3000 words long, 30 references, 250 word structured abstract, 4 tables OR figures.

We would consider reviews on any of the following:

- Good Medical Practice
- Teaching and Training
- Professionalism
- Medical reviews subject to prior discussion with the editorial team as to the appropriateness of the article

Shorter Reflective Practice Articles

We are always pleased to receive short pieces of a thoughtful nature that describes the personal or professional experiences of colleagues working with patients or their relatives. They should have a maximum of 1000 words. As suggested in the Foundation Programme Portfolio (Reflective Practice) these articles should describe:

- What made the experience memorable?
- How did it affect you?
- How did it affect the patient?
- How did it affect the team?
- What did you learn from the experience and what if anything would you do differently next time?

Some aspects to be considered in these articles are:
Communication with the patient, ethical issues, aspect of your works with colleagues, probity and honesty, personal health.

Research Papers

The Foundation Years Journal would welcome research articles on Medical Education. Other research papers would be considered if thought to be of interest to the readership of the Journal. Articles should be written using the following headings (title page, abstract, introduction, methods, results, discussion acknowledgements, references, tables, illustrations legends.). They should be of a maximum of 2500 words of text, plus abstract, 30 references, 3 tables or figures. Manuscripts including a structured abstracts should have a maximum of 250 words using the headings introduction, methods, results, conclusion. The title page should contain (i) the title of the paper; (ii) the full names of the authors; and (iii) the addresses of the institutions at which the work was carried out together with; (iv) the full postal and email address, plus facsimile and telephone numbers, of the author to whom correspondence about the manuscript should be sent.

FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 4

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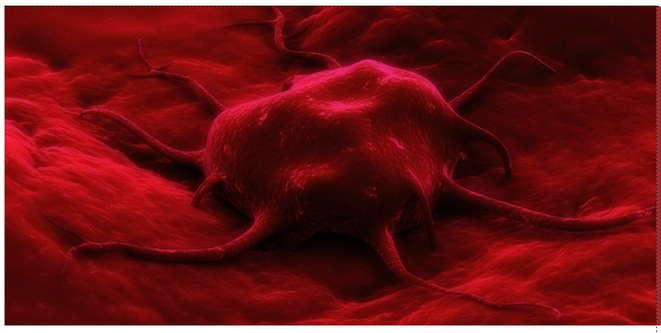
UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild



Oesophageal and gastric cancer are among the leading causes of cancer death in the United Kingdom (UK). Cancer cell shown left. Patient Management.

Relevance to the Curriculum

1.0 Good clinical care
1.1 History, examination, diagnosis, record keeping, safe prescribing and reflective practice
1.5 Clinical governance
1.7 Health promotion, patient education and public health

2.0 Maintaining good medical practice
2.1 Learning
2.2 Research, evidence and guidelines

7.0 Recognition and management of the acutely ill
7.1 Core skills in relation to acute illness
7.5 Selection and interpretation of investigations

Abstract

Oesophageal and gastric cancer are among the leading causes of cancer death in the United Kingdom (UK). This article provides an insight into the epidemiology, presentation, diagnosis and management of these conditions. It focuses specifically on the most common histological types of upper gastrointestinal malignancies, those of oesophageal squamous cell carcinoma and oesophageal adenocarcinoma and gastric adenocarcinoma.

Keywords

Oesophageal squamous cell carcinoma; oesophageal adenocarcinoma; gastric adenocarcinoma; upper gastrointestinal cancer.

Case history 1

A 75-year-old patient presented a 5-week history of dysphagia, which had progressed to dysphagia for liquids in the 2 days prior to his admission. Past medical history included previous myocardial infarction and atrial fibrillation, and a 20-pack year smoking history. He had suffered intermittent symptoms of gastro-oesophageal reflux for many years, but his usual antacids had not helped these symptoms over the preceding few weeks.

Physical examination was unremarkable and serum blood tests were within normal limits.

An urgent gastroscopy was arranged which revealed a malignant appearing stricture, the upper limit of which was at 37cm from the incisors. Multiple biopsies were taken from this area, but due to the degree of stenosis present it was not possible for the endoscope to be passed more distally into the stomach. A nasogastric feeding tube was therefore passed using a guidewire to permit enteral feeding while the results of the biopsies were awaited.

Q. What are the main histological subtypes of oesophageal cancer?

Two main histological subtypes of oesophageal cancer are recognised, and account for 95% of all oesophageal malignancies: squamous cell carcinoma (SCC) and adenocarcinoma (AC). SCC arises from squamous epithelial cells in the upper two thirds of the oesophagus, while AC generally develops more distally, in columnar epithelium, either from the cardia or within Barrett's oesophagus. Biopsy results from the tumour revealed adenocarcinoma.

Q. What do you know of the epidemiology of oesophageal cancer?

In the UK, oesophageal cancer is the 9th most common malignancy, but the 5th most common cause of cancer mortality, accounting for about 5% of all UK cancer deaths annually. It affects men more than women, with a male:female ratio of 3–4:1, and develops more commonly with advancing age. Age standardised incidence ratios per 100,000 population are 8.4 for men and 3.5 for women in the UK. However, it should be noted that incidence rates vary widely, not only between countries, but also between sexes and ethnic groups, and similar global discrepancies are also observed for mortality. Compared to the rest of Europe for example, the UK incidence rates of oesophageal cancer among UK males are second only to those reported in France, and among UK women are higher than in any other EU country. However, the incidence of oesophageal cancer in the UK is dwarfed when compared to parts of China, where rates of 184 and 123 per 100,000 population, for men and women, respectively, are reported.

On a global scale, the incidence of oesophageal cancer has risen in recent decades, but the demographics relating to histological type and tumour site are changing. Although on a global scale the majority (80–85%) of oesophageal tumours are SCC, in Western countries there has been a decline in the number of new cases of SCC and a rapid rise in the number of oesophageal AC diagnosed, particularly among the white male population, such that now in the UK and US approximately 50% of new diagnoses are AC. The reasons for this are not clear, but may be related to the Western diet causing an increased incidence of chronic gastro-oesophageal reflux. This is thought to be the predominant cause for Barrett's oesophagus, which is recognised as the precursor for development of AC, which is found in the distal, rather than proximal oesophagus.

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

Worryingly, the rise in cancer incidence has been almost matched by a rise in mortality from the disease. With few major advances in treatment, and given that 50–60% of patients are found to have either locally advanced or metastatic disease at diagnosis, this renders oesophageal cancer one of the deadliest solid tumours in oncology, with UK 5-year survival rates post diagnosis of only 8% for both men and women in 2001.

In addition to his worsening dyspeptic symptoms and dysphagia, the patient also complained of a persistent, dull retrosternal ache which had increased in severity over several days.

Q. What symptoms do patients with oesophageal cancer commonly present with, and what might the significance of the retrosternal pain be?

The most common presenting symptom of oesophageal cancer is dysphagia. This can be intermittent at first and is usually for solids, but later dysphagia for liquids may develop. Odynophagia (painful swallowing) usually indicates the presence of a large tumour high in the oesophagus. Persistent retrosternal pain, hiccoughs and hoarseness of the voice may also occur, and are generally ominous signs indicating mediastinal and diaphragmatic invasion, and involvement of the recurrent laryngeal nerve, respectively. Weight loss is a common presenting symptom and is associated with a poor prognosis. Rarely, patients may present with symptoms related to direct invasion into mediastinal structures, such as cough or pneumonia secondary to a tracheo-oesophageal fistula, or with massive haematemesis from invasion of the aorta.

The patient was understandably distressed when he was informed of the diagnosis and asked what the likely cause of the condition was.

Q. What risk factors do you know for the development of oesophageal cancer?

The precise aetiology of oesophageal cancer is unknown. Epidemiological studies have identified many factors thought to increase the risk of developing cancer (probably via a process of persistent oesophageal 'irritation'), and given the wide geographical variations observed in incidence some investigators propose genetic factors may also be at play. The most commonly cited factors relate primarily to the risk of developing SCC, except for Barrett's oesophagus which is a precursor for AC:

- **Age** – risk of oesophageal cancer rises with age.
- **Diet** – a diet high in fat content and low in fruit, vegetables and vitamins is thought to increase risk.
- **Tobacco and alcohol** – Both tobacco and alcohol are major independent risk factors for oesophageal cancer, each increasing the risk by up to five times. The effects of both are multiplicative, rather than additive, increasing the risk by 25–100 times. The effect of tobacco on the risk of oesophageal cancer is directly related to the total amount smoked (or chewed!). Risk falls to levels seen for non-smokers 10 years after cessation of smoking.
- **Barrett's oesophagus** – longstanding acid reflux and resulting oesophagitis can result in a change (metaplasia) of cell type from the stratified squamous epithelium lining the lower oesophagus to columnar (gastric-type) epithelium. First described by Norman Barrett in 1957, this is though to be a pre-malignant condition, increasing the risk of oeosphageal AC by 30–40 times. Barrett's oesophagus is found in 10% of patients who undergo investigation for symptoms of reflux, and confers a risk of developing AC of approximately 1% per year.
- **Caustic injury** – damage caused by ingestion of chemical irritants (classically sodium or potassium hydroxide solutions) can increase the risk of developing oesophageal cancer in later life.
- **Achalasia** – a definite increased risk of oesophageal carcinoma is seen in patients with achalasia, but the magnitude of this risk is difficult to quantify, with relative risk varying from 6 to 140 times in the literature.
- **Familial tylosis** – also known as Howell-Evans syndrome, tylosis is an exceptionally rare autosomal dominant syndrome in which hyperkeratosis of the palms of the hands and soles of the feet is associated with an increased risk of developing oeosphageal cancer. Regular endoscopic screening is recommended in this condition.

It was explained to the patient that further tests would be necessary to determine the treatment options available to him.

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

Q. What further investigations might be indicated in this instance?

Diagnostic

• Endoscopy

- first line investigation
- permits direct visualisation of the tumour, an assessment of its location and size, and the degree of luminal obstruction present
- biopsies or brushings of the lesion may be taken to facilitate tissue diagnosis

• Barium or gastrografin swallow

- also a useful first line investigation, especially in patients suspected of having a lesion high in the oesophagus (when the risk of oesophageal perforation during 'blind' endoscopy is higher)
- can be used to delineate lesions where the degree of luminal obstruction is too great to permit the passage of an endoscope.

Staging

• Computed tomography (CT) scan

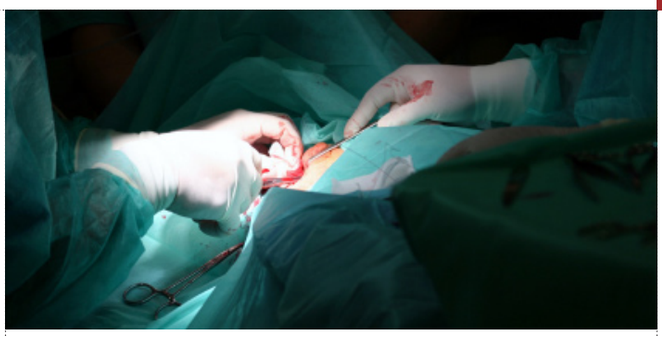
- abdominal and chest CT scans are most useful in assessing the N and M stages of the disease
- CT has a very high sensitivity for detecting distant metastases, which are most commonly found in the liver and lungs

• Endoscopic ultrasound (EUS)

- EUS is indicated in patients in whom a CT scan has excluded the presence of T4 or M1 disease
- has greater diagnostic accuracy than CT in assessing the depth of mural invasion (85% accurate) and involvement of regional lymph nodes (75–80% accurate)
- has greater accuracy in correctly staging T3 and T4 tumours than T1 or T2 tumours
- however, up to 30% of tumours are impassable using the EUS probe and therefore cannot be reliably assessed. In addition, the risk of perforation is significantly higher than for a routine diagnostic endoscopy (up to 5% in some series)

• Positron emission tomography (PET) scan

- fluorine 18-labelled fluorodeoxyglucose (FDG) PET scanning is being increasingly used in conjunction with CT in staging oesophageal cancer
- 'PET/CT' is of limited utility in both T staging and the detection of locoregional lymph node involvement, as the limited spatial resolution of PET makes it hard to differentiate uptake of FDG into the tumour from uptake within adjacent nodes
- its main use is in the detection of metastases which conventional CT and other staging investigations might not have identified, thus avoiding unnecessary surgical intervention
- PET/CT also occasionally identifies unsuspected synchronous tumours, which can occur in 1.5–5.5% of patients diagnosed with oesophageal cancer at first presentation



• Laparoscopy

- laparoscopy is a highly accurate technique used to stage regional lymph nodes
- although it is being superseded somewhat by EUS, it is still useful in cases where it is not possible to pass the EUS transducer through an obstructing tumour

Other Investigations

• CXR

- may detect the presence of pulmonary metastases, and can be used to assess suspected cases of oesophageal perforation

• Bronchoscopy

- this is useful for oesophageal lesions above the level of the carina to evaluate for evidence of tracheobronchial invasion.

Unfortunately a staging CT scan revealed the presence of liver metastases. EUS and PET scan were therefore not performed. At the weekly upper GI cancer multidisciplinary meeting, the consultant oncologist mentions the term 'T4 M1 tumour' when discussing the case.

Q. What does the term 'T4 M1 tumour' mean, and why is the staging of oesophageal tumours important?

The TNM classification is used in staging oesophageal cancer, and is used not only to inform treatment decisions, but also to provide information about prognosis and survival. It is based on information about the depth of tumour invasion into the oesophageal wall (T), the involvement of lymph nodes (N) and the presence of distant metastases (M). This information is obtained from the results of the endoscopic and radiological investigations outlined in the previous section. However, to fully appreciate this system, it might first be useful to briefly revise the histological anatomy of the oesophagus.

Oesophageal anatomy

The oesophagus is essentially a muscular tube, which extends from the pharynx to the stomach. Anatomically it comprises three parts; the cervical (2–3cm long, extending from the proximal oesophageal limit to the thoracic inlet), thoracic (21cm long, extending from the thoracic inlet to the oesophageal hiatus in the diaphragm), and abdominal sections (1–1.5cm long, extending from the oesophageal hiatus to the right side of the stomach).

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

In cross section, four distinct histological layers are identified:

- **Mucosa**
  - comprises a non-keratinised stratified squamous epithelial cell layer, the lamina propria and a layer of smooth muscle known as the muscularis mucosae
- **Submucosa**
  - a loose connective tissue layer rich in capillaries and lymphatics which also contains mucous secreting glands
- **Muscularis propria (also called muscularis externa)**
  - a muscle layer, comprising an inner circular layer and outer longitudinal layer, the composition of which varies according to the anatomical level
- **Adventitia**
  - a 'loose' fatty layer, not strictly a serosa

Using this anatomical knowledge, the T stage of the tumour can be assessed. This is illustrated in Figure 1, and the full TNM staging system is presented in Figure 2.

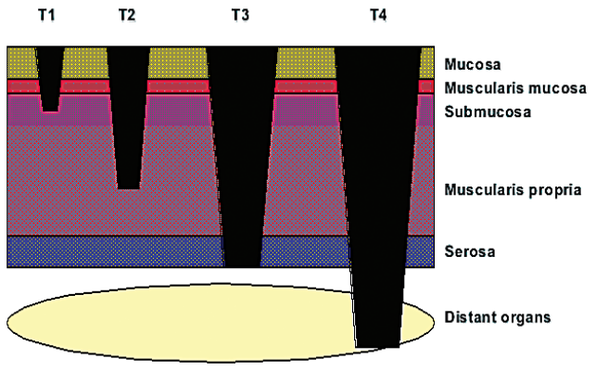


Figure 1: T staging of oesophageal tumours.

The T stage	
Tx	Primary tumour cannot be assessed
T0	No evidence of primary tumour
T1	Tumour invades mucosa and/or submucosa
T2	Tumour invades muscularis propria
T3	Tumour invades the adventitia
T4	Tumour invades local structures
The N stage	
Nx	Lymph node involvement cannot be assessed
N0	No evidence of lymph node involvement
N1	Evidence of locoregional lymph node involvement
The M stage	
Mx	Distant metastases cannot be assessed
M0	No evidence of distant metastases
M1	Evidence of distant metastases (see note below)

It is worth noting that the N1 stage refers only to locoregional lymph node involvement – these are the sites of primary lymphatic drainage from the oesophagus and are usually resected at the time of surgery – which lymph nodes groups are classified as locoregional depends on the site of the primary oesophageal tumour. Non-regional lymph nodes containing tumour are designated M1a for the purpose of staging, while M1b refers to distant organ metastases. Prognostically, patients with M1a disease have worse outcome than those with N1 disease, but better long-term survival than those with M1b disease.

Figure 2: TNM classification for oesophageal cancer.

The patient was informed that in view of the presence of distant metastases that surgery was not a treatment option, but that further therapy would be directed towards alleviating his symptoms. His wife asked if chemotherapy would help.

Q. What are the treatment options available for oesophageal cancer? Does chemotherapy have a role to play in treatment?

SURGICAL

Primary resection of oesophageal cancer is indicated for patients with no evidence of distant metastasis. Two types of procedure are commonly performed, the Ivor-Lewis oesophagectomy and the transhiatal oesophagectomy. There is currently no consensus about which approach is superior. Operative mortality is approximately 10%, but rates as low as 5% have been reported from specialist centres, and both procedures carry significant risk of morbidity related to anastomotic leakage, pulmonary complications and cardiac events. In those who are deemed surgically fit, no difference in outcome has been demonstrated between younger and older (>70 years) patients[1], and age alone should not be used as a determinant for operative suitability.

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

NON-SURGICAL

Radiotherapy

Radiotherapy as monotherapy is rarely used with curative intent. Although previous studies suggested neoadjuvant radiotherapy might increase the rate of surgical resectability, meta-analysis did not demonstrate an overall statistically significant survival benefit[2]. However, when used for palliation, it can provide relief of dysphagia in up to 50% of patients treated.

Chemotherapy

Randomised controlled trials examining the effects of perioperative, neoadjuvant and adjuvant chemotherapy on patient survival in oesophageal cancer have produced conflicting results. Recent meta-analysis suggests an improved 3-year survival in patients with advanced localised oesophageal carcinoma treated with adjuvant chemotherapy after radical surgery versus those treated with surgery alone[3]. However, to date no conclusive evidence exists to support the use of chemotherapy alone as a treatment modality for patients with potentially resectable disease.

Chemo-radiotherapy (CRT)

Research has more recently focused on a combined approach for patients with resectable disease, using neoadjuvant CRT followed by surgical resection. Meta-analysis examining this approach found a 13% 2-year survival benefit for patients with both oesophageal SCC and AC treated with neoadjuvant CRT followed by surgery versus those treated with surgery alone[4]. The same analysis also indicated a 7% 2-year survival benefit in those treated with neoadjuvant chemotherapy versus those treated with surgery alone, although this effect was not significant. Although this approach can result in a significant increase in post-operative mortality (number needed to harm=25), this is offset by the positive effects seen on patient outcome (number needed to treat=10)[5]. It is also worth noting that in patients with potentially resectable disease who are unfit or unsuitable for surgery, CRT has been noted to produce a significant survival advantage over radiotherapy alone (5-year survival rates 26% versus 0%)[6].

Endoscopy

As lymph node or distant metastasis rarely occurs in very early (stage 0 or I) cancer, local therapies have been developed for use in these patients. Endoscopic mucosal resection (EMR) is one technique that can remove pieces of mucosa up to 1cm in diameter and to a depth reaching the deep submucosa. In early cancer associated with Barrett's oesophagus, EMR lead to remission in 97% of cases[7]. However, studies suggest a 1-year recurrence rate of approximately 10%, with 5-year survival rates of 77% (compared to 85% for surgical intervention for tumours of the same stage)[8].

PALLIATIVE

In many instances, palliation involves directing therapy towards the relief of dysphagia. Self-expanding metal stents (SEMS) have been developed for the task, that can be deployed either endoscopically or fluoroscopically, and which then expand to full diameter over a period of a few days (Figure 3). They are able to be successfully positioned in over 90% of cases, but stent placement for very proximal tumours is problematic, as it can lead to a persistent 'foreign body' sensation and problems with airway compromise. Complications arising from stents are common, and include chest pain, bleeding, fistulation (into the trachea or bronchus) and stent migration. The mortality risk associated with SEMS placement, often due to oesophageal perforation, is not insignificant at 1–2%. The recent development of removable SEMS has enabled temporary placement for alleviation of dysphagic symptoms while CRT is undertaken.

Other techniques aimed at alleviating dysphagia by reducing tumour bulk have been tried, and include Nd:YAG laser therapy, photodynamic therapy (PDT), electrocautery and argon plasma coagulation. However, none of these modalities has been shown in trials to be superior to a metal stent; their availability is often limited; and their effects are often very short lived.

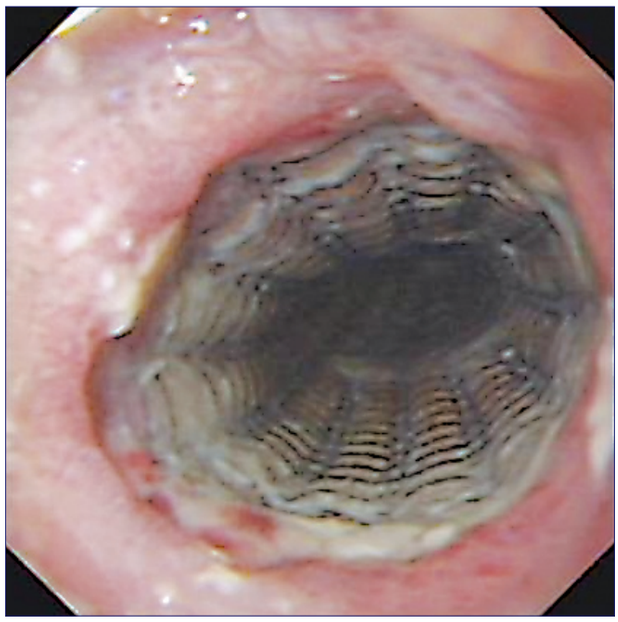


Figure 3: Self-expanding metal stent (SEMS) in situ in the oesophagus.

Placement of a SEMS provided good symptomatic relief, allowing him to tolerate a soft diet and liquids. Prior to discharge, the patient's daughter (having just flown in to the UK from America) asked what his likely prognosis might be.

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

Q. Is it possible to provide survival estimates based on the stage of disease, and if so, what might this be in this case?

The prognosis for oesophageal cancer, in comparison to other solid tumours, is overwhelmingly poor. Overall survival estimates are best made using staging information. The TNM classification is used to determine the clinical stage. A summary of current recommendations for oesophageal cancer therapy and survival times according to disease stage, is shown in Figure 4.

Stage	TNM Staging	Therapy	5-year survival
0	Tis, N0, M0	Surgery endoscopic therapy	75%
I	T1, N0, M0	Surgery CRT+/- subsequent surgery endoscopic therapy	50%
IIA	T2, N0, M0 or T3, N0, M0	Surgery CRT+/- subsequent surgery	40%
IIB	T1, N1, M0 or T2, N1, M0	Surgery CRT+/- subsequent surgery	20%
III	T3, N1, M0 or T4, any N, M0	Surgery CRT+/- subsequent surgery	15%
IV	Any T, any N, M1a or M1b	Palliation	<1%

Figure 4: Therapy and prognosis for oesophageal cancer according to stage of disease.

Case history 2

A 65-year-old patient presented with a 1-month history of persistent nausea, and had vomited on two occasions in the previous week. He complained of increasing lethargy and noted he was unable to manage a flight of stairs without becoming breathless. His wife commented that although he had always been thin, she thought he had lost some weight recently.

At rest he appeared pale and cachectic. Abdominal examination revealed epigastric fullness and the presence of shifting dullness. A laparotomy scar and an enlarged periumbilical nodule were also noted.

Serum blood test revealed a microcytic anaemia and mildly deranged liver function tests.

An urgent CT scan of the abdomen confirmed the presence of ascites, and noted thickening of the wall of the distal stomach and peritoneum. Hepatic metastases were also present. A gastroscopy was arranged which revealed the presence of a large tumour in the gastric antrum, biopsies from which confirmed gastric adenocarcinoma.

The remainder of this article will focus on gastric adenocarcinoma, which accounts for >95% of all gastric neoplasms. Other tumours, not detailed below, include gastric lymphoma, gastric carcinoid and gastrointestinal stromal tumours (GISTs).

Q. What do you know about the epidemiology of gastric cancer?

Gastric adenocarcinoma (GA) has for decades remained one of the leading causes of cancer mortality worldwide. Similar to oesophageal cancer, there is tremendous worldwide variability in incidence, with the greatest rates seen in the Far East where Japan ranks highest in the world. In the UK, it is the 6th most common malignancy in men and the 11th most common malignancy in women. The male:female ratio is 1–1.8:1. Greater than 90% of cases are diagnosed in patients over the age of 55 and rates rise progressively with increasing age. Fortunately, over the past 30 years in the UK, the incidence has more than halved, and the mortality decreased by over 70% in both males and females, such that the age standardised mortality rate had fallen to 5.8 per 100,000 in 2006. Despite this, it is the 7th most common cause of cancer death in the UK.

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

Worldwide, similar rates of decline in both incidence and mortality have been observed, but these have occurred to varying degrees and at differing times between countries for reasons that are not entirely clear. These are not thought to be due to any significant improvements in either diagnosis or treatment, but may be related to modifications in exposure to risk factors.

In contrast, rates of cancer occurring at the gastric cardia have been observed to increase in several countries (including the UK) over recent years. Although this has not offset the overall decline in gastric cancer incidence, it has redefined the anatomical location of tumour preponderance. Whereas most tumours were previously located distally, today only 30% are found in the antrum, 30% are found in the body and 40% are located in the fundus and cardia.

It is important at this point to note also the distinction of two subtypes of GA, which differ in their epidemiological and pathological features. The description above refers to the intestinal form (type I) of tumour, characterised histologically by gland-like tubular structures which mimic intestinal glands, and which tend to be found more distally. This is proposed to develop in a multi-step carcinoma 'sequence', similar to colon cancer, in which normal gastric mucosa transforms, under the influence of a variety of risk factors (described below) and genetic mutations, first to an abnormal hyperproliferative epithelium, then adenoma and eventually carcinoma. In contrast, the diffuse form (type II) is histologically poorly differentiated, lacking any glandular structure, occurs throughout the world at equal frequency, affects individuals at a younger age and tends to occur anatomically more proximally. It is not known whether this type follows a similar sequence of progression.

The patient revealed he had undergone an operation for a bleeding gastric ulcer some 30 years previously.

Q. What are the risk factors for gastric cancer? Is his previous operation relevant to his current presentation?

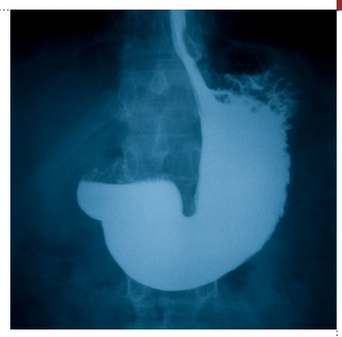
The factors most commonly cited for increasing the risk of developing gastric adenocarcinoma are:

Diet

· High intake of fruits and raw vegetables has consistently been shown to reduce the risk (by as much as 50%), while diets rich in highly preserved foods (containing high levels of salt, nitrates and aromatic amines) correlate with an increased risk.

Smoking

· Smoking increases the risk in a dose-dependent manner. Meta-analysis suggests an overall 1.6 fold increased risk, which is greatest in males.



Helicobacter pylori and atrophic gastritis

· Infection with *H.pylori* invariably results in inflammation within the stomach and a condition known as chronic active gastritis.
· In a subset of these patients, this is associated with a high gastric acid output and the development of duodenal ulcer disease, which may be protective against GA.
· However, in other (genetically predisposed) individuals, chronic *H.pylori* infection results in loss of specialised glandular tissue and the development of chronic atrophic gastritis, at a rate of 1–3% per year.
· Atrophic gastritis arising in this manner is associated with a 6 fold increase in the risk of GA.
· A second type of atrophic gastritis, most commonly associated with antiparietal cell and anti-intrinsic factor antibodies in pernicious anaemia, results in diffuse atrophy of parietal cells predominantly in the body and fundus and a lesser, albeit still increased, risk of developing GA.

Previous gastric surgery

· Patients having undergone gastrectomy for benign disease are at increased risk for developing GA, usually at the anastomotic site, starting 20 years after the initial surgery.
· The risk is 4 fold higher for Billroth II than for Billroth I operations, which may suggest bile reflux into the stomach remnant as an aetiological factor.

Genetic predisposition

· Up to 10% of cases of GA are familial in origin.
· To date, numerous genetic mutations thought to be important in the pathogenesis of gastric cancer have been identified in both sporadic and familial tumours, but the relative importance of each in the carcinoma sequence remains to be determined.

Menetrier's disease

· This rare condition, in which hypertrophy of surface mucous cells and atrophy of parietal and chief cells of the stomach results in a thickened fundal mucosa, a protein losing enteropathy and hypochlorhydria, is associated with GA.

When the patient was informed of the diagnosis of advanced gastric cancer, he was perplexed that he had suffered no symptoms until the few weeks just prior to his admission.

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

Q. In view of the advanced stage of the disease, is his presentation unusual? What other symptoms can be caused by gastric cancer, and what signs might be present on physical examination?

Symptoms

Unfortunately, many patients remain asymptomatic until very late in the disease course, when symptoms result from either local effects of a very advanced tumour (Figure 5), or from the presence of distant metastases. Symptoms of more advanced disease may result from increasing tumour size or tumour ulceration, and include persistent nausea and vomiting, abdominal pain, anorexia, weight loss and haematemesis. Some patients present with anaemia. Early satiety and dysphagia may result from obstruction of the pylorus and the cardia by tumour, respectively. Stercoraceous vomiting may occur via a gastro-colic fistula when an advanced tumour invades the adjacent colon.

Signs

Physical examination can be unremarkable. Signs of weight loss and cachexia may be obvious in more advanced disease. A distended abdomen with an audible succussion splash may be present in cases of gastric outlet obstruction. Occasionally, an enlarged stomach or ascites may also be evident. An enlarged, 'knobbly' liver may be felt with hepatic metastases, and periumbilical deposits are occasionally present (Sister Mary Joseph nodule), usually in association with multiple peritoneal metastases. Pathologically enlarged lymph nodes have gained various eponyms: Virchow's node (left supraclavicular) and Irish's node (anterior axillary). Rarely, paraneoplastic syndromes manifest as neuropathy, thrombophlebitis migrans (Trousseau's sign of malignancy), and disseminated intravascular coagulation. Dermatological manifestations include acanthosis nigricans (velvety hyperpigmentation of the skin in the axilla), dermatomyositis and the Leser-Trelat sign (explosive seborrhoeic dermatoses).

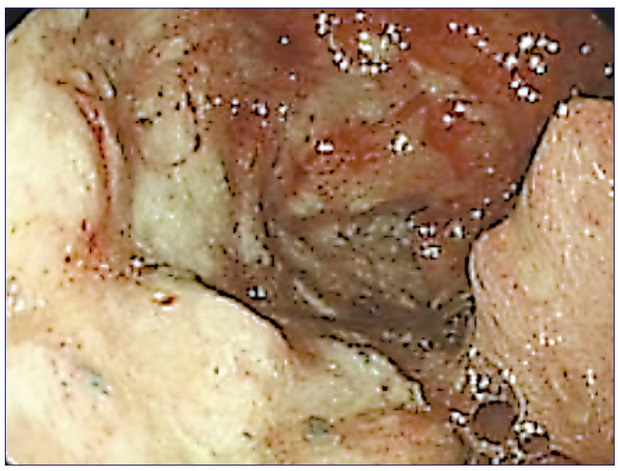


Figure 5: Photograph of an advanced gastric carcinoma.

Q. What investigations are useful in the diagnosis and staging of gastric cancer?

Diagnostic

• Bloods
- FBC to look for anaemia
- LFTs may be abnormal in the presence of hepatic metastases
- CEA and CA19-9 may be elevated in a proportion of cases but are not specific

• Endoscopy
- first line investigation
- 95% sensitive for advanced lesions; 50–60% sensitive for early cancers (which may be easily missed)
- allows anatomical site of tumour to be assessed and biopsies to be taken for tissue diagnosis

• Barium studies
- double contrast studies are 60–70% sensitive and 90% specific for advanced gastric cancer
- poor at detecting early gastric cancer; can be difficult to distinguish between benign and malignant ulcer disease
- should be reserved for cases when large proximal gastric cancers prevent passage of the endoscope into the stomach, and for patients who either refuse or who are unfit for endoscopy
- findings such as an asymmetrical ulcer base, irregular mass or gastric folds, and poor gastric distensibility suggest the presence of malignancy

Staging

• Computed tomography (CT) scan
- CT scanning of thorax, abdomen and pelvis is used primarily for detecting distant metastases, which are most commonly found in the liver
- it can reliably identify enlarged lymph nodes, but cannot distinguish whether this enlargement is due to tumour or reactive change. Similarly, it cannot identify tumour within normal sized lymph nodes
- overall accuracies are 60–70% for T staging, and 40–70% for N staging

• Endoscopic ultrasound (EUS)
- EUS permits excellent visualisation of the five layers of the stomach wall (see Figure 6)
- importantly, it is 90–99% accurate in distinguishing between T1 (early gastric cancer) and T2 (advanced gastric cancer) lesions
- overall, it is up to 90% accurate in T staging and 60–85% accurate for N staging

• Laparoscopy
- may play a vital role in staging patients being considered for surgery, especially when it is not clear whether the tumour involves the full thickness of the gastric wall

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

Other Investigations

- **CXR**
- **Magnetic resonance imaging (MRI)**

- is rarely used routinely as studies have not shown it to be superior to CT in N staging or EUS in T staging

- **Positron emission tomography (PET)**

- its role in staging gastric cancer is currently limited

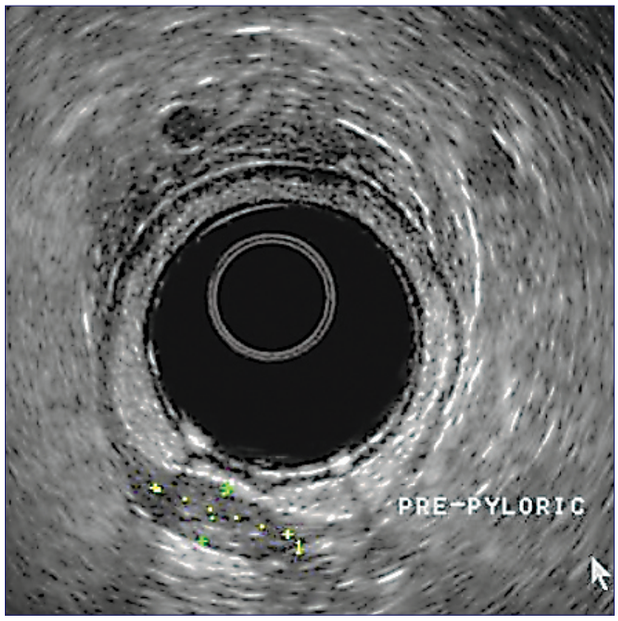


Figure 6: Radial endoscopic ultrasound (EUS) of gastric antrum.

The stomach wall appears sonographically as five distinct alternating hyperechoic and hypoechoic bands, which correspond to the histological layers. The first, innermost layer is hyperechoic (white) and corresponds to the superficial mucosa, while the second layer is hypoechoic (dark) and represents the deep mucosa. The third layer is bright and corresponds to the submucosa. The fourth layer is the hypoechoic muscularis mucosa, and the fifth layer appears white and corresponds to the serosa (which is the equivalent of the adventitia layer of the oesophagus). In this image, an abnormal widening of the muscularis layer can be seen inferiorly (as marked) which was caused by the presence of a GIST.

The patient asked if any further investigations were needed, but was reassured by the consultant that none were necessary as the stage of disease had already been determined by the CT scan.

Q. What staging system is used for gastric cancer?

The staging of gastric cancer is similar to oesophageal cancer, and involves a modified TNM classification system based on information about the primary tumour's depth of invasion into the stomach wall, involvement of lymph nodes and the presence of distant metastases (Figure 7). Previously, the N stage was formulated according to the anatomical site of nodal involvement: N1 denoted perigastric lymph node involvement within 3cm of the primary tumour; N2 denoted regional (left gastric, common hepatic, splenic and coeliac) lymph node involvement more than 3cm from the primary tumour; and N3 denoted more distant nodes. The present system used for N staging examines only the number of regional nodes involved, as it was shown to correlate more accurately with clinical outcome and be a better prognostic indicator than the previous system. In general, the greater the number of lymph nodes involved, the higher the likelihood of recurrence after therapy and the poorer the survival.

The T stage	
Tx	T stage cannot be assessed
T0	No evidence of primary tumour
T1	Tumour invades mucosa and/or submucosa
T2	Tumour invades muscularis propria
T3	Tumour invades the serosa
T4	Tumour invades local structures
The N stage	
Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastases
N1	Metastasis in 1–6 regional lymph nodes
N2	Metastasis in 7–15 regional lymph nodes
N3	Metastasis in >15 regional lymph nodes
The M stage	
Mx	Distant metastases cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Figure 7. TMN classification for gastric cancer

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

During his in-patient stay, the patient developed intractable vomiting and severe abdominal pain. The SpR requested a surgical review.

Q. Is there a role for surgical intervention at this point?

Palliative gastrectomy can be performed to alleviate symptoms, such as pain and haematemesis, even in patients with evidence of distant metastases. Gastric bypass surgery (gastroenterostomy) may be undertaken for symptoms due to gastric outlet obstruction.

Q. What other treatment options are currently available for gastric cancer?

SURGICAL

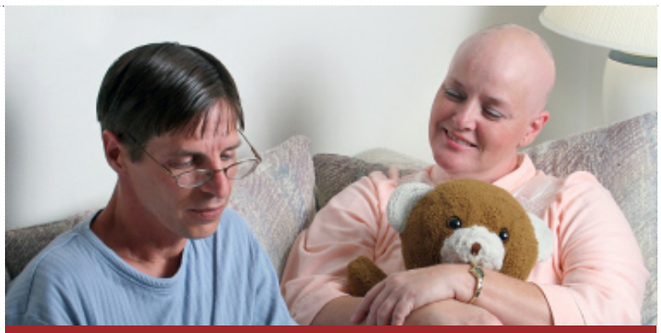
Surgery offers the only potential cure for gastric cancer. Unfortunately, 40% of those operated on develop a recurrence within 5 years (40% locoregional, 60% distant)[9]. In addition, owing to its often late presentation, 75–80% of patients are found to have inoperable or metastatic disease at diagnosis and are therefore unsuitable for operation with curative intent. However, many of these patients may still come to surgery during the course of their disease, as the role of surgery in palliation, especially for patients with symptoms of obstruction, is unquestionable.

Regarding curative surgery, two main controversies exist:

1. The extent of surgery needed to effect maximum chance of cure.
Studies addressing this question have compared subtotal gastrectomy with total gastrectomy outcomes, and found no difference in 5-year survival or operative mortality between the two techniques[10]. Other groups have suggested that performing a splenectomy with gastrectomy might result in a significant survival advantage[11], but subsequent groups in fact found this to be detrimental[12].

2. The extent of lymphadenectomy performed.
A D1 procedure removes the perigastric nodes, while a D2 procedure involves removing the nodes of the coeliac axis and the hepatoduodenal ligament in addition to the nodes taken in the D1 procedure. A multicentre prospective study demonstrated no significant improvement in survival and greater morbidity associated with the more extensive resection[13], but two studies from the UK[14] and Italy[15] in fact found a significant survival advantage to performing a D2 lymphadenectomy without significantly increasing either morbidity or mortality.

In general, a total gastrectomy provides the widest possible resection margins from the primary tumour, and a D2 lymphadenectomy, without splenectomy, involving removal of at least 25 nodes, should be undertaken concurrently. An oesophagogastrectomy may be needed for tumours at the cardia.



To date, no single agent chemotherapeutic regimen has been reliably shown to produce a survival benefit in gastric cancer. Conversely, combination chemotherapy may fulfil a far greater role in the treatment of gastric cancer.

NON-SURGICAL

Chemotherapy

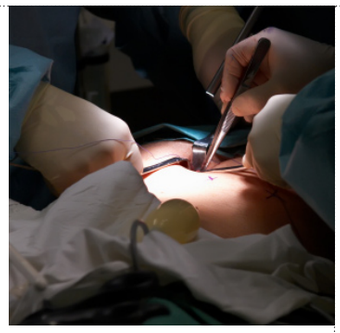
To date, no single agent chemotherapeutic regimen has been reliably shown to produce a survival benefit in gastric cancer. Conversely, combination chemotherapy may fulfil a far greater role in the treatment of gastric cancer.

The recently published MAGIC trial, in which patients with resectable tumours were randomised to receive either surgery alone, or surgery with perioperative (given before and after surgery) chemotherapy using a combination of epirubicin, cisplatin and fluorouracil (ECF), demonstrated greater progression free survival and overall survival in patients receiving chemotherapy compared to those undergoing surgery alone[16].

For patients with more advanced cancer not suitable for operative intervention, a Cochrane database review found a combination chemotherapy regimen significantly improved patient survival when compared to best supportive care and also single agent chemotherapy[17]. The regime producing the best survival results with greatest tolerability was epirubicin, cisplatin and continuous 5-FU.

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild



Endoscopy

Endoscopic mucosal resection (EMR) was pioneered in Japan, where screening programs for gastric cancer detected a very high proportion (50%) of early gastric cancer (EGC – a T1 lesion). The importance of detecting EGC less than 30mm in diameter is that only 3.5% of patients have associated lymph nodes metastases. Removal of these lesions endoscopically using the technique of EMR, where lesions are 'lifted' by submucosal injection of fluid permitting them to be more easily excised using a snare, therefore presents a far less invasive prospect for cure than surgical resection. Studies from Japan have borne this theory out, with similar mortality rates reported for EGC treated with EMR as for those treated surgically18. EMR is now accepted as an established treatment in Japan for T1 lesions. However in the UK, it is not standard practice, a recent Cochrane database review concluding that currently there is a lack of evidence from randomised controlled trials to support the routine use of EMR in treating EGC lesions[19].

PALLIATIVE

Research examining the role of palliative gastrectomy combined with palliative chemotherapy in patients with stage IV disease suggests a combined approach may prolong survival compared to patients not undergoing surgical intervention[20].

The patient underwent palliative bypass surgery from which he made an uneventful recovery and was able to be discharged home. At surgical follow-up, he asked about his likely prognosis from his disease.

Q. Is it possible to estimate what his survival might be?

Similar to oesophageal cancer, the TNM classification system can be used to stratify patients into clinical stages, from which estimates about survival can be made from currently available evidence. This is presented in Figure 8 below. Even given the briefest examination of these figures, it can be seen that except for very early stage lesions, survival prospects following a diagnosis of gastric cancer, in similarity to oesophageal cancer, are universally poor.

Stage	TNM Staging	5-year survival
IA	T1, N0, M0 or T1, N1, M0	60–80%
IB	T1, N2, M0 or T2a/b, N0, M0	50–60%
II	T1, N2, M0 or T2, N1, M0 or T2, N0, M0	30–40%
IIIA	T2, N2, M0 or T3, N1, M0 or T4, N0, M0	20%
IIIB	T3, N2, M0	10%
IV	T1-3, N3, M0 or T4, N1-3, M0 or Any T, any N, M1	Less than 5%

Figure 8: Prognosis for gastric cancer based on stage of disease.

Questions for reflection

Can you describe the epidemiology of oesophageal and gastric cancer?

What are the main risk factors for the development of oesophageal and gastric cancer?

What are the most common presenting symptoms of each cancer type?

Can you describe how you might go about investigating each type of cancer, and what are the potential limitations to the investigations used in the staging process?

Are you aware of how each cancer is staged?

What are the main approaches to treatment, what can be achieved with each treatment type, and can you estimate what a patient's likely outcome following a diagnosis of oesophageal or gastric cancer might be?

UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

Multiple Choice (True/False) Questions

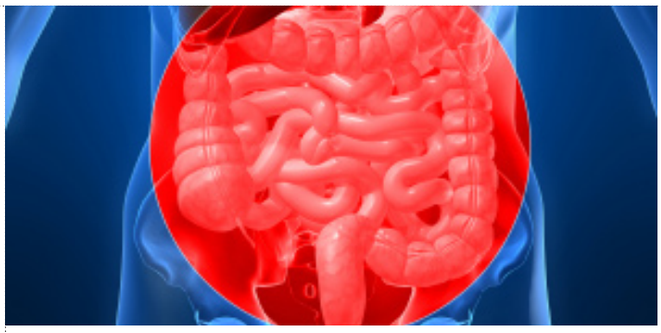
1. Regarding upper gastrointestinal symptoms and investigations:
a. Dyspepsia affects 1-2% of the population and warrants immediate endoscopic investigation.
b. Dysphagia always warrants immediate endoscopic investigation.
c. Dysphagia can be the first presenting symptom of gastric cancer.
d. In patients with unexplained weight loss, upper GI endoscopy is a mandatory first line investigation.
e. Diagnostic upper GI endoscopy is contraindicated in patients with an INR >3.0.

2. Regarding staging investigations for oesophageal cancer:
a. CT is most useful for assessing the M stage of disease.
b. EUS is superior to CT in assessing regional lymph node involvement.
c. The risk of oesophageal perforation at EUS is similar to that for an upper GI endoscopy.
d. PET/CT has good spatial resolution and can be used to accurately assess lymph node involvement.
e. MRI is often used in the staging of oesophageal tumours.

3. Regarding treatment options for oesophageal cancer:
a. Patients >70 years of age are not suitable candidates for oesophageactomy.
b. Neoadjuvant chemo-radiotherapy followed by surgery confers a survival benefit over those treated with surgery alone.
c. Chemotherapy is used routinely in the adjuvant setting.
d. Endoscopic mucosal resection for early (stage 0 or 1) tumours results in an improved 5-year survival when compared to surgery.
e. The risk of oesophageal perforation associated with placement of a SEMS for palliation of dysphagia is approximately 10%.

4. Regarding risk factors for gastric cancer:
a. Diet may play and important role in the development of gastric cancer.
b. Smoking is not an acknowledged risk factor for the development of gastric cancer.
c. Previous gastric resection can increase the risk of developing gastric cancer, even if the surgery was performed for benign disease.
d. Approximately 1% of gastric cancers are thought to be familial.
e. Atrophic gastritis in association with pernicious anaemia is not a risk factor for the development of gastric cancer.

5. Regarding treatment and survival in gastric cancer:
a. Approximately 50% of patients with gastric cancer are found to have metastases at diagnosis.
b. The majority of patients diagnosed with gastric cancer undergo curative surgical resection.
c. Combination chemotherapy given perioperatively has shown no survival benefits over single agent chemotherapeutic regimens.
d. Combination chemotherapy can be used in the palliative setting to improve survival.
e. 5-year survival for a stage I gastric cancer is likely to be greater than 50%.



Answers

1. Regarding upper gastrointestinal symptoms and investigations:
a. False **b.** False **c.** True **d.** False **e.** False

2. Regarding staging investigations for oesophageal cancer:
a. True **b.** True **c.** False **d.** False **e.** False

3. Regarding treatment options for oesophageal cancer:
a. False **b.** True **c.** False **d.** False **e.** False

4. Regarding risk factors for gastric cancer:
a. True **b.** False **c.** True **d.** False **e.** False

5. Regarding treatment and survival in gastric cancer:
a. False **b.** False **c.** False **d.** True **e.** True

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UPPER GASTROINTESTINAL CANCER

Gareth J Sadler, Gehanjali D A Amarasinghe and Penny J Neild

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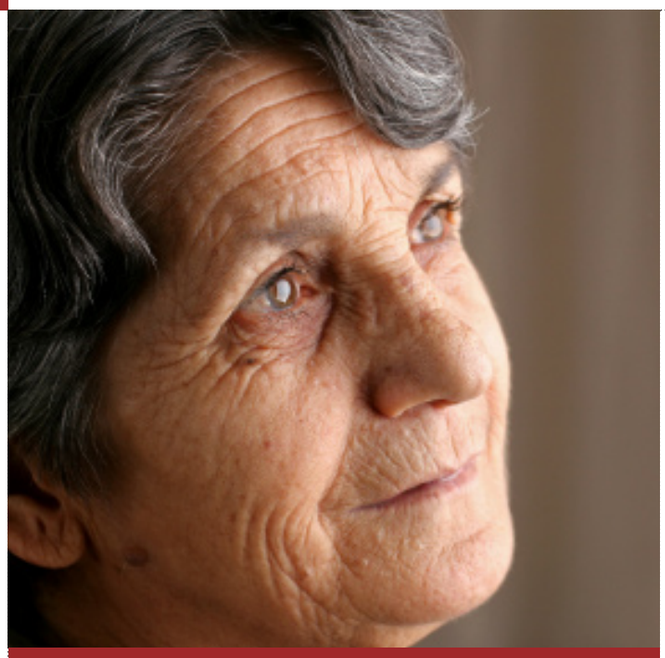
CHRONIC DIARRHOEA

Sanjeev Pattni and Julian RF Walters



A 49-year-old housewife was referred to gastroenterology outpatients with a 2-year history of diarrhoea. The GP letter outlined that she had recently been opening her bowels 6–10 times a day and 1–2 times at night.
Patient Management.

Case History

A 49-year-old housewife was referred to gastroenterology outpatients with a 2-year history of diarrhoea. The GP letter outlined that she had recently been opening her bowels 6–10 times a day and 1–2 times at night. The diarrhoea was described as watery with no blood seen. There was no relation to dietary factors (milk, bread, etc.) and no history of weight loss. She felt that her symptoms were significantly affecting her life and found it embarrassing to leave her flat. There was no family history of colonic neoplasm or inflammatory bowel disease.

She had a previous diagnosis of chronic pancreatitis diagnosed in 1980, but pancreatic enzyme replacement had been ineffective. Currently she was not on any medication except for some PRN imodium. She denied laxative use.

Examination was unremarkable with no signs of malabsorption.

Colonic
Colonic neoplasia
Ulcerative and Crohn's colitis
Microscopic colitis
Small bowel
Coeliac disease
Crohn's disease
Other small bowel enteropathies (e.g. Whipple's disease, tropical sprue, amyloid), intestinal
Lymphangiectasia
Bile acid malabsorption
Lactose malabsorption
Small bowel bacterial overgrowth
Mesenteric ischaemia
Radiation enteritis
Lymphoma
Giardiasis and other chronic infection
Pancreatic
Chronic pancreatitis
Pancreatic carcinoma
Cystic fibrosis
Endocrine
Hyperthyroidism, diabetes, hypoparathyroidism, Addison's disease, hormone secreting
tumours (VIPoma, gastrinoma, carcinoid)
Other
Factitious diarrhoea
Drugs, Alcohol, Autonomic neuropathy
Surgical causes (e.g. small bowel resection, internal fistulae)

Table 1: What are the causes of chronic diarrhoea?

How would you investigate this patient?

Almost all patients with chronic diarrhoea need to be investigated appropriately and taken seriously especially as symptom reporting forms the basis for the diagnosis, there can be considerable overlap between functional bowel disease (IBS) and "true" diarrhoea and potential seriousness of certain conditions, for example, colonic neoplasm that could be missed.

Definition

The perception of diarrhoea can vary widely between the patient and the doctor, particularly in the patient's conception of stool frequency and consistency. A pragmatic and clinical definition of chronic diarrhoea may be defined as the abnormal passage of three or more loose or liquid stools per day for more than 4 weeks and/or a daily weight greater than 200g per day.

CHRONIC DIARRHOEA

Sanjeev Pattni and Julian RF Walters

History and examination

Initial assessments of patients with chronic diarrhoea can be mostly carried out in primary care setting. A detailed history is crucial in assessments of patients with chronic diarrhoea. This can often establish the likelihood that the symptoms are organic or functional and be able to distinguish malabsorption from colonic and inflammatory forms of diarrhoea and assess for specific causes of diarrhoea.

Symptoms suggestive of organic disease include:

- history of diarrhoea less than 3 months
- nocturnal or continuous
- significant weight loss
- steatorrhoea and bulky malodorous pale stools (suggestive of malabsorption)
- liquid stools with blood or mucus discharge (suggest colonic, inflammatory or secretory forms of diarrhoea).

Symptoms suggestive of functional disease include:

Various symptoms have been used to identify IBS, such as Rome criteria, but they can be of less practical significance on an individual patient basis and can overlap with organic causes. Some of these symptoms include:

- intermittent diarrhoea +/- constipation
- bloating, intermittent abdominal discomfort
- altered stool passage (straining, urgency, incomplete evacuation)
- passage of mucus.

These symptoms and a normal physical examination would be suggestive of functional bowel disturbance, but only with a specifity of 52–74%.

Other aspects of the history, such as family history, previous surgery, systemic diseases (e.g. hyperthyroidism, diabetes mellitus), alcohol, drugs (e.g. magnesium containing products, NSAIDs, antibiotics, antiarrhythmics, antihypertensives, some antidepressants, metformin, chemotherapeutic agents) and travel history, can all provide specific pointers to other causes of diarrhoea.

A thorough examination is vital and can provide clues to systemic disease. Clubbing is associated with inflammatory bowel disease. Koilonychia is a sign of iron deficiency anaemia (GI malignancy, coeliac disease). Lymph nodes may indicate underlying malignancy. Previous abdominal surgical scars are important to elicit as ileal resection for Crohn's disease would lead to bile acid malabsorption. Location of abdominal tenderness and/or masses would again direct the focus for further investigation into the cause of chronic diarrhoea.

What would you like to do next?

Blood tests

A basic screen should include FBC, U&Es, LFTs, vitamin B12, folate, calcium, ferritin, ESR or CRP, TFTs, fasting glucose and anti-tissue transglutaminase IgA antibody test (anti-tTG) for coeliac disease. Anti-tTG is increasingly available but anti-endomysial IgA antibodies are also used. Both these tests have a sensitivity of 86–100%, and specificity of 98–100% but TTG is cheaper and easier to perform. Patients should go on to have an endoscopy and duodenal biopsies if serological tests are positive or if a strong suspicion for coeliac disease remains. This is because individuals with lesser degrees of atrophy may be EMA negative and tTG has been found to be falsely positive in other autoimmune disease.

Stool tests

Stool cultures should be requested. If there is any history of travel to high-risk areas then examinations for ova, cysts and parasites should be considered. Clostridium difficile toxin testing should be performed if there has been previous antibiotic use. Screening for laxative abuse should be performed if factitious diarrhoea is suspected by measuring faecal osmolality in a stool sample.

What would you like to do next?

In most patients with chronic diarrhoea, endoscopic investigation will be necessary.

In young patients (<45 years age) flexible sigmoidoscopy would suffice in looking at left colonic pathology and sampling of colonic mucosa for histological examination. It has been shown that in this age group most pathology occurs in the distal colon and this is accessible by flexible sigmoidoscopy.

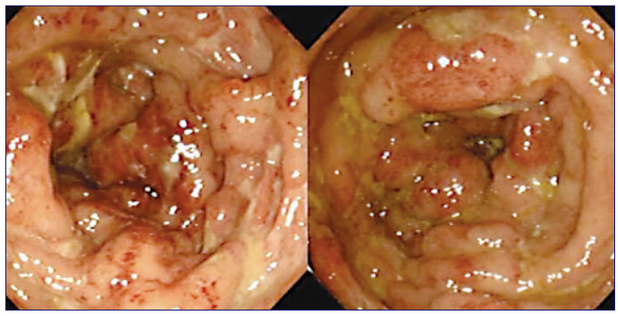


Figure 1: Severe Crohns disease. Source: Gastrosource.

In patients over 45 years with chronic diarrhoea, colonoscopy with ileoscopy is the preferred investigation. This may yield abnormalities in up to 30% of cases, has a better sensitivity than barium enema and allows sampling of colonic mucosa for histological examination.

This patient underwent colonoscopy which was normal and random biopsies did not reveal any evidence for IBD, microscopic colitis or ischaemic colitis.

CHRONIC DIARRHOEA

Sanjeev Pattni and Julian RF Walters

What further tests would be helpful?

1. non-invasive tests for small bowel malabsorption.

a. small bowel imaging (barium follow through) should be reserved for cases where small bowel malabsorption is suspected and distal duodenal histology is normal. Barium enteroclysis (introduction of barium directly into the small intestine via a nasoduodenal tube) is discouraged due to its difficulty in performing the test and patient discomfort.

Video capsule endoscopy is being increasingly used in evaluating small bowel pathology as it allows for direct imaging of the entire small intestine. Although its primary indication is in the investigation of patients with obscure GI bleeding, its uses are expanding and appear promising; these include its use in diagnosing small bowel nonstricturing Crohn's disease, malabsorption syndromes (e.g. primary intestinal lymphangiectasia), small bowel lymphoma, and perhaps coeliac disease. Its suggested use is probably as an adjuvant test or as a secondary investigation.

b. stool tests for fat malabsorption, e.g. faecal fat collections and breath tests for fat malabsorption are outdated and seldom carried out these days. They are poorly reproducible, unpleasant and have low sensitivity for mild or moderate malabsorption. These tests should be discouraged.

2. Non-invasive test for pancreatic disease.

CT imaging can look for calcium deposits and atrophy. This has a sensitivity of 74–90% for pancreatic disease and is higher than that of ultrasound.

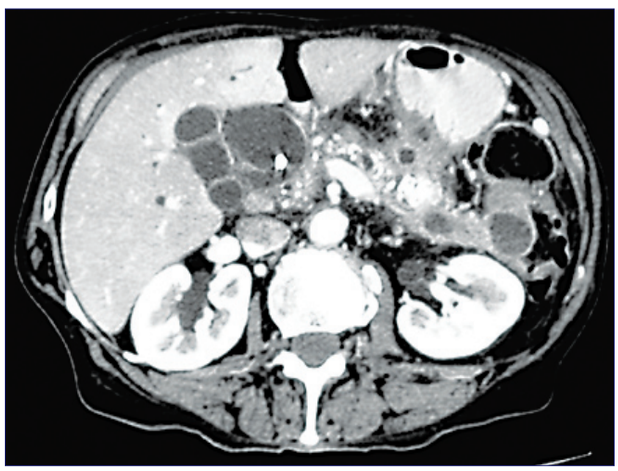


Figure 2: CT abdomen showing features of chronic pancreatitis: atrophy, calcification and pseudocysts .

Magnetic resonance cholangiopancreatography (MRCP) can also provide further information regarding any abnormal ductal system and can be used prior to ERCP, as the latter has issues with complication risks; MRI pancreatography after secretin stimulation may provide functional as well as structural information on the pancreas. Faecal pancreatic elastase is the preferred test to identify moderately severe pancreatic insufficiency, rather than the previously used pancreolauryl and BTP/PABA tests, due to its ease of use. A small random stool sample is sufficient to detect this.

3. Other specific tests.

Glucose hydrogen breath test – small bowel bacterial overgrowth. The sensitivity for this test, however, is only 60% and therefore a culture of jejunal aspirates or unwashed small bowel biopsies remains the gold standard, but practically difficult to perform routinely. Often a therapeutic trial of antibiotics in patients with abnormal gastrointestinal anatomy or physiology is warranted.

Lactose hydrogen breath test – lactose malabsorption. Lactase non-persistence is common, particularly in people whose ethnic origin was not European. However, a significant proportion of ethnic Europeans (10%) have lactose malabsorption which may not have been previously identified from symptoms of milk intolerance.

SeHCAT Scintigraphy – Bile Acid Malabsorption.

This patient underwent a SeHCAT test in the nuclear medicine department with a result of 5% (normal >10%), suggesting that she has bile acid malabsorption.

Bile acid malabsorption

Primary Idiopathic bile acid malabsorption (IBAM) is an under recognised cause of chronic diarrhoea and is often misdiagnosed as diarrhoea predominant irritable bowel syndrome. IBS patients are the largest group of patients seen in a general gastroenterology clinic. Many studies suggest that 30% of patients with previously unexplained chronic diarrhoea have impaired bile acid malabsorption.

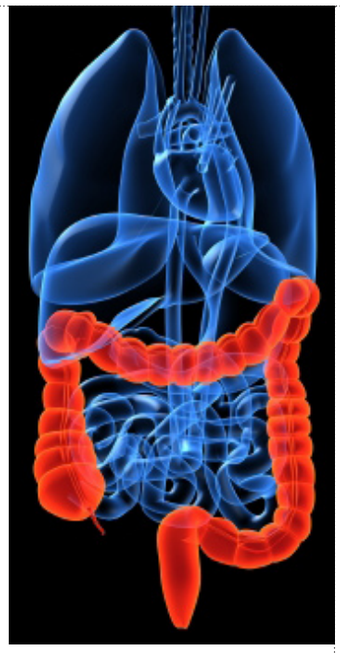
Bile acids are essential in fat digestion and facilitating intestinal absorption and also eliminate cholesterol.

Bile acids are synthesised in the liver from degradation of cholesterol and in the conjugated form are transported into bile ducts. They then accumulate and are stored in the gall bladder where they flow into the duodenum following meal stimulated gall bladder contraction.

The enterohepatic circulation of bile acids are crucial to the recycling of them; 95% of the bile acid pool is reabsorbed in the distal ileum by an efficient and well characterised transport system; they are then returned to the liver by the portal vein where they are taken up by the hepatocytes and resecreted into bile ducts and hence completing the enterohepatic cycling.

CHRONIC DIARRHOEA

Sanjeev Pattni and Julian RF Walters



Failure of absorption of bile acids by the distal ileum results in the spillover of bile acids into the colon where the acids stimulate electrolyte and water secretion, which results in loose to watery stools. BA malabsorption has been divided into three types depending on their aetiology. Type 1 refers to those patients with ileal disease or resection as found commonly in patients with Crohn's disease. Type 2 disease occurs in the absence of ileal disease, a positive SeHCAT retention and response to bile acid sequestrants. This is more commonly known as primary idiopathic bile acid malabsorption. Last, other conditions give rise to bile acid malabsorption and include, post-cholecystectomy, post vagotomy, coeliac disease, diabetes mellitus, pancreatic insufficiency and bacterial overgrowth. This group of patients are collectively banded as type 3.

Diagnosis and treatment of bile acid malabsorption

The most commonly used test for this diagnosis is the SeHCAT (Se-homocholic acid taurine) test. The Se-labelled bile acid is administered orally and the total body retention is measured with a gamma camera after 7 days. Retention value of less than 10% is considered abnormal and indicative of BAM. Diarrhoea in patients with greatly reduced SeHCAT retention usually responds to oral bile acid sequestrants, such as colestyramine or colestipol, which bind to bile acids in the gut. Recently another bile acid sequestrant, called colesevelam, has been introduced which is in a capsule form and hence more palatable. The SeHCAT test is able to evaluate BAM with a sensitivity of 80–90% and specificity of 70–100%.

Questions

1. A 40-year-old Asian woman was referred to the clinic with a 9-month history of watery diarrhoea, abdominal discomfort and bloating. She had frequented her GP who had diagnosed IBS. She has a past medical history of depression and has been on fluoxetine for 2 years and drinks on average 15–20 units of alcohol per week. She has a family history of Crohn's disease. What is the most appropriate initial test for this lady:

a. Colonoscopy.
b. Lactose hydrogen breath test.
c. Stool specimen to measure the osmotic gap to differentiate between osmotic, c. secretory and factitious diarrhoea.
d. CT abdomen.
e. Trial of mebeverine.
f. Tissue transglutaminase IgA antibodies.

2. The most appropriate initial screening test for coeliac disease is:

a. Duodenal biopsy.
b. Trial of a gluten-free diet.
c. Gliadin IgA antibodies.
d. Total IgA measurement.
e. Tissue transglutaminase IgA antibodies.

3. What features are suggestive of Crohn's disease at the time of colonoscopy?

a. Cobblestone appearance.
b. Aphthoid ulceration.
c. Fistula formation.
d. Marked patchy ulceration and mucosal oedema.
e. Deep ulceration with pseudopolyp formation and fixed caecum.

4. The treatment for Primary bile acid malabsorption includes:

a. Short course of oral prednisolone.
b. Cyclical antibiotics.
c. Colestipol.
d. Dietary advice for low fat diet.
e. Imodium for symptom control.

5. Chronic diarrhoea for three months is unlikely to be caused by :

a. Campylobacter jejuni.
b. Metoclopromide.
c. Amoebic infection.
d. Laxative abuse.
e. Ulcerative colitis.

Answers

Question 1.

Answer b or f

Flexible sigmoidoscopy is recommended in patients under 45 in the first instance as the diagnostic yield differs little from the use of colonoscopy in this age group. Stool testing for osmotic gap is seldom of practical use and can be non-specific. In difficult cases, however, this measurement may provide evidence if factitious diarrhoea is suspected. A CT abdomen would be helpful in chronic pancreatitis, but there is no comment of steatorrhoea or weight loss to suggest this diagnosis, but would be considered as a secondary line of investigation. The likely diagnosis in this case and age group would be IBS; lactose hydrogen breath test would be appropriate due to her ethnicity as would tissue transglutaminase IgA antibodies to exclude coeliac disease.

Question 2.

Answer e

This is the preferred serological test for coeliac disease with a sensitivity of 99% and specifity of >90%. One has to bear in mind that this condition is associated with selective IgA deficiency, which will give rise to false negative serum IgA antibody tests. Selective IgA defiency occurs in 1:500 (0.2%)–1:700 (0.14%) of the general population but in 2.6% of patients with coeliac disease.

CHRONIC DIARRHOEA

Sanjeev Pattni and Julian RF Walters

Question 3.

Answer b

Cobblestone appearance is a radiological appearance. Fistulae are difficult to identify at colonoscopy and again are typically seen on a barium study or a MRI scan. Marked patchy inflammation is more common in ischaemic colitis and a fixed caecum is suggestive of tuberous colitis.

Question 4.

Answer c

The treatment for bile acid malabsorption is with a bile acid sequestrant such as colestyramine, colestipol or colesevelam. Steroids would be used in IBD or microscopic colitis. Cyclical antibiotics are used in the treatment of small bowel bacterial overgrowth.

Question 5.

Answer a

Campylobacter jejuni typically is a cause of acute diarrhoea while the others cause chronic diarrhoea.

Further reading

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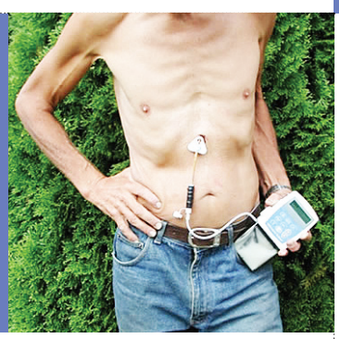
PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Gehanjali D A Amarasinghe, Gareth J Sadler and Penny J Neild

Identifying and addressing malnutrition is an important aspect of patient care. When oral feeding is not adequate or is unsafe, alternative methods of nourishment are imperative. In this article, we focus on percutaneous endoscopic gastrostomy (PEG) – indications, contraindications, consent, pre-procedure checks, what PEG insertion actually involves and aftercare.
Practical Procedures.



Relevance to the Curriculum

1.0 Good clinical care
1.1 History, examination, diagnosis, record keeping, safe prescribing and reflective practice
1.2 Time management and decision-making
1.3 Patient safety
1.4 Infection control
1.5 Clinical governance
1.6 Nutrition care
1.7 Health promotion, patient education and public health
1.8 Ethical and legal issues

2.0 Maintaining good medical practice
2.1 Learning
2.2 Research, evidence and guidelines

4.0 Relationship with patients and communication skills

Abstract
Identifying and addressing malnutrition is an important aspect of patient care. When oral feeding is not adequate or is unsafe, alternative methods of nourishment are imperative. In this article, we focus on percutaneous endoscopic gastrostomy (PEG) – indications, contraindications, consent, pre-procedure checks, what PEG insertion actually involves and aftercare.

Keywords
Percutaneous endoscopic gastrostomy; PEG; enteral feeding.

Introduction
Identifying and addressing malnutrition is an important aspect of patient care as it renders a patient more susceptible to ill health, increases vulnerability to infections, leads to delayed wound healing, impaired function of major organs, muscle weakness and depression[1]. It is surprisingly common in the UK, and is often seen in inpatients. Current NICE guidance state that all inpatients should be screened on admission for malnutrition, and if present, or a patient is at risk, then appropriate steps should be taken to address this issue (ref).

When oral feeding, with or without meal supplements, is not adequate or is unsafe, alternative methods of nourishment are imperative to stave off worsening malnutrition and its consequences. Alternative feeding may be in the form of enteral feeding, using either nasogastric methods or percutaneous methods. If enteral feeding is not possible due to a poorly functional or inaccessible gastrointestinal tract, then parenteral feeding should be considered. This article will focus on enteral feeding via a percutaneous endoscopic gastrostomy (PEG).

PEG insertion was introduced in the 1980s, initially in the paediatric population, and has been steadily growing in practice since. PEG tubes are usually made of polyurethane or silicone rubber. The placement procedure is faster, less expensive and associated with fewer complications, than open gastrostomy[9,10]. Another alternative to a PEG is a radiologically inserted gastrostomy (RIG).

Indications for PEG placement
- Patients who are unable to meet their nutritional requirements orally and require supplementary feeding on a long term basis (> 4 weeks), such as patients with systemic sclerosis, cystic fibrosis or HIV/AIDS.
- neurological dysphagia, persisting for longer than 2–4 weeks and which is unlikely to improve, such as stroke.
- Progressive neurological dysphagia, for example, amyotrophic lateral sclerosis.
- Patients with head and neck cancers undergoing therapy, where oral feeding is compromised.
- Persisting altered level of consciousness, where oral feeding is not possible.

Along with considering specific indications for PEG feeding, it is also important to consider the appropriateness of subjecting a patient to the potential risks of PEG insertion and feeding, especially where severe co-morbidity, poor quality of life or limited life expectancy may weigh against such a decision. It is important to consult widely in such cases, both among carers and other staff members, and to always involve senior medical staff in the final decision making process.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Gehanjali D A Amarasinghe, Gareth J Sadler and Penny J Neild

Contraindications to PEG placement

Below are commonly quoted contraindications to PEG placement. Always discuss these with the gastroenterology department though, as they will be able to give you appropriate advice regarding individual cases or alternatives.

- Total gastrectomy (or other extensive surgery that may make tube placement difficult).
- Abdominal ascites (more likely to develop complications, although risks are lower when ascites are mild to moderate[12]).
- Continuous ambulatory peritoneal dialysis or ventriculo-peritoneal shunts (same as for abdominal ascites, although risks are currently thought to be low[12]).
- Oesophageal obstruction (unable to access the stomach via endoscopy).
- Gastric outflow obstruction.
- Gastric ulcers/cancer. Ulcers should be confirmed to have healed prior to attempting PEG placement.
- Immunocompromised states (associated with higher rates of infection and peristomal leakage[12,15]).
- Bleeding disorders (clotting would need to be corrected).
- Sepsis.
- Anorexia nervosa.
- (advanced dementia)

Consent

The British Society of Gastroenterology has published updated guidance for obtaining valid consent for elective endoscopic procedures[5]. Ensure you are using the appropriate consent form. Information should be provided regarding PEG placement – what it involves, benefits, risks and alternatives (which may be options if PEG placement is not successful). Information leaflets are useful.

- If the patient is able to give valid consent for the procedure, ensure consent is obtained at an appropriate time and in appropriate surroundings.
- If a patient is not deemed competent to give consent:

- Consent may be given by an individual nominated within a valid Personal Welfare Lasting Power of Attorney (LPA), acting in the best interests of the patient[5].
- If there is a valid advanced directive, you must abide by that decision[5].
- If there is no personal welfare LPA, but there are legal next of kin or guardians, PEG insertion may be carried out if the medical team deem it to be in the best interests of the patient. Two senior clinicians involved in the care of the patient must confirm that PEG placement is in the best interests of the patient by completing a special consent form (Department of Health consent form 4: for use where the patient is an adult unable to consent to investigation or treatment) [6]. It is good practice to consult family and next of kin as they are likely to be aware of the wishes and beliefs of the patient[5].
- If an incompetent patient has no identified next of kin or significant friends other than paid carers, an Independent Mental Capacity Advocate (IMCA) must be appointed "to represent and support them when important or potentially life saving decisions are to be made" [5].

Pre-procedure checks

- Ensure valid indications and no contraindications.
- Ensure the patient has given informed consent. If the patient is unable to give their own consent, ensure the correct consent form is completed.
- MRSA swabs – refer to local protocols. Nasopharyngeal MRSA screening and decolonisation has been found to reduce peristomal MRSA infection rates[14].
- Check full blood count and clotting prior to procedure – ensure INR ideally <1.3 but not more than 1.5 and platelets ideally >70 x 10^9/L but not less than 50 x 10^9/L12,16.
- If on warfarin, ensure warfarin is stopped 5 days prior to PEG placement and appropriate anticoagulant cover is prescribed (see Table 1).
- If on clopidogrel, ensure it is stopped 7 days prior to PEG placement (liaise with cardiologist) and appropriate antiplatelet cover is prescribed (see Table 1).
- There is no reason to stop aspirin prior to PEG placement[16].
- Patient should be fasted 6 hours prior to procedure or longer if impaired gastric motility.
- Ensure adequate IV access is available.
- Ensure medication and allergies checked (for example, diabetic patients should be told to omit their morning anti-diabetic medication and be placed early on the list).
- Ensurea prophylactic antibiotic has been prescribed and administered 1 hour pre-procedure – refer to local protocols. Prophylactic antibiotics have been shown to reduce rates of infection related to PEG placement from around 15% to 3%[1,11].
- Ensure a feeding regime has been prepared in advance in liaison with the dietitian so that commencement of feeding is not delayed.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Gehanjali D A Amarasinghe, Gareth J Sadler and Penny J Neild

Warfarin	
LOW RISK INDICATION **For example,** **AF without valvular disease:** • Stop warfarin 5 days before PEG placement • Ensure INR prior to procedure <1.5 • Restart warfarin evening of procedure with usual dose • Recheck INR a week later to ensure therapeutic	**HIGH RISK INDICATION** **For example,** **metal mitral valve:** • Stop warfarin 5 days before PEG placement • Start LMWH 2 days after stopping warfarin • Omit LMWH on day of PEG placement • Restart warfarin evening of procedure with usual dose and continue LMWH until INR is therapeutic
Clopidogrel	
LOW RISK INDICATION **For example, ischaemic heart disease without coronary artery stents:** • Stop clopidogrel 7 days before PEG placement • If on aspirin, continue this. If not on aspirin, consider giving aspirin whilst off clopidogrel • Restart clopidogrel after PEG placement	**HIGH RISK INDICATION** **For example, drug eluting coronary artery stents:** • Consider stopping clopidogrel 7 days before PEG placement. Aspirin should be continued • Liaise with cardiologist. Clopidogrel may be stopped if >12 months after insertion of a drug eluting coronary stent or >1 month after a bare metal coronary stent

Table 1: Current British Society of Gastroenterology guidance on anticoagulant and antiplatelet therapy for patients undergoing PEG placement[16].

PEG procedure

Pre-procedure, oxygen saturation, blood pressure and pulse are checked. Appropriate sedation is administered (usually 1–2mg of midazolam). An oesophago-gastroduodenoscopy (OGD) is performed by the endoscopist to ensure that there are no contraindications to PEG placement.

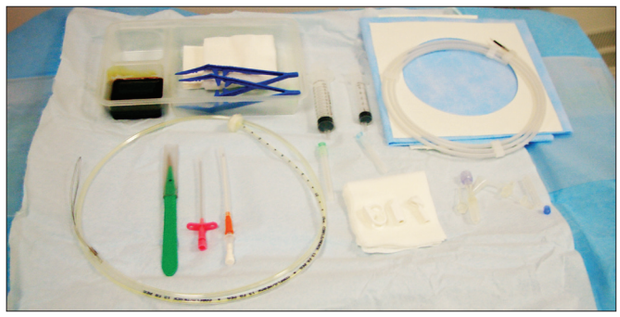


Figure 1: An assistant prepares the necessary equipment for the procedure.

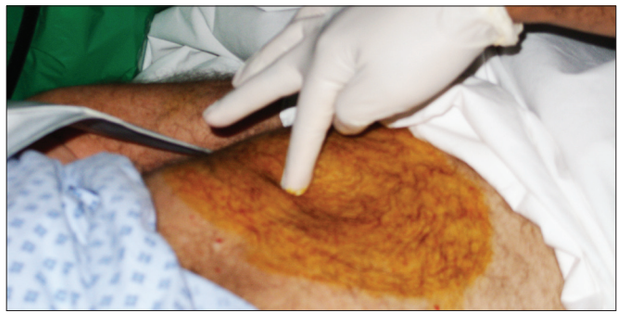


Figure 2: The patient is now placed in a supine position, air is insufflated into the stomach so that the anterior wall of the stomach is in apposition with the anterior abdominal wall. The tip of the endoscope is pointed upwards towards the anterior abdominal wall. An assistant observes the abdomen for transillumination, and the site indented with a finger.

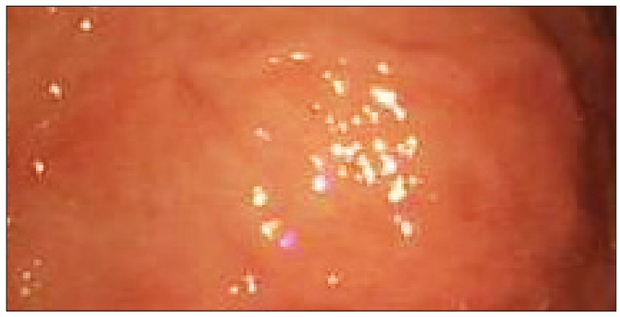


Figure 3: The endoscopist should be able to visualise this indentation from within the stomach with the endoscope.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Gehanjali D A Amarasinghe, Gareth J Sadler and Penny J Neild

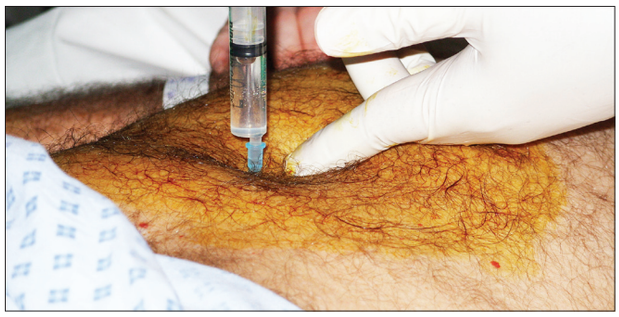


Figure 4: The site is cleaned aseptically and infiltrated with local anaesthetic.

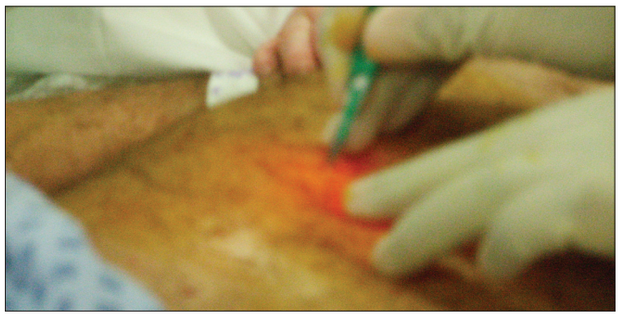


Figure 5: A small incision is made in the skin and a cannula is introduced. Incidentally, transillumination from the endoscope is demonstrated well in this picture.

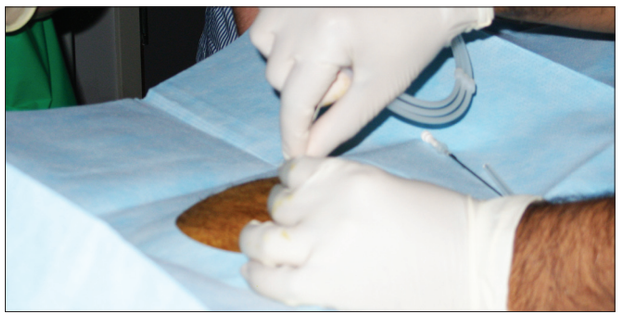


Figure 6: A guide wire is passed through the cannula into the stomach.

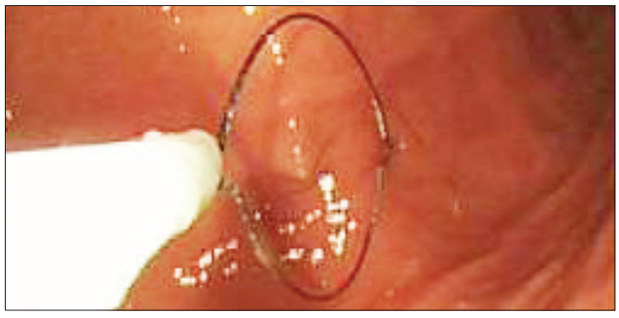


Figure 7: The guide wire is grasped by a snare, which is passed through the endoscope.

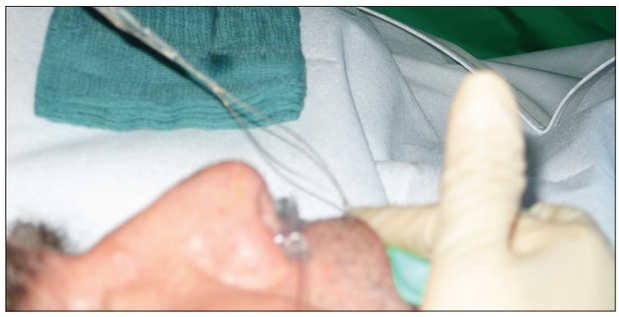


Figure 8: The endoscope and the snare are withdrawn along with the wire out of the mouth (the other end of the wire should remain outside the abdominal wall). The guide wire that is withdrawn with the snare is then secured to the end of the PEG tube.

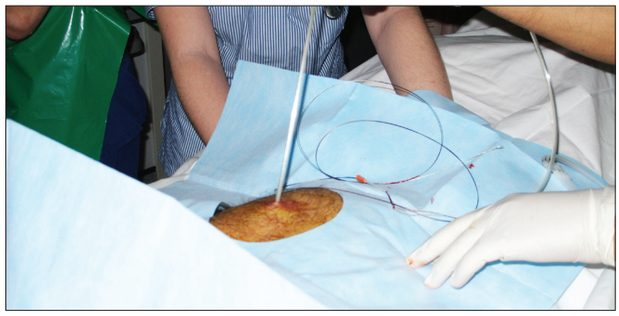


Figure 9: The end of the guide wire that is outside the abdominal wall is now pulled, bringing the PEG tube down the oesophagus and out through the abdominal wall.

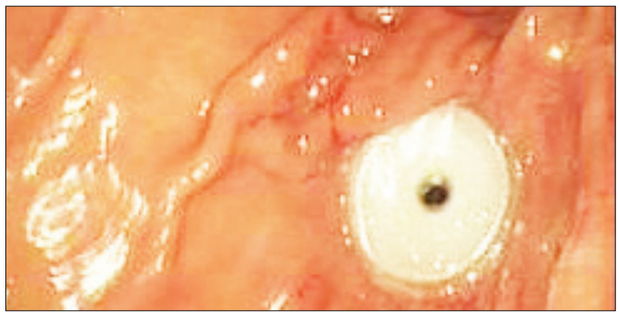


Figure 10: The end of the PEG sitting in the stomach is anchored in the stomach by a flange or "button". The endoscope is passed back into the stomach to check that the "button" is secure and safe.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

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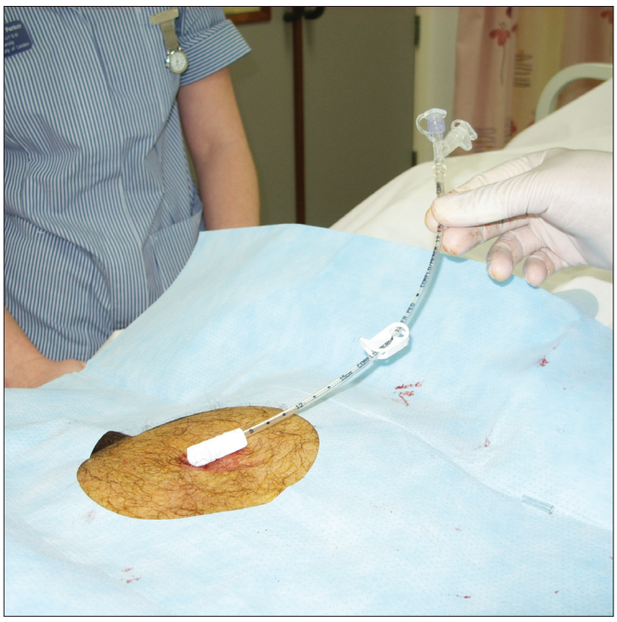


Figure 11: The end of the PEG tube sitting on the skin of the anterior abdominal wall is secured by a fixating device. It is still more commonly used than the "push" technique and the "introducer" techniques. These have not been described in this article. It is important to leave about 1cm of "give" to allow for oedema post procedure (if it is secured too tightly, the skin and tissue may necrose as oedema develops). The PEG tube is trimmed to an appropriate length and the connectors (which will be used to connect the PEG to a feeding pump) are attached.

Note: the above is known as the "pull technique" and was the first technique to be used. It is still more commonly used than the "push technique" or the "introducer technique" that have not been described in this article7.

Immediate post-procedure care

• Oxygen saturations, respiratory rate, pulse, blood pressure and temperature are monitored.

• The site is observed and kept clean.

• Water can be commenced via the PEG as early as 1–2 hours post-procedure[12], although current NICE guidance recommends waiting 4–6 hours post-procedure. If this is tolerated well, then feed may be introduced[1].

Ongoing care and follow-up

• It is imperative that the patient is sat up an hour prior to, during and an hour after feed is given to minimise aspiration[1,2,8].

• If a patient has not received adequate nutrition for some time prior to PEG placement, it is vital that NICE guidelines should be followed to minimise the risk of re-feeding syndrome[1]. These include the daily monitoring of serum magnesium, potassium, sodium and phosphate levels, and replacement where necessary. Feed should be initiated at a low rate and increased over a few days in consultation with the dietitian.

• Measurement of serum urea and creatinine levels are useful to ensure adequate hydration[1].

• The PEG must be flushed with water pre- and post-feed and medications to ensure patency.

• Although the site may initially be kept clean with a dry dressing, after 24 hours, it should be exposed and kept clean and dry[2].

• The site must be observed daily to watch for infection/collections.

• The PEG should be rotated daily to prevent formation of a "buried bumper".

• The patient should not have a bath (only showers!) or swim until 2 weeks post-procedure[2].

Complications

A list of complications associated with PEG placement is included below. Brief explanations have been included where necessary.

Early/peri procedural complications

• *Sore throat* post endoscopy is common, lasting for a few hours.

• *Infection* – this may occur locally in the skin and subcutaneous tissue at the PEG site and it affects about 15% of patients undergoing PEG placement[11,12]. Pre-procedure antibiotics and good infection control measures are important because of this. Ensuring the PEG is not too tightly secured is also an important measure[1,8,11]. When looking out for PEG site infection, note that local erythema <5mm around the site due to local irritation (and not infection) is common[12].

• *Bleeding* – overall rates of 1–2.5% have been quoted, the most common causes being perforation of a gastric wall vessel, gastric pressure ulceration, concomitant peptic ulcer disease and oesophagitis[11,13].

• *Perforation* associated with endoscopy itself is rare (0.008–0.04% 9) and with PEG insertion is 0.5–1.8% 13.

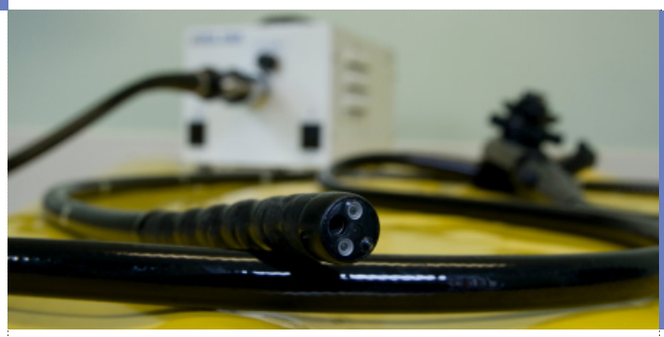
• *Risks of sedation* – using sedative medication sparingly is useful in reducing sedative related cardiopulmonary complications [11,13].

• *Pneumoperitoneum* – this is a benign occurrence seen in about 50% of cases when air leaks into the peritoneum during PEG placement. It is not associated with an adverse outcome and is self-limiting. Very rarely, it may be associated with peritonitis[11,12].

• *Acute and severe complications requiring surgical intervention* – these include life threatening bleeding, perforation and peritonitis and occur in less than 0.5% of cases[12].

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Gehanjali D A Amarasinghe, Gareth J Sadler and Penny J Neild



The business end of a flexible medical endoscope used for exploration of the upper gastroenteric tract (gastroscopy) the lower enteric tract (proctoscopy, colonoscopy & sigmoidoscopy) & the airways (bronchoscopy), also for taking biopsies, performing surgery & removing foreign bodies in human medicine. Practical Procedures.

Later complications

- *Buried bumper* – this is where the "bumper"/internal fixation device gets buried in overgranulated gastric mucosa, and migrates through the stomach wall. Incidence is reported in the literature as 1.5–1.9%[11].
- *Peristomal leakage* – this occurs in 1–2% of cases, and is more commonly seen in those who are immunocompromised[11,15].
- *Peristomal pain* – is common and usually short lived. If it persists, suspect infection[11].
- *Ileus* – some patients experience ileus post-procedure but this is usually short lived. Prolonged ileus however, may occur in 2% of patients[11,13]. If there is associated pain or any suggestion of peritonism, immediately rule out perforation.
- *Aspiration* – this is uncommon during PEG insertion (0.3–1.0%)13, but more commonly related to feeding. Thus, ensure patient is sat up before, during and after feeding. The merits of adding a jejunal extension to a PEG tube to reduce the risk of aspiration are not clear[1, 8, 11, 12] and these extensions have been known to migrate back through the pylorus into the stomach.
- *Accidental removal* – this occurs in 1.6–4.4% of cases[11].
- *Tube blockage* – this may occur in up to 45% of cases[11]. Flushing tubes before and after feed and medication is important (BAPEN drug administration via enteral tubes guideline). It may be more appropriate to use liquid/soluble formulations of medications. If tubes get blocked, seek help from your local dieticians/nutrition support team. Usually flushing with water is helpful, although the use of fizzy solutions and diluted pancreatic enzymes have been described as well.
- *Diarrhoea associated with feed* – discuss with dieticians/nutrition team regarding alteration of feed type/other measures. In rare cases, diarrhoea may be due to gastro-colo-cutaneous fistulae (with bowel interposed between stomach and the anterior abdominal wall).

Removal of PEG tubes

Despite the relatively robust design of PEG tubes, it is possible for both patients and cares to inadvertently dislodge them or completely pull them out. If a tube falls out more than 1 month after insertion, when a more mature tract will have formed, a replacement tube must be inserted into the tract as soon as possible. If it occurs before a mature tract has formed (<2 weeks), or there is a delay in inserting a replacement tube, a new PEG may have to be inserted in another site.

When not required any more, a PEG can be electively removed in the endoscopy department. After removal, a dry dressing may be applied until the wound heals over.

Radiologically inserted gastrostomy (RIG)

This is an alternative to PEG, especially if access by an endoscope is difficult, due to restricted mouth opening or oesophageal stricturing, or where there is concern regarding possible seeding from an oro-pharyngeal tumour. For the procedure itself, sedation and analgesia are given intravenously. Pulse oximetry and blood pressure are monitored. The stomach is distended with air via a nasogastic tube and fluoroscopic imaging is used. Around the chosen site a 2cm square is marked. At the four corners of this square, four "T fasteners" are used to anchor the stomach wall to the abdominal wall. The centre of this square is anaesthetised and a cannula is inserted into the stomach. A guide wire is passed through this into the stomach. A dilator is passed over this to dilate the tract. The gastrostomy tube is placed over the guide wire into the stomach. It is secured in the stomach with a balloon or similar retention device. RIGs usually last for up to about 6 months[15].

Minor complication rates are similar to those that occur with PEG placement. Major complication rates are slightly less compared to those associated with PEG placement and include peritonitis requiring laparotomy, gastric perforation, haemorrhage requiring transfusion, deep stomal infection, septicaemia and aspiration[15].

Summary

It is important to consider nutrition status in every patient. When oral feeding with supplements is inadequate, it is important to consider alternative enteral (or parenteral) feeding measures. Percutaneous endoscopic gastrostomy (PEG) is an effective and safe means of long-term enteral feeding when instituted in the correct patient for the correct reasons, with appropriate pre- and post-procedure care.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Gehanjali D A Amarasinghe, Gareth J Sadler and Penny J Neild

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TRAVELLERS' DIARRHOEA

Melissa Smith

A previously fit and well 28-year-old doctor develops abdominal cramps and watery diarrhoea five times a day on her honeymoon in Vietnam. Good Clinical Care.

A previously fit and well 28-year-old doctor develops abdominal cramps and watery diarrhoea five times a day on her honeymoon in Vietnam. She self-medicates with a 3-day course of ciprofloxacin 500mgs bd. On her return home (day 4), she attends her general practitioner complaining that her symptoms have not abated.

What is the most likely cause of her symptoms?

The patient's presentation is consistent with a diagnosis of travellers' diarrhoea (TD) which can be defined as: the passage of three or more loose stools in 24 hours (with or without the presence of fever, abdominal pain, nausea or vomiting) in an individual travelling abroad.

TD can occur on any trip, but the group most at risk are those from industrialised nations travelling to developing countries, particularly in Latin America, Africa and Southeast Asia. In this group the incidence reaches 40%. However, TD can occur on any trip, the incidence when travelling between developed nations is approximately 2%.

Approximately 800 million people travel abroad each year. Of those affected, 40% have to change itinerary, 20% are bed bound and 1% require hospital treatment. There are also potential long-term consequences which, although mostly rare, can be serious, (see below). This means that TD is a significant public health issue.

The vast majority of TD can be attributed to bacterial infections, although viral infection and parasitic infestation should also be considered. Table 1 shows the overall percentage of TD attributable to individual infectious agents. Although up to 50% of cases remain unexplained in some series, most of these cases are thought to be due to bacterial agents, as they can often be successfully treated with antibiotics and polymerase chain reaction (PCR) studies have proven that enterotoxigenic E. coli is in fact present in a large number of these cases.

Figures vary widely from series to series but approximate proportions are:	
20%	No cause identified (studies suggest the majority of these are bacterial, particularly E. coli)
60%	Bacterial Enterotoxigenic E. coli ~45% Other E. coli, Shigella, Campylobacter ~15% Salmonella, V. cholera – very rare Rates of Campylobacter are higher in SE Asia, even exceeding E. coli for prevalence in some studies
10%	Viral (norovirus, rotavirus – tend to be in outbreaks)
5%	Parasitic (Giardia, Cryptosporidium, Cyclospora, Entamoeba) These agents are more common in SE Asia and account for higher proportions of TD there (up to 12%)
5%	Food poisoning (Staph. aureus, Bacillus cereus, Clostridium perfringens

Table 1: Infectious agents responsible for traveller's diarrhoea.

In this specific case, the time course makes viral agents unlikely and the patient has already completed a course of treatment which would adequately cover most bacterial agents. However, in SE Asia Campylobacter is a common cause of TD and antibiotic resistance is becoming an increasing problem, particularly in this region – this would be the most likely causative agent.

TRAVELLERS' DIARRHOEA

Melissa Smith

What would you do now?

Most TD is self-limiting and the most important aspect to treatment is to ensure that the patient maintains adequate hydration and electrolyte balance. However, in adults TD is very rarely dehydrating and most patients will be able to maintain their fluid/electrolyte balance without difficulty. These patients seek treatment, not due to life-threatening dehydration but in order to alleviate their symptoms and to get back to their travel or other plans as soon as possible.

The severity and duration of attacks can be reduced by the administration of antibiotics and anti-diarrhoeal agents (such as loperamide) and the optimal strategy is to treat with both in combination. Several studies have shown that their actions are synergistic. There are some concerns about the use of loperamide in the presence of entero-haemorraghic (0157 shiga-like toxin producing) E. coli and the American guidelines suggest avoiding anti-diarrhoeals if there is significant blood in the stools. The recommended strategy for most cases of TD, however, would be to treat with both agents together to restore the patient to health as soon as possible.

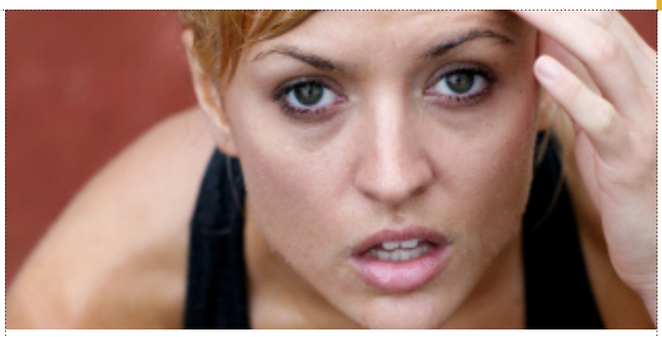
Ciprofloxacin would be the most appropriate first line treatment for most patients presenting with TD. However, our patient has already attempted this approach and in view of the probable diagnosis of fluoroquinolone-resistant Campylobacter the most appropriate antibiotic in this instance would be azithromycin 1g stat or 500mg od for 1–3 days, in combination with loperamide.

Although most guidelines suggest empirical treatment based on history alone, since this patient has already effectively failed this approach, it would also be appropriate to send a stool sample for microscopy, culture and sensitivity (MC&S).

The stool sample is not diagnostic. Despite taking the prescribed antibiotics the patient's diarrhoea and cramps persist and she re-attends the surgery at day 15. What further issues does this raise?

When TD lasts for longer than 2 weeks it is considered "persistent". This affects between 2–18% of those experiencing TD. In this situation parasitic causes should be sought and stool samples sent for "ova, cysts and parasites". However, other causes should be considered. Since the patient has now taken two courses of antibiotics, Clostridium difficile superinfection is a possibility. There have also been some reports of azithromycin-resistant campylobacter from this region.

If the diarrhoea continues beyond 4 weeks the patient should be considered to have chronic diarrhoea. Some series suggest that up to 10% of those exposed to TD go on to develop post-infective irritable bowel syndrome. It is also possible, of course, that some other gastrointestinal illness (such as inflammatory bowel disease or coeliac disease) has coincidentally presented while the patient was abroad.



The patient asks you what she can do to prevent such a problem recurring on future trips. What do you advise?

Preventing TD starts with food hygiene education and the selection of safe food and drink. While it has been difficult to prove that this directly translates to a reduction in TD, there is good evidence that those individuals that make the most "errors" in food selection are at the highest risk of TD. Additionally Jamaican authorities have undertaken a widespread public health programme enforcing high food hygiene standards in hotels and restaurants throughout the island, which has successfully reduced the incidence of TD among travellers there, confirming that this is an effective way to address the problem.

Table 2 lists those foods which should be avoided in high-risk areas. Water and ice are less common sources of TD than food, and a couple of ice cubes in a drink or the use of tap water for brushing teeth is generally considered safe. However, tap water from high-risk areas should not be drunk, even in those hotels advertising that they have a water filter. When purchasing bottled water, always ensure that the seal has not been broken. Patients staying in expensive resorts often assume that the food there will be safe and not subject to the usual dietary constrictions recommended for that region, however the evidence does not support this.

Undercooked meat and shellfish
Moist food served at room temperature, such as salad, fruit which cannot be peeled
Items kept warm on a buffet without a flame underneath and hot tabletop sauces
Unpasteurised milk and cheese
Tap or unsealed bottled water (even in hotels with a water filter)
Street vendors (although food cooked to order and served piping hot may be safe)

Table 2: Food and drink items which are considered high risk for TD and should be avoided.

TRAVELLERS' DIARRHOEA

Melissa Smith

Vaccination against TD is currently the subject of much interest and several different agents are being developed. Vaccines exist against rotavirus, hepatitis A, typhoid and cholera but it is not yet possible to develop one vaccine that will cover all causes of TD. Vaccine development is currently directed against enterotoxigenic E. coli as the most common cause of TD. However, although some of the colonisation factors which create immunity may provide cross-reactivity against other species, it is suggested that, even at best, such a vaccine could expect to prevent 7% of TD episodes.

Although some studies have shown a mild protective effect (up to 20%), trials of probiotics in travellers have, in meta-analysis, failed to demonstrate a reduction in the rate or severity of TD. There is evidence that, particularly in children, a strain specific benefit from *Lactobacillus caseii GG, Lactobacillus reuteri and Saccharomyces boulardii* exists in reducing the duration of attacks of infectious diarrhoea, particularly due to rotavirus. However, in trials specifically aimed at TD, benefit remains unproven. Prebiotics have also proven disappointing with no successful clinical trials to date against TD.

Non-antibiotic agents can be helpful, particularly bismuth sulphate (263mg four times a day) which has been shown to provide 62–65% protection against an attack of TD. However, this agent can cause side effects. Patients report a black tongue, which may be preventable by regular brushing of teeth and tongue. Bismuth has also been associated with mild tinnitus and black stools.

Antibiotics are known to be an effective way to prevent attacks of diarrhoea and historical series have shown protection rates of up to 85%. These drugs are not, however, free from there own problems. Many interact with other medication and all have the potential to cause side effects. Additionally, the extensive adoption of prophylactic antibiotics has been associated with the development of increasing numbers of resistant bacterial strains. As the importance of this has become more apparent, the use of antibiotics for TD prophylaxis has fallen out of favour.

However, work in this area has recently been rejuvenated as rifaximin, (a non-absorbed antibiotic) has been successfully demonstrated to prevent TD (72% protection, 200mg bd) while causing no more side effects than a placebo and being free from drug interactions (including the combined oral contraceptive pill). Thus far clinically relevant resistance to rifaximin has not been observed and it is hoped that, as it is not absorbed it may escape these issues. However, Campylobacter is not susceptible to rifaximin which may limit its usefulness in trips to SE Asia.

While they are known to be effective, the use of other antibiotics as prophylaxis against TD is recommended only in specific circumstances. First, there are patients where the medical consequences of infection could be severe (such as those with ileostomies, severe intercurrent medical conditions or immunosuppression). Second, there are trips of high importance (Olympic athletes, politicians, etc.). Then there are those who may be at higher risk of catching TD (the immunosuppressed and those with gastric acid suppression). There is increasing evidence that some people have a genetically determined increased susceptibility to TD. At its most basic, those with blood group O are more susceptible to cholera and norovirus but now specific genetic markers are being uncovered, for example, an SNP in the IL-8 promotor has been found to confer susceptibility to enteroaggregative E. coli.

If a patient does not fall into any of these categories, accepted opinion is that the risks of antibiotic prophylaxis both to the individual (side effects, drug interactions) but, even more importantly, the community are in favour of adopting an alternative strategy. These individuals should be recommended to use early empirical treatment rather than prophylactic antibiotics and be provided with a 3-day course of ciprofloxacin 500mg bd (some studies even suggest that one 750mg dose is effective), loperamide and advice about food hygiene. The use of this empirical treatment has been shown to reduce the severity of attacks and cut 20–65 hours off their duration. Those moving to a high-risk area for a prolonged length of time are better off avoiding prophylactic antibiotics as, without them, they will acquire natural immunity to the local pathogens and their risk of developing diarrhoea will steadily fall until it reaches that of the local population.

TRAVELLERS' DIARRHOEA

Melissa Smith

What are the potentially serious medical sequelae of an episode of TD?

While most episodes of TD are rapidly self-limiting, up to 18% of patients experience long-term symptoms. Ten per cent develop a true post-infective irritable bowel syndrome, a proportion of which will be seriously debilitating. Additionally, there are the rare but more serious complications of gastrointestinal infection, such as Guillain-Barré syndrome, post-infective arthropathy (both associated with Campylobacter infection) and haemolytic uraemic syndrome (following an attack of enterohaemorrhagic E. coli).

What guidelines are available to aid our treatment of TD?

There are currently no BSG or NICE guidelines about the treatment of TD. However, there are American guidelines (drawn up by the American College of Gastroenterology and the American Society of Infectious Diseases). These are available at **www.idsociety.org/**

Questions

1. An 18-year-old male returning from a gap year trip to Central America presents to A&E with a 2-day history of severe diarrhoea and abdominal pain. On examination his pulse and blood pressure are normal with no postural drop. Abdominal examination reveals only a mild generalised tenderness. What would be the best course of action?

a. culture stool and await result before prescribing.
b. prescribe empirical ciprofloxacin 500mg bd for 3 days with loperamide.
c. recommend oral rehydration solution and loperamide.
d. prescribe bismuth sulphate 526mg qds for 3 days with loperamide.
e. prescribe azithromycin 1g stat and a course of loperamide.

2. A 38-year-old lady attends your practice on return from her holiday to Egypt. She reports that she has had diarrhoea with no other symptoms for 3 days. She is given a 3-day course of ciprofloxacin and a sample sent for MC&S. The stool sample is culture negative and her symptoms are slowly improving.

a. She is likely to have fluoroquinolone-resistant Campylobacter and should be given azithromycin 1g stat.
b. This means the attack was most likely to be viral.
c. The cause of her attack is still most likely to be bacterial and no further action is required.
d. She should be investigated for a non-infectious cause of her diarrhoea.
e. She has a 20% chance of developing post-infective irritable bowel syndrome.

3. Regarding TD, which of the following statements are true or false?:

a. Haemolytic-uraemic syndrome is a most commonly associated with campylobacter infection.
b. Ciprofloxacin should be recommended as prophylaxis for the average traveller to high-risk areas for TD.
c. Up to 18% of those who experience TD suffer persistent symptoms.
d. Drinking unsafe water is the commonest source of TD.
e. Bismuth sulphate is as effective as antibiotics at preventing TD.

4. True or False, the following are considered high risk for developing TD:

a. A cocktail containing a small amount of ice.
b. Hamburgers in a local restaurant.
c. Meat curry from the hotel buffet.
d. Table top sauces.
e. Cleaning your teeth in the hotel's tap water.

Answers

1. b – ciprofloxacin and loperamide.

Empirical antibiotics and anti-diarrhoeals in combination are likely to provide the best symptomatic improvement. Although rehydration is an important consideration, it is very rare for an adult to dehydrate due to TD and this patient has no evidence to suggest that he has not managed to maintain adequate hydration. Bismuth is more commonly used to prevent attacks of TD, it can be used to treat attacks but is less effective that antibiotic treatment and has side effects. Azithromycin should be used as a second line agent and he has not been travelling in an area where ciprofloxacin resistance is prevalent. Sending stool cultures would be appropriate if this first line treatment fails.

2. c – the attack is most likely to be bacterial, specifically due to enterotoxigenic E. coli and should have been treated by her course of ciprofloxacin. No further action is required unless her symptoms are still troubling her after 2 weeks. The incidence of post-infective IBS is 10%.

3.
a. F – HUS is most commonly caused by E. coli 0157 but can also be associated with shigella, salmonella, yersinia and campylobacter
b. F **c.** T **d.** F **e.** F

4.
a. F **b.** T **c.** T **d.** T **e.** F

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PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG)

Nasser Khan and Steven Mann

A 79-year-old lady presented to the A&E with sudden onset dysarthria, dehydration and left-sided weakness.
Good Medical Practice.

Case Report

A 79-year-old lady presented to the A&E with sudden onset dysarthria, dehydration and left-sided weakness. She had a history of previous transient ischaemic attacks and hypertension. She lived alone and was self-caring with a supportive family. On examination she had a dense left hemiplegia and dysarthria. A CT scan confirmed an early right cerebral hemisphere infarct and she was transferred to the stroke ward. Over the next 48 hours, the patient was assessed by the speech and language therapist who concluded that, as a result of her stroke, she was at significant risk of aspiration from her impaired swallow. A nasogastric feeding tube was passed and feeding commenced. Two weeks later, she was clinically stable with little signs of recovery and was still dysphagic and dysarthric with a hemiplegia. She remained at risk of aspiration according to the speech and language therapist assessment.

What are the indications for PEG placement?

PEG for enteral feeding has been used since 1980[1]. Conditions in which a PEG may be appropriate are numerous but can generally be classified as follows:

1. Neurological swallowing disorders – cerebrovascular accident (CVA), multiple sclerosis, motor neurone disease, Parkinson's disease.
2. Cognitive impairment and depressed consciousness – head injury, brain injury.
3. Mechanical obstruction to swallowing – oropharyngeal or oesophageal cancer.
4. Long-term partial failure of intestinal function requiring supplemental intake – short bowel, fistulae, cystic fibrosis.

The PEG route has a number of advantages over nasogastric tube feeding including:

1. More comfortable.
2. Less unsightly and less stigmatising.
3. Less prone to becoming displaced.
4. A theoretically slightly lower risk of aspiration than NG feeding although NEITHER protects against aspiration of vomited feed[2].

What are the contraindications to PEG placement?

Contraindications to placing a PEG include any contraindication to having an upper GI endoscopy.

Absolute contraindications:
obstructing lesion of the upper GI tract impeding passage of the endoscope.
coagulopathy (INR>1.4, plt <100) [3]
gross ascites
oesophageal varices
gastric malignancy
gastric outlet obstruction
non-functioning GI tract
life expectancy < 30 days.
Relative contraindications:
morbid obesity
hepatomegaly
previous abdominal/gastric surgery
acute coronary syndrome within 3 months of procedure
intercurrent chest infection
respiratory compromise (pO2 sats <90%)
active gastric ulceration
severe gastro-oesophageal reflux (risk of aspiration)
late pregnancy.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG)

Nasser Khan and Steven Mann

When should a PEG be inserted?

Typically artificial nutrition support is needed when oral intake is absent or likely to be absent for a period of 5–7 days, initially as nasogastric feeding. PEG feeding should be considered for any patient who is unable to meet their nutritional needs via the oral route and who is likely to require artificial nutrition support for more than 4 weeks.

According to the British Society of Gastroenterology guidelines on enteral nutrition, health care professionals should aim to provide adequate nutrition to every patient unless prolongation of life is not in the patient's best interests[4].

'Situations in which artificial nutrition support are not appropriate:

1. The prognosis is so poor that survival beyond a few weeks is unlikely.
2. The patient refuses treatment and is legally competent to do so.
3. The patient has no capacity but has made a valid, clear and unambiguous advance directive that artificial nutrition support be withheld.
4. The patient has lost capacity, but PEG feeding would prolong a life that is "demonstrably awful" due to pain and distress.

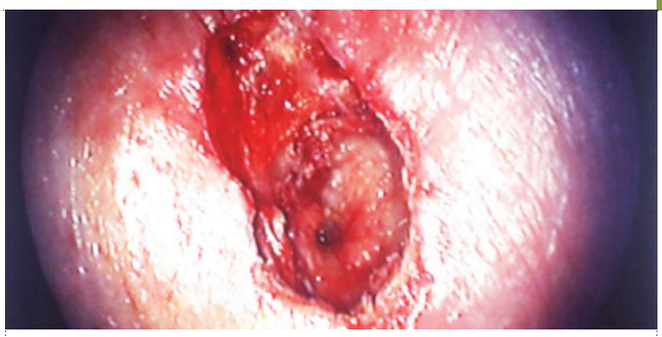
In the case of a stroke patient, as in all patients, tube feeding should never be started without consideration of all related ethical issues and must be in the patient's best interests. In the eyes of the law, tube feeding is considered a medical treatment.

Therefore starting, stopping and withholding PEG feeding is subject to the same medical decision-making processes as any other medical intervention, such as dialysis, ventilation, chemotherapy, etc. In stroke medicine, PEG feeds do not affect cerebral recovery and one-third of patients discharged from hospital die within 1 year.

Some patients do recover their swallow function after discharge, so regular reviews of swallow and nutritional needs are essential.

Who should be involved in the decision to PEG feed a patient?

Decision-making regarding artificial nutrition support is best made by the patient's own medical team in conjunction with a multidisciplinary nutrition team which can include a gastroenterologist, nurse specialist, dietician and speech and language therapist. Relatives and carers and also the GP should also be incorporated into the discussions, particularly when the decision to feed via the PEG route is not so straightforward.



What are the complications of a PEG insertion?

The risks/complications of PEG placement include those general hazards associated with any upper GI endoscopy:

1. Oversedation/respiratory depression.
2. Aspiration.
3. Perforation (although this is technically a necessary component of PEG) and subsequent peritonitis.
4. Haemorrhage.

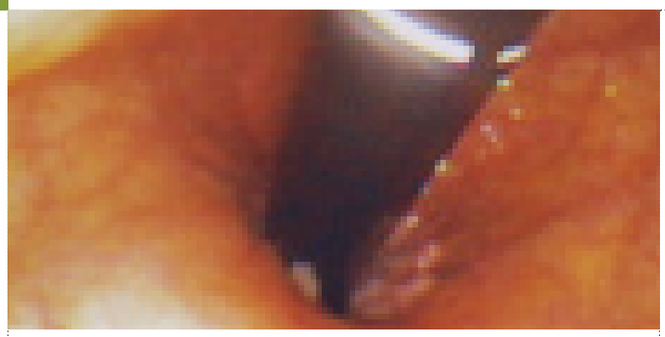
Minor complications:
wound infection
peristomal leakage
pneumoperitoneum
ileus
bleeding
ulceration
clogging of tube
tube dysfunction
hypergranulation
Major complications:
necrotising fasciitis
buried bumper syndrome
colocutaneous fistula
peritonitis
inadvertent PEG removal
death

Major complications occur in about 3% of cases and direct procedure-related mortality can occur in 0.7–2%[3].

Independent risk factors which portend a poor prognosis in PEG placement include older age, low serum albumin, dementia, aspiration pneumonia and co-morbidities (e.g. sepsis, neoplasm, cardiac failure).

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG)

Nasser Khan and Steven Mann



How do I prepare the patient for a PEG and how is it done?

Once the team (patient, clinicians, MDT, family) have reached a decision to insert a PEG, there is a preprocedure checklist that is necessary.

1. The patient has consented or has had a Consent Form 4 (for those without capacity to give consent) completed by the referring team.
2. The haemoglobin is >9g/dL.
3. The platelets are >100.
4. The INR is 1.4 or less.
5. Clopidogrel should have been stopped for 1 week preprocedure. There is no evidence that aspirin needs to be discontinued.
6. Stop warfarin for 3 days before and check INR immediately preprocedure.
7. Discontinue any subcutaneous fluids into the abdominal wall.
8. Consider refeeding syndrome risks.
9. Ensure that oral/nasogastric feeds are stopped at least 6 hours preprocedure.
10. The patient is cannulated for prophylactic antibiotics and sedation.
11. The patient can lie flat, safely be sedated and tolerate no more than 2 litres of oxygen via nasal cannulae.

• During the procedure the patient is very lightly sedated and prophylactic antibiotics are given.
• A standard diagnostic gastroscopy is performed in all cases and then the stomach is insufflated with air.
• A combination of transillumination and digital pressure on the abdominal wall helps to identify the optimal site for puncture.
• Using aseptic technique, the site is infiltrated with lignocaine and a trochar is inserted through the skin of the abdomen and directly into the gastric cavity.
• A string is passed through this trochar which is grasped by forceps or a snare inserted through the endoscope.
• The endoscope is withdrawn with the string and the end of the string is tied to the end of the feeding tube.
• This is then pulled down into the stomach, out through the trochar site and eventually secured.
• The PEG can now be used.

A video of the procedure can be seen at
http://uk.youtube.com/watch?v=Jfx_QbRr5Z0&feature=related

How do you assess capacity to consent for the procedure?

A patient has capacity if they can understand, retain, use and weigh up the information needed to make a decision, and can communicate their wishes[5].

You must assess a patient's capacity to make a particular decision at the time it needs to be made.

You must take account of The Mental Capacity Act 2005 and other relevant guidance.

If you are in any doubt about the patient's capacity, you should seek advice from nursing staff, carers or relatives and colleagues with relevant specialist experience in these matters.
If you are still in doubt about the patient's capacity to make a decision, you must seek legal advice.

What are the principals of the Mental Capacity Act 2005?

The Mental Capacity Act 2005 came into force partly in April 2007 and in its entirety in October 2007. The act protects people who lack the capacity to make decisions. It is underlined by five key principals[6]:

1. Capacity should always be assumed. A patient's diagnosis, behaviour or appearance should not lead you to presume that capacity is absent.
2. A person's ability to make decisions must be optimised before concluding that capacity is absent. All practical steps must be taken, such as giving enough time for assessments, repeating the assessments if fluctuating capacity, using interpreters, sign language, etc.
3. Patients are entitled to make unwise decisions. It is not the decision but the process by which it is reached that determines if capacity is absent.
4. Decisions made for people who lack capacity must be in their best interests.
5. Such decisions must be the least restrictive for their basic rights and freedoms.

When would you involve an IMCA (Independent Mental Capacity Advocate)?

Contacting an advocate is advisable if the patient who lacks capacity is "unbefriended" and in need of:

• serious medical treatment (ventilation, major surgery, discontinuation of artificial nutrition and hydration).
• hospital accommodation for more than 28 days
• placement in a care home for more than 8 weeks
• a change in a care home or hospital is being planned.

Their recommendations do not need to be adhered to by clinicians, although they should be taken into account as part of the decision-making process.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG)

Nasser Khan and Steven Mann

How should you manage a blocked PEG tube?

If the tube is blocked try and administer 50ml warm water down the tube and leave for 30 minutes (3/4 of a cup of tap water and 1/4 of a cup of boiling water). This should dissolve any medication or soften any feed plugging the tube.

If unsuccessful try to administer 50ml of fizzy water and leave for 30 minutes. Do not use coke, pineapple juice or cranberry juice as these agents are highly acidic and can coagulate the feed formula and increase the risk of blockages.

If the tube is still blocked consider the use of a proprietary product which contains digestive enzymes which may break down the protein plug. Pancreatic enzymes or "clog zapper" can be used.

Do not use a syringe smaller than 50ml to avoid the tube being ruptured with the high pressure of a smaller syringe.

Never attempt to push any guidewires or sharp objects down the tube.

If all these measures are unsuccessful, the PEG will need replacing.

How should you manage a patient whose PEG tube fell out?

Accidental removal of PEG tubes is more common with balloon retaining devices where the internal balloon punctures preventing the tube from being held in place. 'A PEG tract is usually mature in 7–10 days but this maturation process can be delayed for up to 4 weeks, particularly if the patient is malnourished or is on corticosteroids. Once the PEG is out, an immature tract can close very rapidly with 6–12 hours, but usually takes up to 24 hours'.

If the PEG tube came out more than 3–4 weeks after placement (i.e. when the tract is mature) preservation of the original tract is crucial and should really only be attempted by a practitioner suitably trained and competent in this procedure. To prevent tract closure, a soft 12 Fr Foley catheter can be gently passed into the tract and the balloon inflated according the manufacturers instructions until a replacement PEG can be placed. Correct position can be confirmed by aspiration of the gastric contents and checking that pH is <5.5 (if not on PPI/H2RA). Alternatively position can be confirmed by injecting water soluble contrast via the PEG to clarify its position.

A PEG tube that is accidentally removed within 4 weeks of insertion must be replaced endoscopically or radiologically. No attempt should be made to place a Foley catheter or to reinsert the tube as the risk of inducing peritonitis is high.

Discussion

Dysphagia in neurological disorders is the most common indication for artificial nutrition support via the PEG route. Dysphagia is present in 23–50% of all stroke cases admitted to hospital. Dysphagia is an independent major risk factor for malnutrition. Malnutrition is associated with a poor outcome in stroke patients with increased mortality, worse functional outcome, increased risk of pressure sores and increased length of stay. However, nutritional supplementation in this population remains controversial.

The FOOD (Feed Or Ordinary Diet) trials were three large international multicentre randomised controlled trials involving 18 countries that were designed to answer key questions about feeding in hospitalised stroke patients[7].

1. Does routine oral nutritional supplementation of a normal hospital diet improve the outcome after stroke?

4,023 patients were enrolled in 15 countries and randomised to a regular diet or a regular diet plus oral supplements. At 6 months there was no difference in survival or functional outcome between the two groups. This suggests that routine nutritional supplements are not necessary in adequately nourished stroke patients who can swallow.

2. Does early tube feeding improve the outcomes in dysphagia stroke patients?

859 patients were enrolled in 15 countries and randomised to early enteral feeding (via nasogastric or PEG) or no feeding within seven days of the stroke. There were only non-significant trends towards a reduced mortality in the early fed group at 6 months.

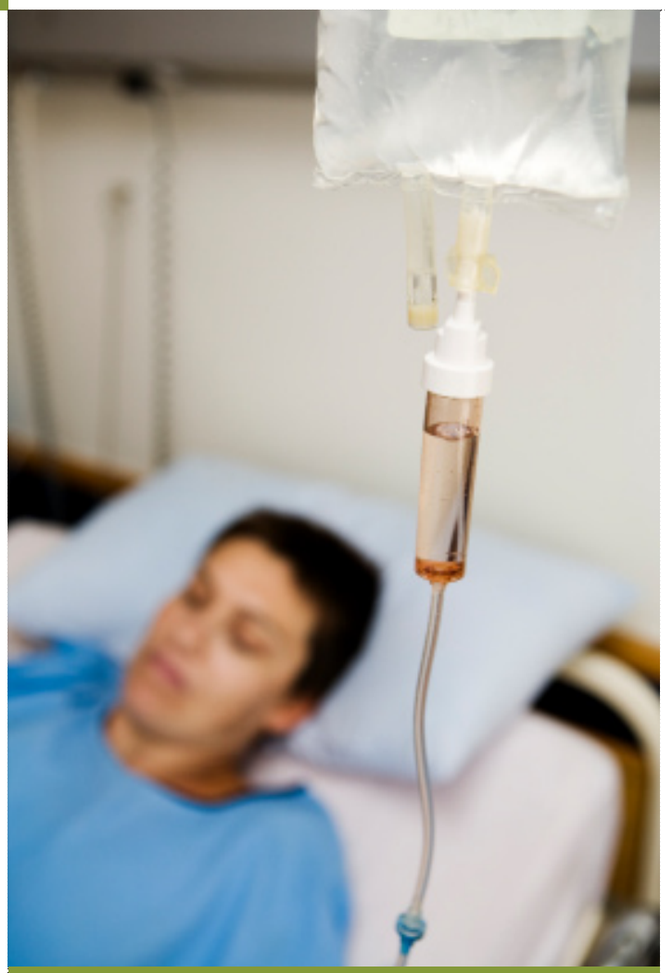
3. Does tube feeding via a PEG result in better outcomes than that via nasogastric tube?

321 patients in 11 countries with unsafe swallow were randomised to PEG or nasogastric feeding. There were non-significant trends towards increased mortality in the PEG fed group at 6 months.

In conclusion the evidence is still not definitive regarding the timing and route of feeding in stroke patients, although early enteral feeding may be beneficial if dysphagia is present. It seems logical to hydrate the patient with intravenous fluids during the first few days while observing the swallowing function and taking the time to discuss tube feeding issues with the patient and the family. Then feed via the nasogastric route if dysphagia is still present switching to PEG after 3 weeks if long-term feeding is still needed.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG)

Nasser Khan and Steven Mann



A patient with a long-term PEG attends A&E. His carer says that the PEG fell out just a few hours ago and nobody brought the displaced PEG with the patient. The patient cannot manage anything by mouth.
Good Medical Practice.

PEG Questions

1. A patient with a long-term PEG attends A&E. His carer says that the PEG fell out just a few hours ago and nobody brought the displaced PEG with the patient. The patient cannot manage anything by mouth.

What is the next thing you should do?
a. Admit the patient with IV fluids and discuss with the gastroenterology team in the morning.
b. Send the patient home and plan to inform the gastroenterologists in the morning.
c. Start a nasogastric feed.
d. Carefully insert a Foley catheter into the PEG track and inflate the balloon; then admit the patient for gastroenterology review in the morning..

2. Following a stroke, a patient who has been fed via the nasogastric route for a few weeks needs a PEG but has severe lung disease and would not tolerate sedation without respiratory compromise.

What is the next step?
a. A surgical gastrostomy with general anaesthetic.
b. An endoscopic procedure with just throat spray.
c. A radiologically inserted gastrostomy (RIG).
d. Long-term nasogastric feeding.

3. A patient with a recently inserted PEG suffers from recurrent aspiration of feed despite prokinetics and upright position during feeds. They are known to have diabetic gastroparesis.

What would you do next?
a. Ask for a surgical opinion with a view to performing a surgical jejunostomy.
b. Remove the PEG.
c. Restart NG feeding.
d. Request that a PEG-J is inserted (PEG with a jejunal extension).

4. A patient with a dense cerebrovascular accident cannot tolerate a nasogastric feed and keeps vomiting the tube up despite antiemetics. The multidisciplinary team has recommended PEG feeding. The patient has residual confusion following the CVA but is otherwise stable and rehabilitating well. Her daughter says that she has no objection to a PEG being inserted but says that her mother often commented that she would "not want to be kept alive" if she was very debilitated.

What is the right thing to do?
a. After discussion with the family and ward staff, recommend that nutrition is withdrawn in accordance with what you have heard from the daughter.
b. After conferring with the multidisciplinary team, explain to the daughter that her mother is doing well in other respects but needs a PEG to maintain her nutritional status.
c. Persist with nasogastric feeding.
d. Start parenteral nutrition.

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG)

Nasser Khan and Steven Mann

5. You are looking after a patient with an established PEG on the ward. On your ward round you notice that the PEG site is becoming progressively more tender, red and inflamed but otherwise the PEG is working well. An ultrasound reveals no collections and the patient is well apart from a low grade pyrexia and a CRP of 30.

What should you do next?

a. Start parenteral antibiotics for cellulitis but continue PEG feeding.
b. Start parenteral antibiotics but stop PEG feeding and start a nasogastric feed.
c. Remove the PEG.
d. Apply local antiseptic cream .

Answers

1. The correct answer is d.

If the PEG track is mature then it should be kept open with a Foley catheter. A replacement balloon PEG can then be inserted without the need for an endoscopy or fresh incision.

2. The correct answer is c.

If a sedated endoscopy is contraindicated then it is reasonable to perform a RIG. The stomach is insufflated via the nasogastric tube and then the skin is punctured under fluoroscopic guidance. This allows the insertion of a feeding tube directly into the stomach. RIGs are more prone to displacement and thus usually less commonly performed than PEGs.

3. The correct answer is d.

A PEG-J is relatively straightforward once a PEG is in situ. Essentially, a feeding tube is introduced into the stomach via the PEG and this tube is endoscopically guided into the jejunum. Any subsequent feed bypasses the stomach.

4. The correct answer is b.

Although the daughter's comments may be correct, this does not constitute a written advance directive. Under these circumstances you must act in the best interests of the patient who cannot give or withhold consent. Of course, this must be explained with care and sensitivity to the daughter.

5. The correct answer is a.

The patient is likely to have local cellulitis and with the pyrexia and high CRP this is likely to require parenteral antibiotics. There is no reason to stop using the PEG during this time, however, the patient will do better if they are well nourished.

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CASE BASED INVESTIGATION: ERCP AND CHOLANGIOCARCINOMA

LA Possamai and Shahid A Khan

A 74-year-old Caucasian woman is referred to hospital on the acute medical take by her GP. She reports a 3-week history of vomiting associated with unintentional weight loss of 3–4kg.
Patient Management.

Case history

A 74-year-old Caucasian woman is referred to hospital on the acute medical take by her GP. She reports a 3-week history of vomiting associated with unintentional weight loss of 3–4kg. Four days earlier the patient's daughter had commented on a yellow discolouration to her skin and eyes, which had since become more pronounced. The patient denied abdominal pain. She commented that her appetite had been poor for a few weeks and she was eating very little. She had noticed dark discoloration of her urine 1 week earlier and when directly questioned admitted her stools had been pale in colour and offensive smelling. She had never experienced similar symptoms in the past.

Past medical history included type II diabetes mellitus, hypertension, hypercholesterolaemia and ischaemic heart disease. She had undergone elective coronary stenting for angina 2 years previously. Her medication included aspirin, metformin, bendroflumethiazide, ramipril, atenolol and simvastatin. There had been no recent changes to her medication. She took a daily fish oil supplement but no other over the counter medication.

The patient was a retired secretary; she lived alone in a one bedroom flat, having been widowed 4 years earlier. She was independent in all activities of daily living, had quit smoking 30 years ago and had a 20 pack year smoking history. She drank 4–8 units of alcohol/week.

On examination she was afebrile with a BP 105/60 and a regular tachycardia of 105 beats per minute. She had icteric sclearae and dry mucous membranes. There were no stigmata of chronic liver disease or lymphadenopathy. Cardiorespiratory examination was normal. Abdominal examination revealed a soft, non-tender abdomen with no masses or palpable organomegaly. Neurological examination and cognition were normal.

Initial investigations

ECG – sinus tachycardia 110 beats per minute. No ischaemic changes.

Plain chest radiograph – clear lung fields, normal cardiac contour, no bone or soft tissue abnormalities.

Blood tests

Sodium 132mmol/L (135–145mmol/L)

Potassium 5.2mmol/L (3.5–5.0mmol/L)

Urea 12.8mmol/L (2.5–6.6mmol/L)

Creatinine 197mmol/L (60–125mmol/L) (Creatinine 108mmol/L 2 months prior to current admission)

Glucose 4.5mmol/L (4–11mmol/L)

Bilirubin 104mmol/L (5–17mmol/L)

Aspartate aminotransferase (AST) 124IU/L (<40IU/L)

Alanine transaminases (ALT) 245IU/L (<40IU/L)

g-glutamyl transpeptidase (gGT) 1408IU/L (<52IU/L)

Alkaline phosphatase (ALP) 413IU/L (30–130IU/L)

Albumin 38g/L (35–51g/L)

Amylase 69U/L (< 68U/L).

C-reactive protein <5mg/L (<5mg/L)

Haemoglobin 10.1g/dL (11.5-15.1g/dL)

White cell count 8.7 x 10^9/L (5.1–11.4 x 10^9/L)

Platelets 376 x 10^9/L (147 – 397 x 10^9/L)

MCV 91fl (84 – 98fl)

International Normalised Ratio (INR) 1.5 (0.9–1.2)"

CASE BASED INVESTIGATION: ERCP AND CHOLANGIOCARCINOMA

LA Possamai and Shahid A Khan

What would be your initial management of this patient?

The clinical history, examination findings and blood results suggest this patient has obstructive jaundice and acute renal failure, complicated by coagulopathy, normocytic anaemia, hyponatraemia and dehydration.

Her bendroflumethiazide, ACE-inibitor and metformin were withheld. She was rehydrated with intravenous crystalloid and a urinary catheter was inserted to monitor urine output. Intravenous vitamin K at a dose of 10mg once daily was administered. She was commenced on oral ciprofloxacin to cover potential cholangitis due to biliary stasis. An anti-emetic was prescribed to treat her nausea and vomiting.

What are the causes of obstructive jaundice?

Obstruction to biliary flow causing the symptoms of jaundice, dark urine, steatorrhoea and pruritis can be caused by a number of different pathologies. It is useful to classify these into three groups: first, obstructing lesions within the lumen of the bile duct; second, those pathologies affecting the bile duct wall; and last, pathology outside the biliary tree that causes obstruction by extrinsic compression.

Intraluminal

Choledocholithiasis

Liver flukes (e.g. *fasciola hepatica)*

Intramural

Cholangiocarcinoma

Primary biliary cirrhosis (PBC)

Primary sclerosing cholangitis (PSC)

Choledochal cysts

Congenital biliary atresia

Extraductal

Carcinoma of the head of the pancreas

Lymphadenopathy at the porta hepatis

Pancreatic pseudocysts

Mirizzi's syndrome

Drugs can also cause cholestasis and an obstructive pattern of liver function tests in the absence of a mechanical obstruction to bile flow. The drugs that most commonly cause this phenomenon are antibiotics, particularly co-amoxiclav, erythromycin, flucloxacillin, fusidic acid and nitrofurantoin. Oral contraceptives, tricyclic antidepressants, irbesartan, prochlorperazine, sulfonylureas and clopidogrel can also be associated with this pattern of liver function test derangement.

What investigation would you order next?

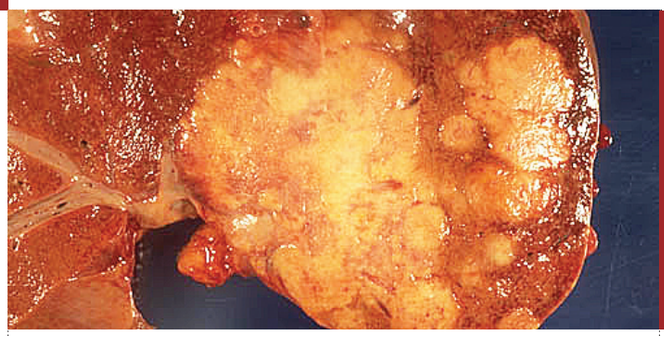
Ultrasound of the abdomen including renal tract was requested, to investigate possible biliary obstruction and exclude an obstructive cause for the acute renal failure.

Ultrasound scan of the abdomen showed an unobstructed renal tract with normal-sized kidneys. Intrahepatic biliary ducts were dilated with a maximum diameter of 12mm. The common bile duct was collapsed. There were no gallstones present and the gall bladder was normal. The liver parenchyma was of normal echotexture with no focal lesions. Doppler studies showed normal direction flow in the portal and hepatic veins.

The ultrasound appearances of dilated intrahepatic ducts and a collapsed CBD are suggestive of obstruction at the level of the liver hilum. This narrows the differential diagnosis to those lesions that can occur in this anatomical position and is particularly suggestive of a hilar cholangiocarcinoma or "Klatskin tumour".

CASE BASED INVESTIGATION: ERCP AND CHOLANGIOCARCINOMA

LA Possamai and Shahid A Khan



This is a cholangiocarcinoma. Presence of a single dominant mass. Patient Management.

What further tests would be helpful?

Tumour markers CA19.9, CEA, CA125 and AFP were sent.

Carbohydrate antigen (CA19.9) 6327 U/mL (<31 U/mL)
CA125 23 U/mL (<34 U/mL)
Carcinoembryonic antigen (CEA) and alpha-fetoprotein (AFP) normal

Magnetic resonance cholangiopancreatography (MRCP) with standard MRI and MR angiography was performed.

MRI – dilated intrahepatic ducts. Hilar tumour with local spread, including encasement of the hepatic artery. Coeliac lymphadenopathy.

What would you tell the patient?

The patient's case and results of the ultrasound, MRI and tumour markers were discussed at the local specialist hepatobiliary multidisciplinary meeting. The findings were highly suggestive of an advanced cholangiocarcinoma. The evidence of lymph node metastatases and hepatic artery involvement meant she was not suitable for surgical resection of the tumour.

Median survival for patients with inoperable hilar cholangiocarcinoma is approximately 3 months without biliary drainage and 4–10 months with biliary drainage. Non-operative biliary drainage is considered as the first choice in such cases.

The patient was informed of her diagnosis and lack of curative options.

What further treatment could be offered to the patient?

The patient was referred to a specialist oncology service for consideration of palliative chemotherapy.

She was offered an ERCP for biliary drainage and to attempt tissue diagnosis.

The MRCP images were used to plan stenting of the patient's hilar stricture. An ERCP was performed and brushings taken for cytology. A metal stent was placed across the stricture.

On the day following ERCP the patient complained of severe epigastric and back pain with associated vomiting. Her serum amylase was elevated at 805U/L (normal range<68U/L) and a diagnosis of post-ERCP pancreatitis was made. She was treated with supportive therapy, IV fluids, anti-emetics and analgesia. Her vomiting settled and serum amylase returned to normal after 5 days. Successful biliary decompression was demonstrated by a fall in her serum bilirubin. Unfortunately she developed a hospital-acquired pneumonia, which further prolonged her admission.

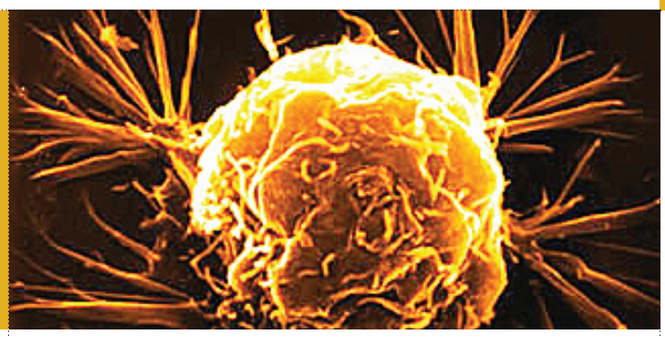
Brush cytology showed adenocarcinoma with cells that express cytokeratins 7 and CEA, consistent with cholangiocarcinoma.

Ultimately the patient declined palliative chemotherapy and was discharged home with follow-up from the community palliative care team.

MANAGEMENT OF COLORECTAL CANCER: FOCUS ON POPULATION SCREENING

Mohammed Nizamuddin and Marta Carpani de Kaski

Mrs CB, a 60-year-old female, presented to her GP with a 4-month history of tiredness and loose stools, having had a long life history of tendency to constipation, and weight loss (6 kg in the past 6 months). She has a family history of cancer though not colonic. Good Clinical Care.



Case study

Mrs CB, a 60-year-old female, presented to her GP with a 4-month history of tiredness and loose stools, having had a long life history of tendency to constipation, and weight loss (6 kg in the past 6 months). She has a family history of cancer though not colonic. Her past medical history was irrelevant. Mrs CB's GP suspected a gastrointestinal malignancy and referred her to the local GI department for investigation.

The Government set targets for NHS Trusts in England and Wales to see patients with suspected cancer within two weeks of an urgent referral by their GP[1] , where "suspected" was defined as either a perceived level of probability or a hunch[2]. This was achieved by the implementation of the Two-Week Rule (TWR) for fast tracking suspected cancer referrals from primary to secondary care[3,4].

NICE Guidelines for Colorectal Cancer (CRC) detection (2004)

Refer urgently patients:

- Aged 40 years and older reporting rectal bleeding with a change of bowel habit towards looser stools and/or increased stool frequency persisting 6 weeks or more IC.

- Aged 60 years and older, with rectal bleeding persisting for 6 weeks or more without a change in bowel habit and without anal symptoms IC.

- Aged 60 years and older, with a change in bowel habit to looser stools and/or more frequent stools persisting for 6 weeks or more without rectal bleeding.

- Of any age with a right lower abdominal mass consistent with involvement of the large bowl IC.

- Individuals of any age with a palpable rectal mass (intraluminal and not pelvic); a pelvic mass outside of the bowel would warrant an urgent referral.

However, even this rapid patient referral may not be enough to tackle the problem early enough. Colorectal cancer is the second most common cause of cancer-related death and the third most common cancer in the UK, affecting more than 30,000 people each year with an average 5-year survival of 40%[5]. Currently, 80% of cases are not diagnosed until the cancer has spread through the bowel wall or beyond[6]. Such cases have a much worse prognosis than cancers confined to the bowel wall. As many patients with CRC do not develop symptoms until the cancer is advanced, as in Mrs CB's case, the detection of a greater proportion of cases at an earlier stage can only be achieved by screening asymptomatic people. Regular bowel cancer screening has been shown to reduce the risk of dying from bowel cancer by 16%[2].

Assessment of Mrs CB in the GI department revealed the presence of anaemia (haemoglobin 10.8g/dL) and a non-tender mass in the right ileac fossa. There were no palpable lymph nodes and PR examination was normal. As these findings were highly suggestive of a malignancy, the patient was referred immediately for a colonoscopy, which confirmed the presence of a caecal tumour (Figure1). Biopsies were taken and histological assessment revealed the tumor to be an adenocarcinoma.

CT scans of the chest, abdomen and pelvis and a PET scan were requested to further characterise the tumour (cancer staging) and assess the patient's risk.

Findings: multiple liver metastases (Figure 2)

Colon cancer staging is an estimate of the amount of penetration of a particular cancer. It is performed for diagnostic and research purposes, and to determine the best method of treatment. The systems for staging colorectal cancers largely depend on the extent of local invasion, the degree of lymph node involvement and whether there is distant metastasis[7].

Definitive staging can only be done after surgery has been performed and pathology reports reviewed. An exception to this principle would be after a colonoscopic polypectomy of a malignant pedunculated polyp with minimal invasion. Preoperative staging of rectal cancers may be done with endoscopic ultrasound. Adjuncts to staging of metastasis include Abdominal Ultrasound, CT, PET Scanning and other imaging studies.

MANAGEMENT OF COLORECTAL CANCER: FOCUS ON POPULATION SCREENING

Mohammed Nizamuddin and Marta Carpani de Kaski

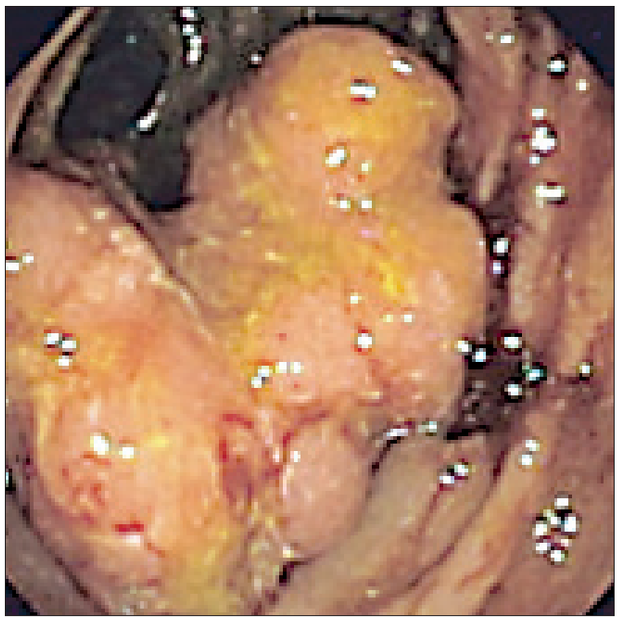


Figure 1: Caecal tumour.

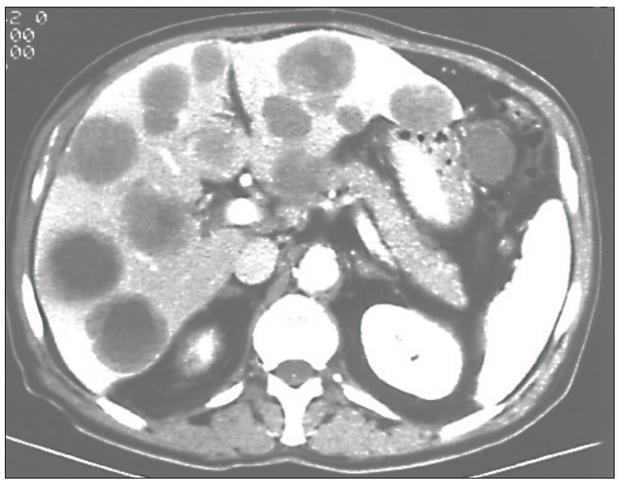


Figure 2: Multiple liver metastases.

Staging systems

1. Dukes classification

First proposed by Dr Cuthbert E. Dukes in 1932, identifies the stages as:

- A – Tumour confined to the intestinal wall
- B – Tumour invading through the intestinal wall
- C – With lymph node(s) involvement (this is further subdivided into C1 lymph node involvement where the apical node is not involved and C2 where the apical lymph node is involved)
- D – With distant metastasis

2. TNM system

The most common current staging system is the TNM (for tumors/nodes/metastases) system, though some places still use the older Dukes system. The TNM system assigns a number7:

- T – The degree of invasion of the intestinal wall
  - T0 – no evidence of tumor
  - Tis – cancer in situ (tumor present, but no invasion)
  - T1 – invasion through muscularis mucosa into submucosa
  - T2 – invasion through submucosa into the muscularis propria (i.e. proper muscle of the bowel wall)
  - T3 – invasion through the muscularis propria into subserosa but not to any neighbouring organs or tissues
  - T4 – invasion of surrounding structures (e.g. bladder) or with tumour cells on the free external surface of the bowel
- N – the degree of lymphatic node involvement
  - N0 – no lymph nodes involved
  - N1 – one to three nodes involved
  - N2 – four or more nodes involved
- M – the degree of metastasis
  - M0 – no metastasis
  - M1 – metastasis present
  - Mx – distant metastases cannot be assessed

3. AJCC (American Joint Committee on Cancer) stage groupings

The stage of a cancer is usually quoted as a number I, II, III, IV derived from the TNM value grouped by prognosis; a higher number indicates a more advanced cancer and likely a worse outcome.

- Stage 0 - Tis, N0, M0
- Stage I - T1, N0, M0 / - T2, N0, M0
- Stage IIA - T3, N0, M0
- Stage IIB - T4, N0, M0
- Stage IIIA - T1, N1, M0 / - T2, N1, M0
- Stage IIIB - T3, N1, M0 / - T4, N1, M0
- Stage IIIC - Any T, N2, M0
- Stage IV - Any T, Any N, M1

MANAGEMENT OF COLORECTAL CANCER: FOCUS ON POPULATION SCREENING

Mohammed Nizamuddin and Marta Carpani de Kaski

Bowel cancer – survival rates by stage at diagnosis

Patients who are diagnosed at an early stage have a much better prognosis than those who present with more extensive disease (Table 1). While patients with a tumor stage A have an excellent survival rate, those in stage D have a very low survival expectancy at 5 years of follow-up (Table 1).

Dukes' Stage Modified	Approximate Frequency At Diagnosis	Approximate Five-Year Survival
A	11%	83%
B	35%	64%
C	26%	38%
D	29%	3%

Table 1: Approximate frequency and 5 year relative survival (%) by Duke's stage.

Based on the above staging systems, Mrs CB's tumour was considered to represent Stage IV. As per standard practice, this patient's case was discussed at the hospital's MDT to decide on the appropriate therapeutic strategy.

MDT suggestions:

1. Proceed to surgery: Right hemicolectomy

Resection of the liver metastases was not an option in Mrs CB's case given their large number and location.

2. Chemotherapy: The role of palliative chemotherapy was discussed in terms of intended benefits and potential side effects. The patient declined the use of any adjuvant therapy. Instant contact with the MacMillan care was made through the MDT.

Evidence suggests that a 6-month course of intravenous chemotherapy following surgery significantly reduces the chance of colon cancer recurring and improves 5-year survival by 5–6%[8]. Chemotherapy should be made available to patients following surgery for Dukes' stage C if they are well enough to tolerate it; patients with metastatic or locally inoperable primary cancer (stage D) require careful evaluation, and may be appropriate for palliative chemotherapy and/or radiotherapy.

Surgery was carried out uneventfully. Material was sent to the pathologists who reported the tumour to be a moderately differentiated adenocarcinoma of the caecum involving the subserosal fat, 4 out of 12 nodes were involved. Tumour was therefore classified as: Stage T3N1Mx (See previous staging description) (See Figure 3)

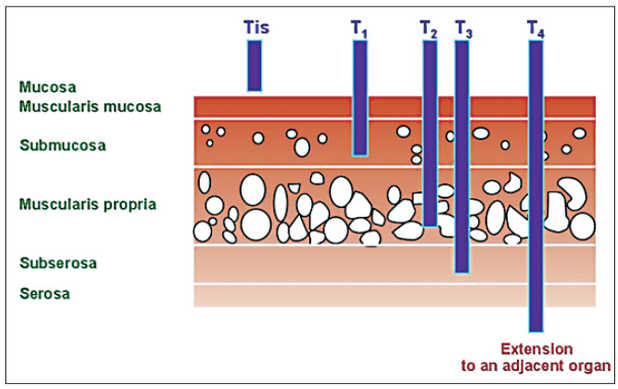


Figure 3: Colorectal cancer – TNM classification and definition of primary tumour (T).

Mrs CB was managed in accordance to guidelines set up by NICE recently.

In 1997, the Department of Health published a document called Improving Outcomes in Colorectal Cancer. NICE has now published an updated version for the NHS in England and Wales. Some of the original recommendations have been updated, and further recommendations have been added[8].

The key recommendations are:

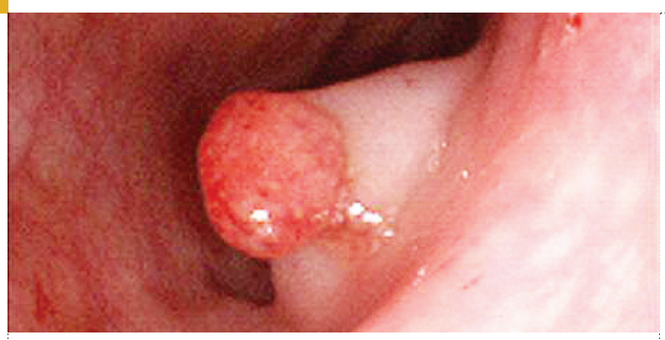
- people who may have colorectal cancer should be offered rapid referral for endoscopy
- endoscopy should be available for diagnosis
- people should be treated by a multidisciplinary team
- colorectal teams treating people with rectal cancer should have special training
- people who need emergency treatment should be treated by a colorectal cancer team
- information and support should be appropriate.

Clinical outcome: The patient had an uneventful post-operative recovery and was discharged home. She continued to be monitored by the oncology team at her hospital but unfortunately 4 months after surgery she became unwell and was readmitted. She was then complaining of intense abdominal pain. A further PET scan was performed and multiple disseminated metastases were detected. She again refused treatment with chemotherapy and died at home.

There is evidence that chemotherapy for metastatic colorectal cancer can improve survival and should be considered in all patients not suitable for surgery[8,9].

MANAGEMENT OF COLORECTAL CANCER: FOCUS ON POPULATION SCREENING

Mohammed Nizamuddin and Marta Carpani de Kaski



Colorectal cancer screening

As many patients with CRC do not develop symptoms until the cancer is advanced, the detection of a greater proportion of cases at an earlier stage can only be achieved by screening asymptomatic people. Regular bowel cancer screening has been shown to reduce the risk of dying from bowel cancer by 16%[2].

The target groups and method of screening is still somehow controversial, but there is good evidence that screening for CRC in the general population over the age of 50 would be at least as cost-effective as mammography screening is for breast cancer.

Screening asymptomatic individuals at standard risk of CRC aims to detect premalignant lesions i.e. polyps, or cancer at a curable stage. The present options used for screening are faecal occult blood (FOB) or flexible sigmoidoscopy although, particularly in the USA, the use of colonoscopic screening in the general average risk population is advocated[3].

Decisions about whether and how to screen persons under the age of 50 years require consideration of many factors, such as life expectancy, cost, natural history of non-malignant advanced neoplasia and individual risk. A major study in the USA[4] on the prevalence of colorectal lesions in persons 40–49 years of age, suggested that the prevalence is low and results were compatible with the current strategy of starting to screen for colorectal cancer at the age of 50 among persons at average risk.

The NHS Bowel Cancer Screening Programme is now being rolled out nationally and will achieve nation wide coverage by 2009[5].

Programme hubs operate a national call and recall system to send out faecal occult blood (FOB) test kits, analyse samples and despatch results. Each hub is responsible for coordinating the programme in their area and works with up to 20 local screening centres.

The screening centres provide endoscopy services and specialist screening nurse clinics for people receiving an abnormal result. Screening centres are also responsible for referring those requiring treatment to their local hospital multidisciplinary team (MDT).

Faecal occult blood testing

Haemoccult screening is the only test shown in a randomised controlled trial to reduce mortality from CRC (annual frequency – 33% reduction; biennial frequency – 20% reduction) Unlike the alternative (flexible sigmoidoscopy screening) it has no effect on the incidence of CRC. Pilot projects have taken place in Australia[12] The problems are low take-up rate (54–75%), poor sensitivity (30–50% for cancers and <20% for adenomas), false positive results due to components of the diet.

The British Society of Gastroenterology has issued endoscopic screening guidelines (2002) for high risk individuals.(Figures 4 and 5) These include previous colorectal cancers, inherited polyposis syndromes, ulcerative colitis and Crohn's disease colitis and acromegaly(Table 2)[13].

	Screening procedure	Time of initial screening	Screening procedure and inter-val	Annual procedure 300,000 population
Colorectal cancer	Consul-tation, LFTs and colonoscopy	Colonoscopy within 6 months of resection only if colon evaluation pre-op incomplete	Liver scan within 2 years post-op Colonoscopy five yearly until 70 years	175

Figure 4: Guidelines for follow up after resection of colorectal cancer.

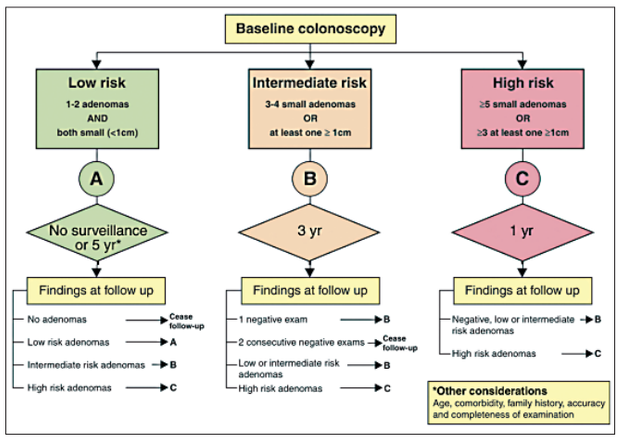


Figure 5: Surveillance strategy following adenoma removal.

MANAGEMENT OF COLORECTAL CANCER: FOCUS ON POPULATION SCREENING

Mohammed Nizamuddin and Marta Carpani de Kaski

	Screening procedure	Time of initial screen	Screening procedure and interval	Annual procedures 300,000 population
UC and Crohn's colitis	Colonoscopy & biopsies every 10cm	Pancolitis 8 years, left sided colitis 15 years from onset of symptoms	Colonoscopy 3 yearly in second decade, 2 yearly in third decade, subsequent-ly annually	46
IBD and primary sclerosing cholangitis +/-OLT	Colonoscopy	At diagnosis of PSC	Annual colonoscopy with biop-sies every 10cm	6

Table 2: Guidelines for screening and surveillance of asymptomatic colorectal cancer in patients with inflammatory bowel disease.

If dysplasia is detected, the biopsies should be reviewed by a second gastrointestinal pathologist. The appropriateness of surveillance should be discussed with patients who have ulcerative colitis or Crohn's colitis and a joint decision made on the balance of benefit to the individual[7].

The DOH has in 2004, through NICE, set guidelines for General Practitioners on the referral of patients with suspicion of colorectal cancer[14].

There is ongoing work for the production of the new NICE guidelines on *Diagnosis and management of colorectal cancer* due for completion in 2011.

MCQ Evaluation

1. Which of the following are NOT features commonly associated with colorectal cancer (CRC):
a. Family history of CRC.
b. Dietary history.
c. Tiredness.
d. History of inflammatory bowel disease.

Tiredness is a non-specific symptom present in many other conditions.

2. Which are the features characteristically associated with right-sided colorectal tumours:
a. Bright red bleeding per rectum.
b. Iron deficient anaemia.
c. Acute large bowel obstruction.
d. Seldom palpable abdominal mass.

3. Which of the following investigations do you think is the "gold standard" for detecting CRC?
a. Barium enema.
b. CT scan of chest, abdomen and pelvis.
c. Colonoscopy.
d. Faecal occult blood (FOB).

4. Which of the following statements regarding CRC is correct?
a. The majority of patients should have chemotherapy.
b. Surgery is the mainstay of treatment.
c. Radiotherapy is part of the radical management of colon cancer.
d. Most patients present with metastatic disease.

5. Regarding the case described in the present article, which of the following statements are correct?
a. Mrs CB should be offered adjuvant chemotherapy as she has node disease.
b. Mrs CB has Stage III disease.
c. Adenocarcinoma cell type disease is associated with poor prognosis.
d. Involvement of lymph nodes is associated with poor prognosis.

6. Indicate which of the following statements regarding surgery for liver secondary's in CRC are correct:
a. It can be associated with long-term survival.
b. The associated mortality is 15%.
c. Surgery should not be performed if more than one lesion is present.
d. Surgery is the only option for treatment of local metastases.

Up to 40% 5-year survival has been reported.

MANAGEMENT OF COLORECTAL CANCER: FOCUS ON POPULATION SCREENING

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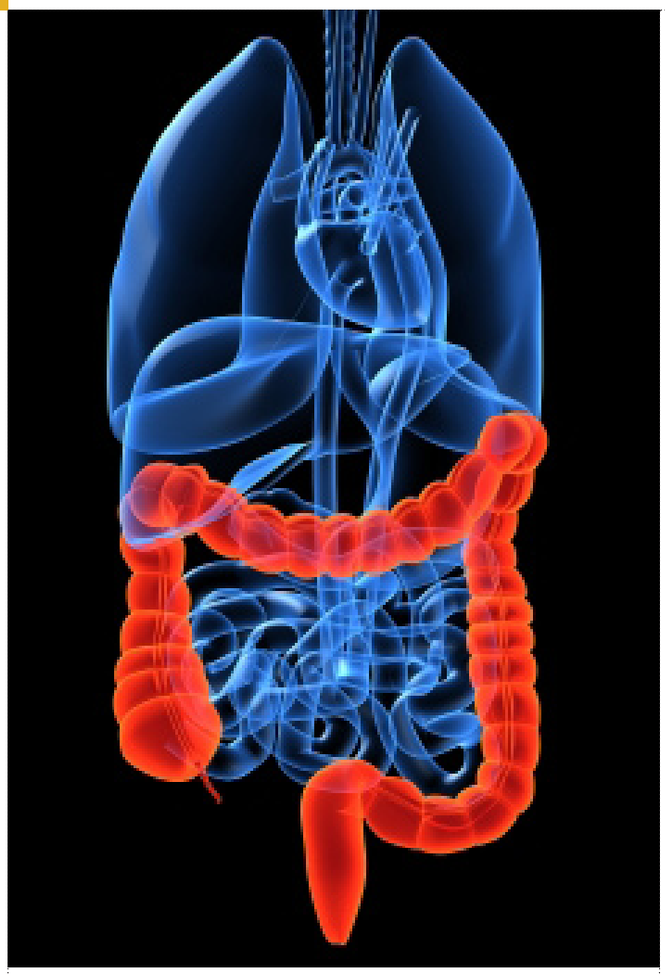

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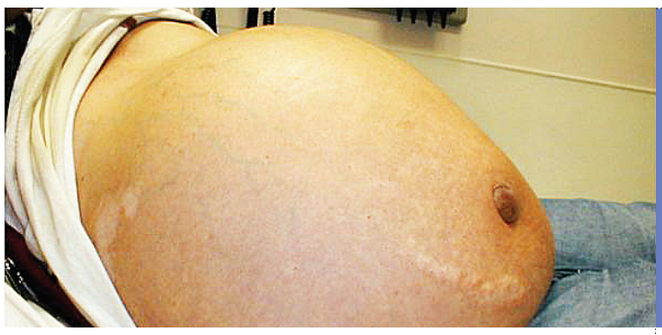
CASE-BASED PRACTICAL PROCEDURE: ABDOMINAL PARACENTESIS

Peter Malcomson and Abhishek Sharma



Abdominal paracentesis is a relatively safe and easy procedure used to drain ascitic fluid from the abdominal cavity. Practical Procedures.

History

A 56-year-old gentleman with a history of alcohol misuse and decompensated liver disease attends the gastroenterology outpatient clinic. He complains of increasing abdominal distension over the last 6 weeks. He has been drinking 15 units of alcohol per week and has not been compliant with his medications that include vitamin B tablets (2 tablets three times daily), thiamine (50mg four times daily) and spironolactone (200mg daily).

On examination he has evidence of tense ascites. A semi-urgent admission is planned within the next week for abdominal paracentesis.

Abdominal paracentesis in liver disease - the procedure

Abdominal paracentesis is a relatively safe and easy procedure used to drain ascitic fluid from the abdominal cavity.

Indications

In the setting of liver disease abdominal paracentesis is indicated for tense ascites or refractory ascites. The latter is ascites that cannot be treated adequately with diuretic therapy. Refractory ascites can be divided into "diuretic resistant ascites" which is unresponsive to maximal diuretic therapy and aggressive dietary sodium restriction, and "diuretic intractable ascites" where diuretic therapy is unsuccessful due to complications, such as renal failure, hypotension or encephalopathy[1].

Contraindications

Absolute contraindications include patient refusal and an acute abdomen requiring surgical intervention. Relative contraindications are uncorrected coagulopathy or thrombocytopenia, pregnancy, a distended urinary bladder, abdominal wall cellulitis, the presence of distended bowel loops and intra abdominal adhesions[2]. There is no evidence to support the administration of fresh frozen plasma (FFP) before the procedure in the presence of coagulopathy[1]; however it is common practice for FFP to be administered if the International Normalised Ration (INR) is greater than 1.6 – local policies may vary so this should be checked with senior staff. For thrombocytopenia, advice from haematologists may be needed regarding the cut-off level for platelet transfusion; local policies may differ.

Complications

Complications of ascitic paracentesis include bleeding, persistent leakage of ascitic fluid from the skin puncture site and wound infection. Abdominal wall haematomas occur in up to 1% of patients but are rarely serious or life threatening. The risk of haemoperitoneum or bowel perforation are less than 1/1000 procedures[1]. In the setting of liver disease if salt poor albumin is not administered then dilutional hyponatraemia, hypotension and hepatorenal syndrome can develop[3].

Preparation

It is essential that the need for paracentesis is documented by senior medical staff and informed consent, preferably written, is obtained from the patient before drain insertion. This should fully explain the indication for the procedure and possible complications that might arise. It is essential that salt poor human albumin solution (20 or 25%) has been ordered and prescribed on the fluid chart – for example, 1 unit of 20% human albumin after the first 5 litres has been drained and then for every 3L drained[1]. Finally, check that the patient has an intravenous cannula in situ prior to the procedure. Before inserting the drain it is necessary to have all the equipment set out on a trolley to take to the patient's bedside (Figure 1). It is very useful to have an assistant for the procedure.

Sterile pack
Sterile gloves
Chlorhexidine
Green needle x 2
Orange needle
10ml syringe x 2
1% lignocaine
Scalpel blade
Swabs x 2
Ascitic drain (Bonanno catheter pack)
50ml syringe x 1
Scissors and adhesive dressings
Specimen sample pots x 3
Blood culture bottles
Catheter bag and stand
Large sharps box

Figure 1: Equipment checklist.

CASE-BASED PRACTICAL PROCEDURE: ABDOMINAL PARACENTESIS

Peter Malcomson and Abhishek Sharma

Procedure

Positioning

Position the patient on their back in a slightly recumbent position toward the site of paracentesis with their arms behind their head. Make sure the bed is at a height that is comfortable to work at.

Identify the site

Identify the site by percussion techniques and mark a cross with a pen. The most common site for an ascitic drain is approximately 15cm lateral to the umbilicus, with care being taken to avoid an enlarged liver or spleen, and is usually done in the left or the right lower abdominal quadrant. The inferior and superior epigastric arteries run just lateral to the umbilicus towards the mid-inguinal point and should be avoided[1].

Prepare the site

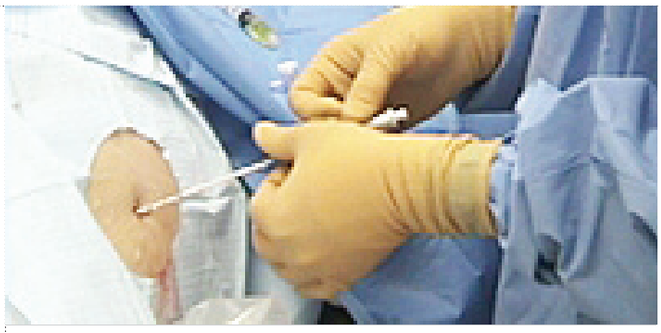
Using strict sterile conditions, a sterilised trolley surface and an aseptic non touch technique (ANTT), don sterile gloves and prepare the equipment at the patient's bedside. The assistant can help open the equipment so that sterility of the practitioner can be maintained. Prepare a sterile field by cleaning from the site of planned puncture outwards in a circular motion with chlorhexidine.

Local anaesthetic

Draw up 1% Lignocaine with the *green* needle into a 10ml syringe and infiltrate the site with an *orange* needle, aspirating before each infiltration of local anaesthetic. Infiltration of local anaesthetic with a *green* needle may be required. During anaesthetic infiltration ascitic fluid may be aspirated – this is usually straw coloured, but may be darker or blood tinged. Note the angle and depth of needle at which ascitic fluid has been obtained. Make a small superficial incision (~ 2–3mm) at the skin site with the scalpel. This facilitates insertion of the wider bore drain. Minor bleeding may occur but this can be stopped by pressure with a sterile swab.

Drain insertion

Prepare the bonanno catheter by straightening it and inserting the needle through it using the straightener provided in the pack. Once fully inserted the needle tip will be around half a centimetre in front of the plastic drain. Place a 10ml syringe on the back of the drainage catheter and insert it using a "Z" track technique as follows. The skin is punctured perpendicularly and the needle is advanced obliquely in the subcutaneous tissue while aspiration is maintained on the syringe. The needle direction is made perpendicular once more to enter the peritoneal cavity. This ensures that the needle track has puncture sites on the skin and peritoneum that do not overlie each other, reducing the likelihood of leakage from the skin[1]. Keeping the needle perpendicular to the abdominal wall the needle is advanced while aspirating until ascitic fluid appears in the syringe. The needle is then advanced around half a centimetre into the abdomen and the drain is advanced over it into the peritoneal cavity. Aspirate 50ml of ascitic fluid to place in 3 specimen sample pots and send to the lab for investigation.



After drain insertion

Secure the drain on the abdominal wall using adhesive dressings. A stitch is usually not necessary. Attach the drain to a catheter bag and leave on free drainage. Make sure the patient is comfortable and nursing staff are aware that fluid balance must be recorded accurately so that albumin can be replaced. Regular observations (pulse, blood pressure, temperature, respiratory rate and oxygen saturations) will also be needed during the paracentesis. The drain must be removed no later than 6–8 hours after insertion to minimise infection risks. Carefully dispose of all sharps in the sharps box.

The fluid should be drained to dryness as quickly as possible with albumin replacement as outlined earlier. This may require gentle mobilisation of the drain or moving the patient if necessary[1]. After the drain is removed a plaster can be placed at the insertion site and the patient should be encouraged to lie on their opposite side for 2 hours to minimise the risk of ascitic fluid leakage.

Investigations

Ascitic fluid should be sent off for a white cell count (>250cells/mm^3 suggests spontaneous bacterial peritonitis), culture and inoculated into blood culture bottles at the bedside as this has been shown to increase the chance of detecting organisms[4]. Fluid should also be sent for protein and albumin, glucose, cytology and amylase if pancreatitis suspected. Spontaneous bacterial peritonitis will require antibiotic therapy; discussion with microbiologists for local prescribing guidelines may be needed.

Patients with one episode of spontaneous bacterial peritonitis should be commenced on prophylaxis with continuous norfloxacin 400mg/day or ciprofloxacin 500mg once daily[1].

Documentation

Document in the notes that consent was obtained, time of procedure, physician who performed the procedure, their assistant, the method above in brief, any complications that occurred, the investigations requested and a time by which the drain is to be removed.

CASE-BASED PRACTICAL PROCEDURE: ABDOMINAL PARACENTESIS

Peter Malcomson and Abhishek Sharma

Questions

1. Which of the following is not a contraindication (absolute or relative) for abdominal paracentesis?

a. An acute (or "surgical") abdomen.
b. Coagulopathy.
c. Abdominal wall cellulitis.
d. Abdominal pain.
e. Distended bowel loops on abdominal X-ray.

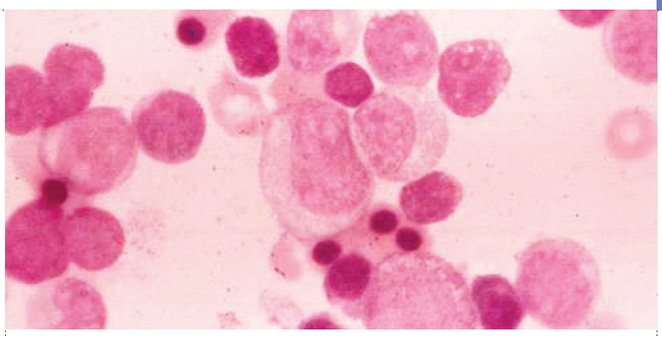
2. After paracentesis, which of the following is not a routinely recommended test for ascitic fluid analysis?

a. Amylase.
b. White cell count.
c. Gram stain.
d. Protein content.
e. Cytology.

Answers

1. Answer d.

a. An acute abdomen is an absolute contraindication for abdominal paracentesis – these patients require urgent surgical input and imaging to find the cause. Drain insertion may cause more damage in this case.
b. Coagulopathy is a relative contraindication for abdominal paracentesis. Most practitioners would advise that fresh frozen plasma (FFP) is given when the INR is above 1.6, however local policies may vary slightly so guidance should be sought prior to drain insertion.
c. Abdominal wall cellulitis is a relative contraindication to paracentesis as there is a risk of introducing infection into the peritoneal cavity. If at all possible then a site should be chosen where cellulitis is not present, e.g. on the other side of the abdomen provided that paracentesis would still be safe (check for organomegaly).
d. Abdominal pain is not a contraindication for paracentesis as long as an acute abdomen is not present. Tense ascites and spontaneous bacterial peritonitis may in fact cause pain.
e. Distended loops on abdominal X-ray are a relative contraindication to paracentesis. In this situation if paracentesis is still indicated then this should be undertaken under ultrasound guidance to minimise risks of perforation.



2. Answer c.

a. Ascitic fluid amylase should be sent as this will be elevated in cases of pancreatitis.
b. An ascitic fluid white cell count is mandatory to exclude spontaneous bacterial peritonitis. This is suggested by a cell count >250cells/mm3. Urgent antibiotic therapy should be initiated[1].
c. Gram stain of the fluid is not recommended as it is rarely helpful1. A white cell count and inoculation of blood culture bottles are recommended if bacterial peritonitis is suspected.
d. Ascitic fluid protein content, especially albumin, is useful as this may suggest the cause of the ascites[1].
e. Ascitic fluid cytology should be sent to rule out malignancy, however the yield is less than 10%. This can be maximised by sending off a large volume of fluid (a few hundred millilitres) and asking the pathology lab to spin the sample to look for malignant cells.

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THE INAPPROPRIATE REQUESTING OF THE LIVER FUNCTION TEST IN A TYPICAL DISTRICT GENERAL HOSPITAL - A SUCCESSFUL INTERVENTION AND IMPLICATION FOR FOUNDATION TRAINING

Vishal Luther, Michael Marks, Tony Everitt And Stuart Smellie

Abstract

Background

The use of hospital laboratory testing is increasing at considerable cost to the Health Service. A significant proportion of the tests ordered are inappropriate, a finding attributed to inadequacies in medical training. The Foundation Curriculum was set up to improve quality of practice, and highlighted the need for junior doctors to learn about appropriate testing.

Aim

Our aim was to explore, through audit, whether junior doctors on medical wards in a typical District General Hospital (DGH) were inappropriately requesting the Liver Function Test (LFT), and if so, to identify strategies to improve requesting behaviour.

Standard

There should be a minimum of retesting for LFTs of 2 days for inpatients.

Disregarding the exceptions, a new finding of abnormal LFT allows for daily repetition of the LFT for a further two days. The patient must have been admitted for more than five days and have not been labelled as Medically Fit for Discharge.

Method and Results

A computer based retrospective analysis of the LFT results of each patient were undertaken. One hundred and twenty-three patients met the criteria, and 906 LFT results were subsequently analysed. Two hundred and ninety-nine (33.00%) inappropriate LFT tests requests were identified.

Following an intervention involving compulsory consultant teaching and a ward poster, a reaudit 2 months post intervention which included 114 patients showed the overall number of LFT requests to have fallen to 514, and the total number of inappropriate tests to have fallen to 101 (19.65%) ($p<0.0001$).

Conclusion

Approximately one-third of all the requests for LFTs in this District General Hospital were inappropriate. Assuming the marginal cost of each test to be 60 pence, and approximately 70,000 inpatient LFT samples processed each year, this costs an additional marginal cost of £14,000 each year. Despite being defined as a competency within the Foundation Curriculum, Foundation doctors need education about appropriate testing. A simple strategy of education appears enough to dramatically correct this problem.

Background

Blood tests in many hospitals are requested either electronically or via a paper form containing a collection of tick boxes for each specific blood test component. As junior doctors are predominantly ward based, a significant proportion of the work done by junior doctors involves the requesting and subsequent interpretation of blood tests[1, 2].

The use of laboratory testing has increased inexorably at considerable cost to the Health Service[3]. Previous studies have suggested that this may be partly due to an increase in inappropriate testing[4], the junior doctors being a group incriminated[5]. Inappropriate testing causes unnecessary patient discomfort, generates false-positive results, increases the workload of the diagnostic services, and uses valuable health care resources.

The Foundation Programme was established in 2005 as a major reform of the existing postgraduate medical training, as it was felt there was a need for a new system that would provide better care for patients[6]. Section 4; 7.5 of the Foundation Curriculum states that "doctors must develop the ability to select appropriate investigations and interpret the results", and importantly, "learn to evaluate when investigations are not needed and are not cost-effective"[7].

The liver function test (LFT) is a test frequently requested by junior doctors. It consists of a panel of biochemical components that indicate structural or functional damage to the liver (see Table 1)[8].

Component	Half-life
Albumin	20 days
Alkaline phosphatase (ALP)	3 days
Alanine Aminotransferase (ALT)	2 days
Bilirubin	20 days

In 2002, a large pathology department located in Australia (Southern Cross) estimated that the inappropriate requesting of LFTs cost their health service approximately £14,000 each year. In light of the half-lives of the components of the LFT, Southern Cross implemented a minimum of retesting interval for LFTs of 2 days for inpatients[9].

Aim

The aim of this audit was to explore whether doctors on medical wards in a typical district general hospital were inappropriately requesting LFTs.

Methods

Sample Selection Criteria

1. Patients belonging to medical wards in Basildon Hospital during the month of December 2008.

2. Patients who have been an inpatient for five days or more (to ensure adequate time for assessment of inappropriate requesting and exclude tests performed in the acute admission period).

3. Patients who have not been labelled as Medically Fit for Discharge (as blood test requesting reduces in these cases).

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Standard

The Southern Cross Pathology Department guidance states that there should be a minimum retesting interval for LFTs of 2 days for inpatients.

An abnormal LFT is defined as having any component of the LFT outside its defined reference range as stated by our local Pathology Handbook.

In the event of a newly found abnormal LFT, agreed good practice in this hospital produced locally by two consultants (gastroenerologist and clinical biochemist) allows for daily repetition of the LFTs for a further 2 days, after which retesting should return to a maximum of alternate days. This is to allow the doctor to explore any trends, changes or differentials.

The exceptions include patients with diagnosed acute hepatitis, acute pancreatitis, acute cholecystitis and acute cholangitis.

Analysis

A computer based retrospective analysis of the LFT results of each patient who met the assessment criteria from all the medical wards was undertaken. The date of admission and the associated LFT result was recorded as normal or abnormal, and the date and result of each subsequent result was tabulated. The marginal cost of an LFT was set at 60 pence from information provided by the biochemistry laboratory.

Following the completion of this audit, a compulsory lecture was delivered to the Foundation doctors by a Consultant biochemist regarding both the findings of this audit and guidance as to the appropriate requesting of laboratory investigations in hospital. In addition, a poster illustrating the results of this audit and the guidelines for LFT requesting that should be implemented by the junior doctors was delivered to all medical wards and posted on the trust's Intranet.

Results

Over the study period, a total of 123 patients had a total of 906 LFTs requested. Two hundred and ninety-nine of the requests (33%) did not meet the criteria for an appropriate test. The Total number of patient days assessed was 2,566 days, thus the average number of days per LFT test per patient was 2.83 days.

We reaudited the requesting behaviour of LFTs 2 months post intervention. Over the post-intervention period, a total of 114 patients had a total of only 514 LFTs requested. Thus the average number of days per LFT request per patient rose significantly to 5.19 days ($p<0.0001$, chi squared test). Equally, only 101 of the requests (19.65%) failed to meet the criteria for an appropriate test, demonstrating a significant fall ($p< 0.0001$, chi squared test) in the inappropriate requesting behaviour of the LFT.

Conclusion

Approximately one-third of all the requests for LFTs from the medical wards were inappropriate. As there are approximately 70,000 inpatient LFT requests each year in this hospital, assuming 1/3 to be inappropriate, the avoidable marginal cost of these tests is £14,000 each year. This is a minimalist estimate as it fails to consider the potential cost of collecting, processing and analysing the test.

In addition, the guidance allowing LFT daily testing of patients with at least one 'abnormal' result outside of the context of clinical hepatitis is very permissive as repeats of minor LFT 'abnormal' values would often be deemed clinically unnecessary unless related to the patient's underlying medical condition. Many incidental 'minor' anomalies are probably also being monitored.

Discussion

Increasing the number of tests requested by a doctor does not improve the quality of care provided to a patient[10, 11]. The use of laboratory testing, however, is increasing, with the cost to the health services. Many of the tests that are being ordered are inappropriate, a finding which may be attributable to failings in medical education.

The Foundation Curriculum was set up in 2005 to improve quality of practice. It stresses that doctors must learn to select appropriate investigations, and evaluate when investigations are not needed. However, this audit, undertaken in a typical district general hospital has revealed that approximately one-third of all the requests for the liver function test made within the hospital are inappropriate, wasting about £14,000 each year. Such wastage is also prevalent in other hospitals[5]. This figure is also a minimalist estimate as it does not include any of the potential labour and materials savings which could be achieved if a more appropriate testing strategy avoided the sample actually being taken, with the resultant savings in phlebotomy, laboratory sample processing and other marginal costs.

Previous studies have suggested this apparent increase in inappropriate requesting arises from several factors: "routine" diagnostic testing, where the same set of blood tests are requested for all patients independent of clinical indication; defensive behaviour, difficulty in remembering when the last test was done, ignorance of the cost of tests and their recommended minimum retesting intervals[5,12]. This audit suggests that a "box-ticking" culture on the blood test request form is being employed.

Though the Foundation Curriculum has stated that doctors must learn to select appropriate investigations, this audit suggests the current Foundation Training Programme has yet to allow this competency to develop. There is, thus, an urgent need for this to be prioritised within the Foundation programme or the health service will continue to experience considerable unnecessary laboratory expenditure.

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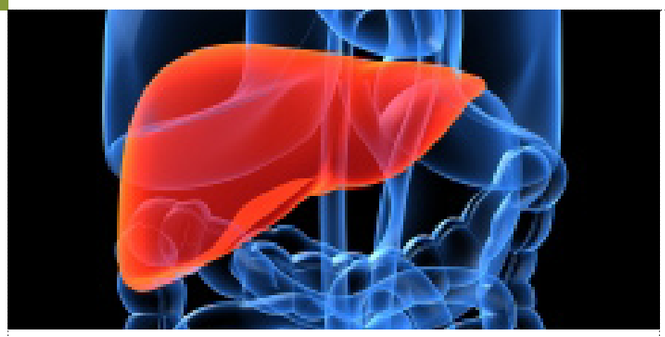

Successful strategies to control inappropriate requesting have included educating doctors about test indications and costs, developing guidelines and audit[12, 13].

The reaudit confirms the value of education in minimising the levels of inappropriate requesting. The concern is whether this effect is temporary. Effective approaches at reducing inappropriate test requesting behaviour by juniors long-term have involved programmes of regular senior education and supervision[14,15]. In particular, direct feedback to the junior about their practice has been very successful[16]. While setting aside additional time for such continuous teaching would be demanding in terms of consultants' workload, this teaching could be easily delivered during the ward round itself, where a proactive approach can identify inappropriate testing made by their juniors, and offer an opportunity for direct feedback to learn from. Greater opportunity for regular involvement from laboratories in the teaching process should also be considered in view of the large part played by diagnostics in Foundation doctors' workload. This would help to ensure junior staff achieve Foundation Curriculum competency, and the health service could save precious resources[1].

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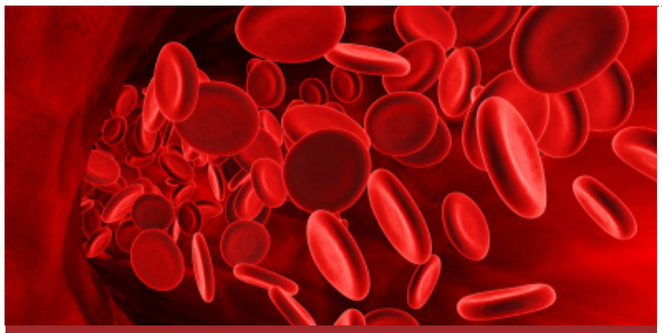
MANAGEMENT OF ACUTE (NON-VARICEAL) UPPER GASTROINTESTINAL BLEEDING: A REVIEW OF GUIDELINES

Dr A Watret



On admission the patient should be stratified according to risk and transferred to an environment in which optimisation of care can be confidently given. Patient Management.

Risk Stratification

On admission the patient should be stratified according to risk and transferred to an environment in which optimisation of care can be confidently given. This is usually an acute medical ward or, if severe, then ITU/HDU admission may be warranted.

Management depends on:

- Age (mortality <0.1% if <60 years old and 20% if >80 years[1])
- Severity and cause of the bleeding (most recurrent haemorrhages occur within 24 hours and pose a higher risk (20% of all cases)[1]
- Compounding co-morbidities
- Presence or absence of shock at any time throughout treatment (increases mortality) and endoscopic findings
- Melaena is usually less hazardous than haematemesis.

Although the British Society of Gastroenterology Endoscopy Committee guidelines 2002 are available, the group states that the situation should be guided by clinical judgement and therefore deviation away from the guidelines is at the clinician's discretion.

Rockall et al.[2-3] have constructed a scoring system for risk of rebleeding and death after admission to hospital for acute gastrointestinal bleeding. It incorporates independent risk factors enabling an accurate prediction of death.

	Rockall Score			
VARIABLE	**0**	**1**	**2**	**3**
AGE (Y)	**<60**	**60–79**	**≥80**	
Shock	No shock (systolic BP >100, pulse <100)	Tachycardia (systolic BP >100, pulse >100)	Hypo-tension (systolic BP <100, pulse >100)	
Co-morbidity	Nil Major		Cardiac failure, ischaemic heart disease, any major co-morbidity	Renal failure, disse-minated malignancy
Diagnosis	Mallory-Weiss tear, no lesion and no SRH	All other diagnoses	Malignancy of upper GI tract	
Major SRH (on endos-copy)	None or dark spot		Blood in upper GI tract, adherent clot, visible or spurting vessel	
Each variable is scored and the total score calculated by simple addition SRH, stigmata of recent haemorrhage				

Table 1: Rockall scoring system for rebleeding and risk of death.

A score of <3 has an excellent prognosis

A score of >8 is associated with a high risk of death

Unfortunately the Rockall score is retrospective and can only be calculated post-endoscopy. It predicts those at risk of death, not those who require intervention to control haemorrhage. Therefore, prospective risk scoring systems have been proposed for junior staff to follow in an emergency.

Blatchford et al.[4] have identified key and readily available clinical and laboratory variables (excluding endoscopic findings) to determine appropriate care and potential need for active treatment. In contrast to The Rockall score, it stratifies the need for clinical intervention including blood transfusions and surgical or endoscopic procedures. The score was devised from a prospective audit of 1,748 admissions for upper gastrointestinal haemorrhage in West Scotland. A subsequent simplified 'fast track' score was constructed and identified 99% of patients who required intervention and 32% with minor bleeds who did not[5].

MANAGEMENT OF ACUTE (NON-VARICEAL) UPPER GASTROINTESTINAL BLEEDING: A REVIEW OF GUIDELINES

Dr A Watret

This screening tool, therefore, has a sensitivity adequate to provide a good degree of clinical safety. This method highlights patients who are at low risk of needing acute inpatient care and therefore facilitates outpatient assessment and management. Thus it becomes a suitable tool for patient triage and allows division of patients into three groups: early discharge, admission to HDU or urgent endoscopy.

Prospective assessment is the focus of a recent publication in *The Lancet*[6]. The article quotes the first prospective analysis of The Glasgow-Blatchford Score (GBS) by Stanley and colleagues. It analyses the feasibility of adopting it in emergency departments. Patients need to score 0 to be managed as an outpatient. (Heart rate <100 beats per minute, systolic blood pressure ≥ 110 mmHg, absence of melaena, syncope, cardiac failure, or liver disease, haemoglobin of ≥130g/L for men or ≥120g/L for women and urea <6.5 mmol/L). Their findings suggest that utilisation of this score would produce a mean reduction of 1.2 days (in bed days) per patient presenting. This represents a saving of £13.6 million in a population of 60 million (assuming a daily hospital cost of £227 and an annual upper GI haemorrhage rate of 100 per 100,000 people). Furthermore this will reduce patient exposure to unnecessary hospital acquired risks. However further studies are required before establishment of GBS.

Table 2 outlines the admission risk markers and associated score component values of the GBS[4].

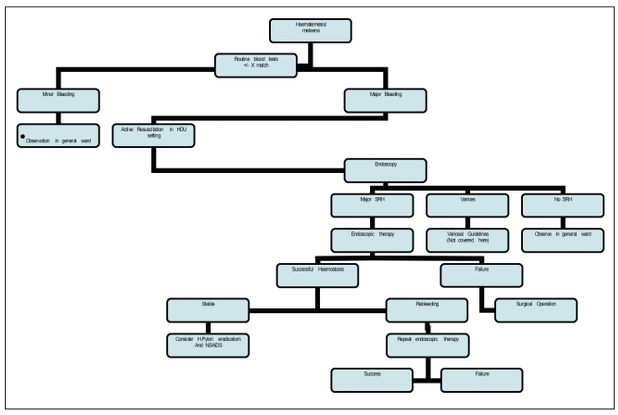
Admission Risk marker	Score component value
BLOOD UREA (mmol/L)	
<6.5	0
≥ 6.5<8.0	2
≥8.0<10.0	3
≥10.0<25.0	4
≥25	6
HAEMOGLOBIN (g/L) FOR MEN	
≥130	0
≥120<130	1
≥100<120	3
<100	6
HAEMOGLOBIN (g/L) FOR WOMEN	
≥120	0
≥100<120	1
<100	6
SYSTOLIC BLOOD PRESSURE (mmHG)	
≥110	0
100–109	1
90–99	2
<90	3
OTHER MARKERS	
Pulse ≥100 (per min)	1
Presentation with melaena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac Failure	2

Table 2: Risk Stratification Algorithm to aid management of acute gastrointestinal haemorrhage.

MANAGEMENT OF ACUTE (NON-VARICEAL) UPPER GASTROINTESTINAL BLEEDING: A REVIEW OF GUIDELINES

Dr A Watret

HDU, High Dependency Unit
NSAIDS, non-steroidal anti-inflammatory drugs
SRH, stigmata of recent haemorrhage[2]



Further Management[2]

Endoscopy is diagnostic, prognostic and therapeutic. It is usually performed as a semi-elective procedure within 24 hours of admission. Resuscitation takes priority over and above endoscopy and the patient should be stabilised before the procedure.

If performed as an urgent 'out of hours' procedure facilities should be available for all eventualities including:

- Fully equipped endoscopy unit
- Experienced endoscopists competent in achieving haemostasis
- Personnel experienced in endotracheal intubation and airway management
- Assistants with adequate training.

Endoscopic treatments include:

- Injection of 1:10,000 adrenaline in normal saline achieving primary haemostasis in up to 95% of patients. However bleeding will recur in 15–20%[2]. Other agents, such as sclerosants (polidoconal and ethanolamine), are less convincing and may cause life threatening necrosis at injection sites[2]. Injection of absolute alcohol shows no advantage over adrenaline and risks perforation. Injections of clot stimulators, such as fibrin glue or thrombins, have shown effectiveness but are not readily available
- Thermal haemostasis using a heater probe or multipolar coagulation have been shown to have equal efficacy[2]. Laser therapy is now redundant. The heater probe is applied repetitively until haemostasis is achieved and a blackened area is formed. Combined pressure tamponade and heat application is as effective as adrenaline injection[2]. The heater probe has a powerful water jet which removes the overlying clot and therefore aids therapeutic intervention. The BSG Endoscopy Committee guidelines quote trials comparing the outcome of combination adrenaline with heater probe therapy versus adrenaline alone and as yet a consensus has not been reached. However, in patients with active arterial bleeding, combination therapy appears to confer a more favourable outcome.
- Mechanical clips can be applied directly to bleeding areas and are useful for large actively bleeding vessels. Application is difficult in awkwardly placed ulcers.

Drug therapy

- Acid suppressing drugs increase stability of a clot. A pH of >6 is required for platelet aggregation and conversely clot lysis occurs below this threshold. H2 receptor antagonists do not reliably reduce stomach acidity. In general, the use of omeprazole (proton pump inhibitor) in upper GI bleeding has shown benefit.

Daneshmend et al.[7] conclude from their large two centre RCDBT (in which patients received intravenous boluses of omeprazole or placebo) that omeprazole lowers endoscopy evidence of persistent bleeding but other endpoints including mortality, rebleeding and transfusion requirement were similar in each group. Their results suggest that acid inhibition is capable of influencing intragastric bleeding but conclude that their data does not justify the routine use of these medications in haematemesis and melaena.

Lau et al.[8] undertook a large RCDBT and conclude that after endoscopic treatment of bleeding peptic ulcers, a high dose infusion of omeprazole substantially reduces the risk of recurrent bleeding (6.7% versus 22.5% rebleeding within 30 days in the omeprazole versus placebo group), blood transfusion requirement and duration of hospital stay. Mortality tended to be less but this did not achieve clinical significance.

The committee therefore recommend high dose omeprazole therapy in patients presenting with major ulcer bleeding at a dose of 80mg stat followed by an infusion of 8mg hourly for 72 hours[2].

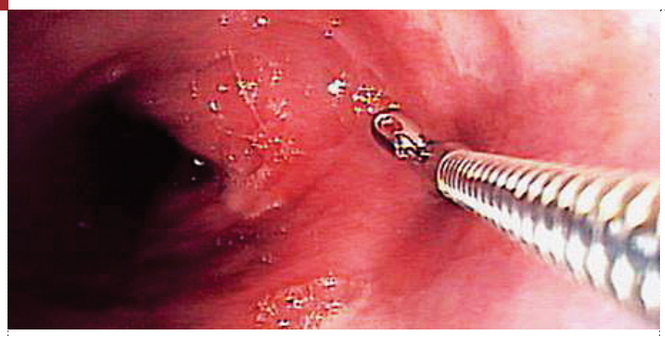
- Somatostatin at a high intravenous dose suppresses secretion of acid and reduces splanchnic blood flow. It is therefore a potential haemostatic agent, although limited evidence exists to support its use.

- Antifibrinolytic drugs, such as tranexamic acid, have been shown to reduce mortality and the need for surgical intervention in ulcer bleeding patients. These agents do not, however, appear to reduce ulcer rebleeding rates and are currently not recommended for use.

- Sucralfate may act by protecting the mucosa from acid-pepsin attack in gastric and duodenal ulcers. It is a complex of aluminium hydroxide and sulphated sucrose but has minimal antacid properties. It should be used in caution in patients under intensive care, especially those receiving concomitant enteral feeds or those with predisposing conditions, such as delayed gastric emptying, following reports of bezoars formation (BNF, September 2007, *BMJ Publishing Group Ltd.* www.bnf.org).

MANAGEMENT OF ACUTE (NON-VARICEAL) UPPER GASTROINTESTINAL BLEEDING: A REVIEW OF GUIDELINES

Dr A Watret



Post endoscopy management[2]?

Close monitoring following endoscopy is required (notably, pulse, blood pressure and urine output). If the patient is haemodynamically stable 4–6 hours after endoscopy (with or without endoscopic therapeutic intervention) they should be allowed to drink and start a light diet as there is no evidence to suggest that prolonged fasting confers any advantage. If there is evidence of further rebleeding (melaena, haematemesis and deterioration of vital signs) then confirmatory repeat endoscopy is recommended. If bleeding is severe then surgical intervention without repeat endoscopy may be warranted. This is based on clinical judgement and Rockall score of risk stratification. For the majority of patients a watch and wait policy is adopted and surgery is considered after rebleeding for a second time[2].

Explanation/advice for the patient

The following points should be highlighted:

- what has happened (especially if the patient is elderly and anxious)
- the need to closely monitor in the acute environment
- the results of the endoscopy
- subsequent management including a repeat endoscopy in 6 weeks +/- biopsies to monitor the ulcer/exclude malignancy and PPI cover until then
- potential for surgery if an ulcer re-bleeds or perforates
- *helicobacter pylori* eradiction
- cessation of causative factors, for example, NSAIDs and aspirin. If absolutely necessary then damage control should be advocated by using the least harmful options (Ibuprofen/COX-2 specific anti-inflammatory[1]).

Summary

Upper gastrointestinal bleeding is a common presentation to A&E.

It requires prompt medical attention and stratification according to risk factors.

Fluid resuscitation (and blood) is imperative if clinical need dictates.

Endoscopy is diagnostic, prognostic and therapeutic.

Drug therapy has a role to play: high dose PPIs are the drugs of choice.

Re-endoscopy +/- surgery is required if recurrent bleeding occurs.

It is mandatory to follow-up patients after an upper gastrointestinal bleed.

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DYSPEPSIA IN A PRIMARY CARE SETTING - ARE WE MEETING THE GUIDELINES?

Dr Neel Sharma and Dr Janet Kirton

Introduction

Approximately 2–5% of the population consult their general practitioner each year with symptoms suggestive of dyspepsia[1]. And it is estimated that dyspepsia costs the NHS over £500 million per year[2]. The four major causes of dyspepsia include peptic ulcer disease, gastro oesophageal reflux disease (GORD), malignancy and non-ulcer or functional dyspepsia.

Functional dyspepsia was originally defined as pain or discomfort centred in the upper abdomen[3]. However the major flaw with such a definition is that dyspepsia is essentially polysymptomatic. Approximately 99% of patients report more than two symptoms, over 80% report more than five symptoms and less than 0.1% report one symptom[4].

The main classical symptoms of dyspepsia include abdominal pain, heartburn, acid reflux, nausea and flatulence. In light of this the definition of functional dyspepsia was modified and this led to the development of the Rome III criteria[5].

The Rome III Criteria includes for at least 3 months, with onset at least 6 months previously, one or more of the following:

- *Bothersome postprandial fullness*
- *Early satiation*
- *Epigastric pain*
- *Epigastric burning*

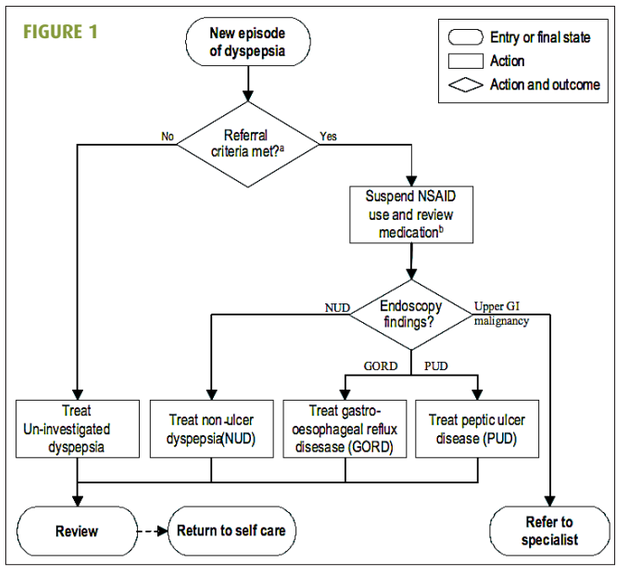
And

No evidence of structural disease (including at upper endoscopy) that is likely to explain the symptoms5.

According to the American Gastroenterological Association the general consensus is that patients who complain predominantly of heartburn or acid regurgitation which occurs typically more than once a week are classified as having gastro-oesophageal reflux disease and are not part of the definition of dyspepsia[1]. However, they recognise that there is considerable symptom overlap between dyspepsia and GORD in the un-investigated patient in the primary care setting.

NICE have developed guidelines for dyspepsia management in primary care which depend essentially on whether patients meet the referral criteria for an endoscopy. Referral criteria for such an investigation include chronic gastrointestinal bleeding, weight loss, dysphagia, vomiting, iron deficiency anaemia, evidence of an epigastric mass on clinical examination and patients aged 55 or over with recent onset dyspepsia[6].

FIGURE 1



a) Immediate referral is indicated for significant acute gastrointestinal bleeding.

Consider the possibility of cardiac or biliary disease as part of the differential diagnosis

Urgent specialist referral* for endoscopic investigation is indicated for patients of any age with dyspepsia when presenting with any of the following: chronic gastrointestinal bleeding, progressive unintentional weight loss, progressive difficulty swallowing, persistent vomiting, iron deficiency anaemia, epigastric mass or suspicious barium meal.

Routine endoscopic investigation of patients of any age, presenting with dyspepsia and without alarm signs, is not necessary. However, in patients aged 55 years and older with unexplained** and persistent** recent-onset dyspepsia alone, an urgent referral for endoscopy should be made.

Consider managing previously investigated patients without new alarm signs according to previous endoscopic findings.

DYSPEPSIA IN A PRIMARY CARE SETTING - ARE WE MEETING THE GUIDELINES?

Dr Neel Sharma and Dr Janet Kirton

Offer lifestyle advice, including advice on healthy eating, weight reduction and smoking cessation, promoting continued use of antacid/alginates.
Good Medical Practice.

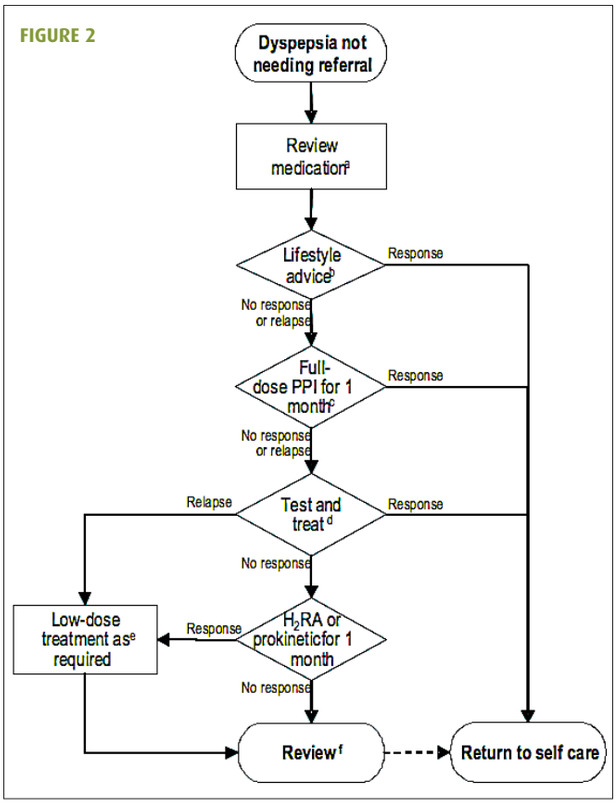
b) Review medications for possible causes of dyspepsia, for example, calcium antagonists, nitrates, theophyllines, bisphosphonates, steroids and NSAIDs. Patients undergoing endoscopy should be free from medication with either a proton pump inhibitor (PPI) or an H2 receptor (H^2RA) for a minimum of 2 weeks.

* The Guideline Development Group considered that 'urgent' meant being seen within 2 weeks.

** In the referral guidelines for suspected cancer (NICE Clinical Guideline no. 27), 'unexplained' is defined as 'a symptom(s) and/or sign(s) that has not led to a diagnosis being made by the primary care professional after initial assessment of the history, examination and primary care investigations (if any)'. In the context of this recommendation, the primary care professional should confirm that the dyspepsia is new rather than a recurrent episode and exclude common precipitants of dyspepsia such as ingestion of NSAIDs. 'Persistent' as used in the recommendations in the referral guidelines refers to the continuation of specified symptoms and/or signs beyond a period that would normally be associated with self-limiting problems. The precise period will vary depending on the severity of symptoms and associated features, as assessed by the health care professional. In many cases, the upper limit the professional will permit symptoms and/or signs to persist before initiating referral will be 4–6 weeks.

Figure 1: NICE guidelines on the management of patients who present with dyspepsia as a first episode[6].

FIGURE 2



a) Review medications for possible causes of dyspepsia, for example, calcium antagonists, nitrates, theophyllines, bisphosphonates, steroids and NSAIDs.
b) Offer lifestyle advice, including advice on healthy eating, weight reduction and smoking cessation, promoting continued use of antacid/alginates.
c) There is currently inadequate evidence to guide whether full-dose PPI for 1 month or *H. pylori* test and treat should be offered first. Either treatment may be tried first with the other being offered if symptoms persist or return.
d) Detection: use carbon-13 urea breath test, stool antigen test or, when performance has been validated, laboratory-based serology. Eradication: use a PPI, amoxicillin, clarithromycin 500 mg (PAC_{500}) regimen or a PPI, metronidazole, clarithromycin 250mg (PMC_{250}) regimen. Do not retest even if dyspepsia remains unless there is a strong clinical need.
e) Offer low-dose treatment with a limited number of repeat prescriptions. Discuss the use of treatment on an as-required basis to help patients manage their own symptoms.
f) In some patients with an inadequate response to therapy it may become appropriate to refer to a specialist for a second opinion. Emphasise the benign nature of dyspepsia. Review long-term patient care at least annually to discuss medication and symptoms.

Figure 2: NICE guidelines on the management of patients who present with dyspepsia as a first episode and who do not meet the referral criteria[6].

DYSPEPSIA IN A PRIMARY CARE SETTING - ARE WE MEETING THE GUIDELINES?

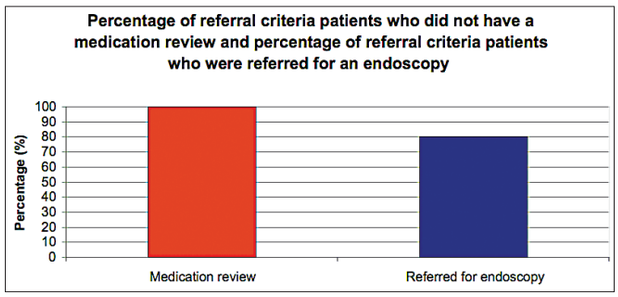
Dr Neel Sharma and Dr Janet Kirton

Audit overview

An audit was performed centred on all patients who presented to an inner city East London practice in a one year period with symptoms suggestive of dyspepsia. The objective being whether those patients met the referral criteria as laid out by NICE and if so were they managed accordingly. And if they did not meet such criteria were patients investigated and treated appropriately.

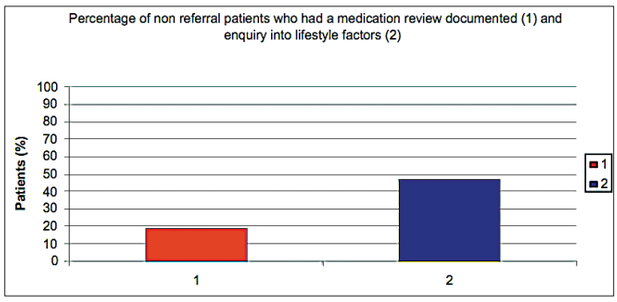
Results

Referral

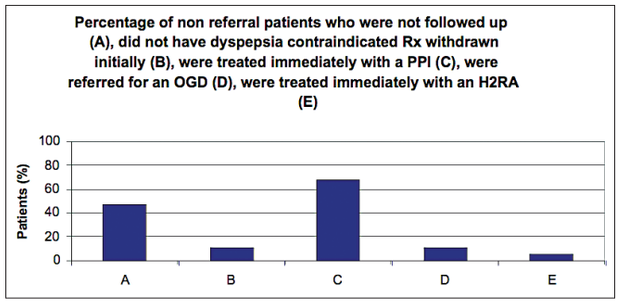


Of those who met the referral criteria no single patient had a medication review documented. And 80% of patients who met the referral criteria were referred for endoscopy.

Non-referral



Of those patients who did not meet the referral criteria approximately 18% had a medication review documented. And 46% of patients were asked about lifestyle factors.



Rx – Medication
PPI – proton pump inhibitor
OGD – oesophagogastroduodenoscopy
H2RA – H2 Receptor Antagonist

Of those patients who did not meet the referral criteria, 47% were not followed-up (A), 12% did not have drugs contraindicated in dyspepsia withdrawn until later reviews (B), 68% were treated with a PPI on the first presentation (C), 12% were inappropriately referred for endoscopy (D) and 6% were treated with an H^2RA on the first presentation (E).

Discussion

Analysing the patients who met the referral criteria confirms that 80% of patients were referred for endoscopy. However all patients did not have a review of their medication documented. NICE recommends this is an initial key step prior to referral for endoscopy.

Of those who fell under the non-referral umbrella, only 18% had a medication review documented and 46% of patients were asked about lifestyle factors. According to NICE it is essential to undertake these steps as an initial assessment of dyspepsia as evidence suggests that such simple measures are sufficient in reducing symptomatology in a large proportion of individuals. Further analysis showed that 12% of the practice population were in fact on medication contraindicated in dyspepsia and that this was only noted and subsequently withdrawn on follow-up reviews. This therefore further emphasises the importance of a medication review at first presentation.

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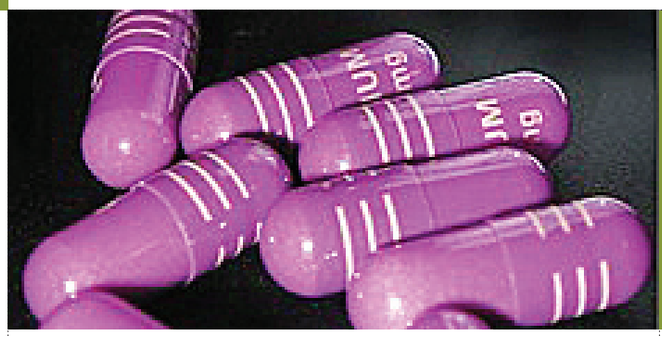

NICE clearly defines that patients should not be immediately treated with a proton pump inhibitor (PPI). Good Medical Practice.

A large proportion of non-referral patients (68%) were treated with a PPI at the first presentation. NICE clearly defines that patients should not be immediately treated with a proton pump inhibitor (PPI) and if so only if initial steps such as a medication review, lifestyle advice in addition to alginate/antacid use has been attempted. In 2006, approximately £425 million was spent in England alone on PPI use[7]. Globally this equated to £7 billion[8]. And further research has shown that 25–70% of patients who take PPIs have no real indication for doing so[9]. With regard to medication, 6% of non-referral patients were treated initially with a H2RA which should only be trialled if simple measures such as alginates/antacids and PPIs have been unsuccessful.

Twelve per cent of non-referral patients were referred inappropriately for endoscopy. Approximately 450,000 endoscopies are performed at a cost of £90 million each year[2]. And the vast majority of dyspepsia is functional. Of those who undergo endoscopy only 10% have evidence of peptic ulcer disease2. Therefore only if patients meet the referral criteria should they be referred as a matter of urgency for endoscopic investigations. A final learning point from the study is that it is important to follow-up all patients at whatever stage of treatment they are on to ensure their symptoms have resolved. The study highlights that 47% of the practice population of non-referral patients were not followed-up.

Key Learning Points

Referral

- Document medication review. Refer if criteria met.

Non-referral

- Enquire and document medication review and lifestyle factors first. Review after these steps have been made
- Avoid immediate treatment with PPI.
- Follow-up patients in all cases
- Refer appropriately.

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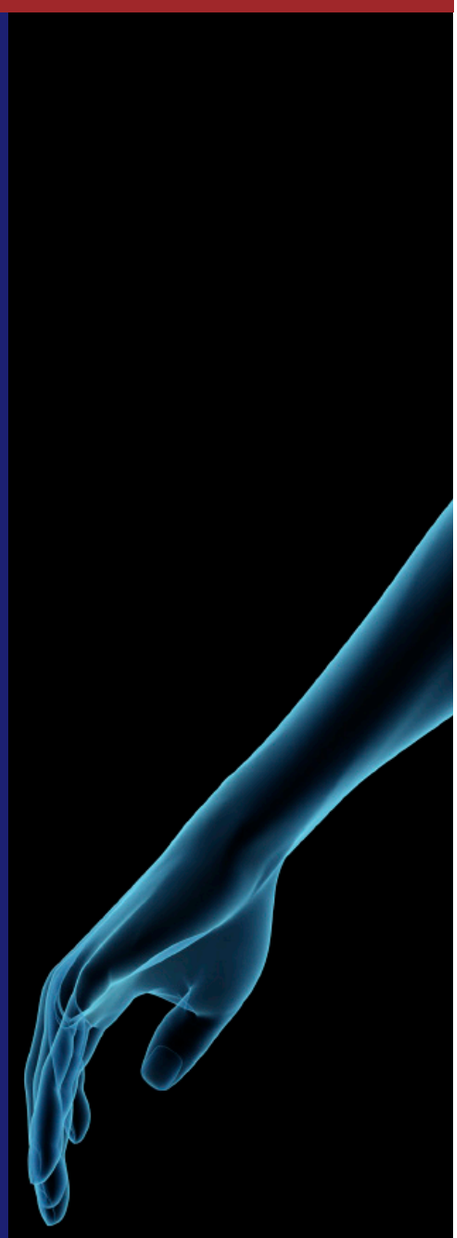

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