123 Doc EDUCATION

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FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 10

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.

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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:

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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.

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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.

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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.

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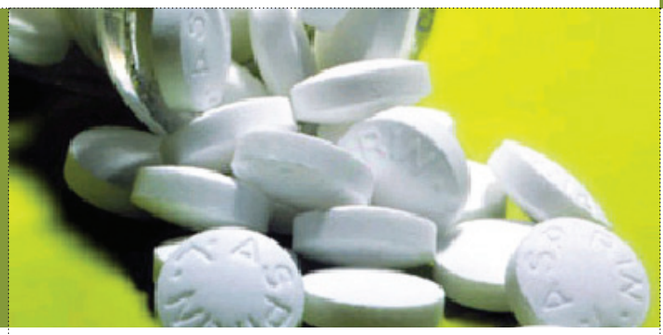
CLINICAL AUDIT: THE USE OF GASTRO PROTECTION IN TRAUMA PATIENTS RECEIVING NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

Sunil Sharma, Gokulan Phoenix, James Chan, Graham Sleat and Alastair Graham

A clinical audit of trauma patients in a district general hospital was performed to assess and improve the adherence to evidence-based guidelines for gastro protection when prescribing non-steroidal anti-inflammatory drugs (NSAID). Good Medical Practice.



Abstract

Background

A clinical audit of trauma patients in a district general hospital was performed to assess and improve the adherence to evidence-based guidelines for gastro protection when prescribing non-steroidal anti-inflammatory drugs (NSAID).

Methods

A clinical audit was conducted over an 18-week period to assess pre-intervention practice. Subsequently, changes including prescriber and pharmacist education were implemented and following this, data was collected over a 9-week period to assess any change in performance. Case notes and prescription charts of all adults (aged 18 and over) commenced on regular NSAID on admission to the trauma unit were reviewed.

Patients were risk stratified according to the number of risk factors, which included age over 65 years, major co-morbidity, oral steroids, anticoagulation, history of upper gastrointestinal ulceration or bleeding and prescription above the generally recommended dose of NSAID. The use of gastro protective drugs was recorded to measure adherence to evidence-based guidelines.

Results

A total of 644 patients were reviewed over the study period, 451 pre-intervention and 193 post-intervention. 100 patients fulfilled the inclusion criteria pre-intervention and 49 post-intervention. The proportion of high-risk patients co-prescribed gastro protection was 25.3% pre-intervention and 73.1% post-intervention. The likelihood of a patient receiving gastro protection increased significantly with the presence of high risk as compared to background risk both pre- ($p=0.002$) and post-intervention ($p<0.001$).

Conclusions

- The majority of trauma admissions are at high risk for developing gastrointestinal haemorrhage.
- Initial adherence to national guidelines for safe prescription of NSAID in our trauma unit was low but improved dramatically with intervention, including education of prescribers and pharmacists.
- Awareness of gastro protection guidelines must be raised in trauma units to minimise the risk of GI complications.

Introduction

Non-steroidal anti-inflammatory drugs (NSAID) are effective analgesic and anti-inflammatory agents that have been recommended by the World Health Organisation for the first line management of pain. However, their side effect profile includes adverse gastrointestinal events[1-4].

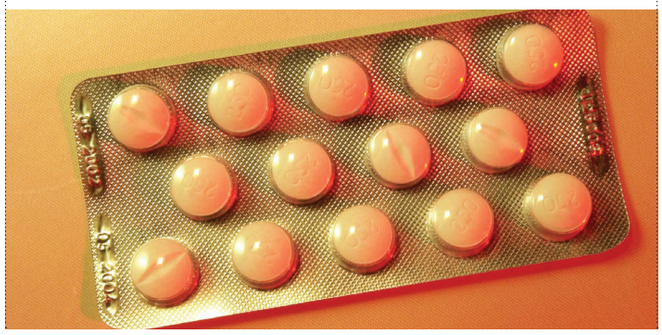
The widespread use of NSAID, together with the high turnover of orthopaedic trauma patients means that the safe prescription of NSAID represents an important clinical and epidemiological issue. There is much evidence to show that gastrointestinal toxicity is associated with chronic NSAID use, especially in the context of rheumatoid and osteoarthritis[5]. However, it has also been well documented that acute GI haemorrhage can occur as a result of short-term NSAID administration[6].

Guidelines for the safe prescription of NSAID have been developed by the American Rheumatological Society for pain control in patients who undergo elective orthopaedic surgery[7]. These recommend the use of gastro protection for patients on NSAID who have one or more risk factors.

The purpose of this audit was to assess adherence to evidence-based national guidelines on gastro protective strategies in new NSAID users in the orthopaedic trauma setting in a District General Hospital (Stoke Mandeville Hospital, Aylesbury) and to assess the association between risk factors and prescription of a gastro protective agent.

STANDARD

American Rheumatological Society guidelines recommend the use of gastro protection for patients on NSAID who have one or more risk factors[7].



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Method

The audit population, obtained from the local trauma and orthopaedics admissions access database (Microsoft Corporation, Seattle), consisted of emergency admissions under trauma and orthopaedics.

Case note and prescription chart review was carried out for all patients and inclusion criteria included: 1) minimum age of 18 years; and 2) commencement of NSAID at or during admission. Subjects on long-term NSAID and those who were commenced on NSAID on an irregular, as required, basis were excluded. Demographic, diagnostic, management information, type of NSAID, type of gastro protection and upper GI event during admission were also recorded.

Patients were risk stratified according to following[7-9]:

- age>65 years
- major systemic co-morbidities
- past history of upper GI haemorrhage or peptic ulcer disease
- concomitant use of oral steroids
- concomitant use of anticoagulants
- high or twice normal dosage of NSAID.

Those with no risk factor were deemed to have a "background risk" of GI haemorrhage whereas those with one or more risk factors were at "high risk".

Evidence-based guidelines state that those at "high risk" should be prescribed gastro protection (omeprazole 20mg) whereas those at "background risk" should not[7]. Adherence to these guidelines was recorded. Statistical data analysis involved the use of the Fishers Exact Test via the Simple Interactive Statistical Analysis package[10].

Pre-intervention, an audit was performed over an 18-week period between April and August 2008 inclusive (451 admissions). Various strategies aiming to improve practice were implemented (education of surgical prescribers and pharmacists, and posters to raise awareness of guidelines). Post-intervention, the same assessment of prescribing practice over a 9-week period between September and November 2008 (193 admissions) was performed.

Results

Pre-intervention

Of a total of 451 admissions, 100 patients (22%) satisfied the inclusion criteria, including 37 males and 63 females, giving a male:female ratio of 1:1.7. Of these patients, 75% (75 patients) met the definition of "high risk".

Of the 100 eligible patients in the audit, 25 patients (25%) had no risk factors, 15 patients (15%) had 1 risk factor, 29 patients (29%) had 2 risk factors, 24 patients (24%) had 3 risk factors and 7 patients (7%) had 4 risk factors for adverse GI events.

Of the "high-risk" patients, 67/75 patients (89%) had age ≥65 as a risk factor. 49/75 patients (65%) had concomitant use of anticoagulants, 38/75 patients (51%) significant co-morbidities, 10/75 patients (13%) concomitant prescription of glucocorticoids and 5/75 patients (7%) history of peptic ulcer disease/GI bleeds.

Adherence rate to the national evidence-based guidelines for the prescription of gastro protective agents with NSAID was 19/75 patients (25%)7.

Sixty-seven out of 100 (67%) patients were prescribed diclofenac and 33/100 (33%) patients were prescribed ibuprofen.

Of the patients who received gastro protection, 16/19 patients (84%) received omeprazole 20mg once daily, 1/19 patients (5%) received lansoprazole 30mg once daily and 2/19 patients (11%) received ranitidine. No patient had documented intolerance of any gastro protective agent.

The likelihood of a patient receiving gastro protection significantly increased with the presence of high risk as compared to background risk (p=0.002). None of the patients with background risk were co-prescribed gastro protective agents (see Table 1 and Graph 1).

Post-intervention

Of a total of 193 admissions, 49 patients (25%) satisfied the inclusion criteria, including 28 males and 21 females giving a male:female ratio of 1.3:1. Twenty-six patients (53%) met the definition of high risk.

Of the 49 eligible patients, 23 patients (47%) had no risk factor, 14 patients (29%) had 1 risk factor, 11 patients (22%) had 2 risk factors, and 1 patient had 3 risk factors (2%) for adverse GI events.

Of the high-risk patients, 20/26 patients (77%) had age ≥65 as a risk factor, 1/26 patients (4%) had concomitant use of anticoagulants, 17/26 patients (65%) significant co-morbidities and 3/26 patients (12%) concomitant prescription of glucocorticoids.

Adherence rate to the national evidence-based guidelines for the prescription of gastro protective agents with NSAID was 19/26 patients (73%)[7].

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Sunil Sharma, Gokulan Phoenix, James Chan, Graham Sleat and Alastair Graham

Thirty-six out of 49 (74%) patients were prescribed diclofenac and 13/49 (27%) patients were prescribed ibuprofen.

Of the patients who received gastro protection, 13/21 patients (62%) received omeprazole 20mg once daily, 8/21 patients (38%) received lansoprazole 30mg once daily and none received ranitidine. No patient had documented intolerance of any gastro protective agent.

The likelihood of a patient receiving gastro protection significantly increased with the presence of high risk as compared to background risk ($p<0.001$). Two of the patients with background risk were co-prescribed gastro protective agents (see Table 2 and Graph 2).

Discussion

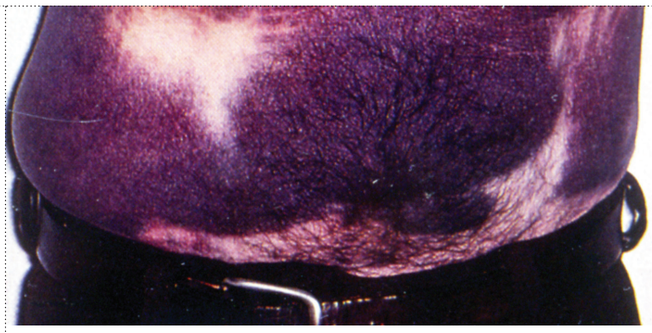
The pre-intervention adherence to evidence-based guidelines for the safe prescription of NSAID in our trauma unit was low (25%). Safe prescription of NSAID is particularly important in this context because trauma patients tend to be elderly, which in itself is a risk factor for upper GI haemorrhage (86% of patients in our audit were aged ≥65). Given the widespread use of NSAID, the incidence of orthopaedic trauma patients and the proportion of such patients with 1 or more risk factors, it is clear that the safe prescription of NSAID, in this context, is a major clinical issue.

Post-intervention, adherence to guidelines improved dramatically from 25% to 73%. Furthermore, a smaller proportion of NSAID receivers fell into the high-risk category from 75% to 57%. Ranitidine, which is not a recommended gastro protective agent according to evidence-based guidelines, was prescribed in 11% of cases pre-intervention but was no longer used post-intervention[7].

In this audit 52% of patients had 2 or more risk factors. Although this audit was not powerful enough to allow quantitative analysis on the increase in risk of GI bleed for each risk factor or combination of risk factors, it would be logical to assume that multiple risk factors will place the patient at greater risk than a single risk factor. It might therefore be prudent to consider alternative analgesia in this subgroup of patients.

This audit was limited by the fact that the sample size was not powerful enough to demonstrate the relationship between NSAID prescription and acute GI bleed. However, there is sufficient evidence in the literature to show that NSAID use, both short term and chronic, can lead to upper GI haemorrhage[1].

The guidelines that we have used as the standard for this audit were based on elective orthopaedic patients, rather than the acute orthopaedic trauma setting. Assuming that the pathogenesis of upper GI bleed secondary to NSAID is consistent, these guidelines should apply to all patients receiving NSAID.



In view of the findings of this audit, we are currently in the process of updating the Local Trust Policy regarding the safe prescription of NSAID according to the evidence-based guidelines in collaboration with the local pharmacy and gastroenterology departments. These results demonstrate that the intervention strategies implemented were effective and we urge other departments to pursue similar measures to minimise the risk of upper GI side effects in NSAID users.

Conclusions

The majority of trauma admissions are at high risk for developing gastrointestinal haemorrhage.

Initial adherence to national guidelines for safe prescription of NSAID in our trauma unit was low, but improved dramatically with intervention, including education of prescribers and pharmacists. Awareness of gastro protection guidelines must be raised in trauma units to minimise the risk of GI complications.

Number of patients on gastro protection pre-intervention				
		Gastro protection		Total
		No	Yes	
Level of risk	Background risk	25	0	25
	High risk	56	19	75
Total		81	19	100

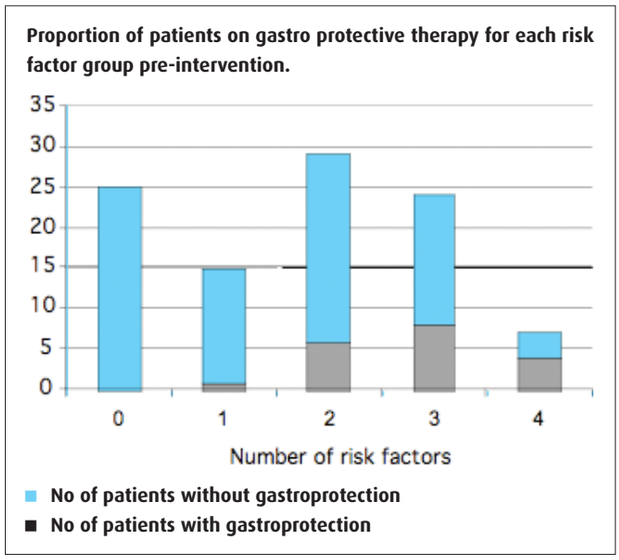
Table 1: Number of patients on gastro protective therapy for each risk factor group pre-intervention.

Number of patients on gastro protection post-intervention				
		Gastro protection		Total
		No	Yes	
Level of risk	Background risk	21	2	23
	High risk	7	19	26
Total		28	21	49

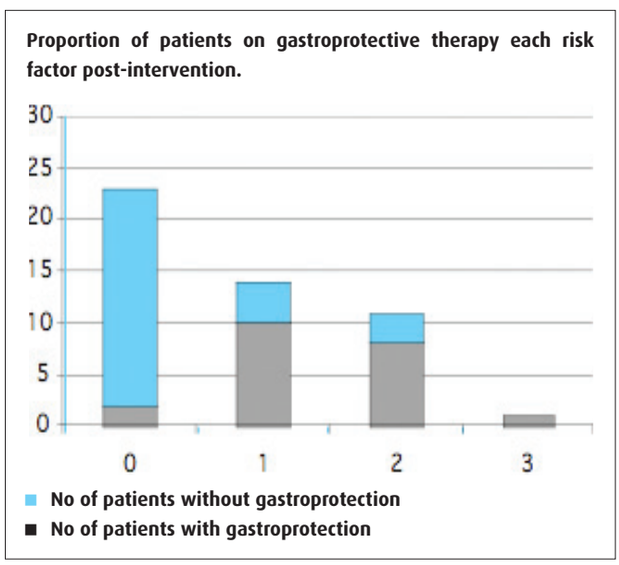
Table 2: Number of patients on gastro protective therapy for each risk factor group post-intervention.

CLINICAL AUDIT: THE USE OF GASTRO PROTECTION IN TRAUMA PATIENTS RECEIVING NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

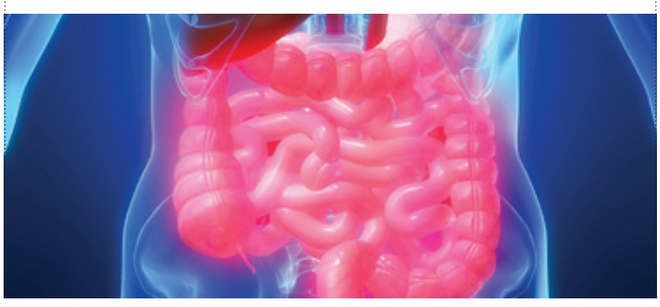
Sunil Sharma, Gokulan Phoenix, James Chan, Graham Sleat and Alastair Graham



Graph 1: Proportion of patients on gastro protective therapy for each risk factor group pre-intervention.



Graph 2: Proportion of patients on gastro protective therapy for each risk factor group post-intervention.



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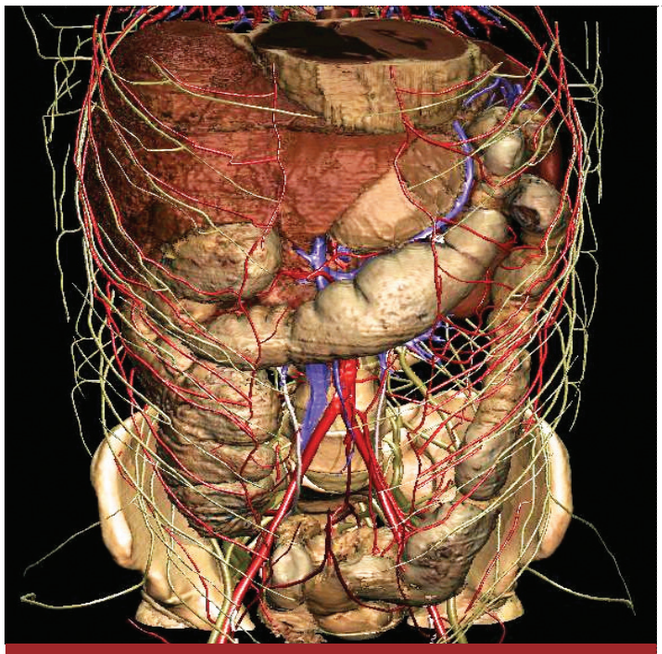
UROLOGICAL EMERGENCIES

Paul Lyon, Hari Ratan, Kenneth Elder, Stephen Thomas and Gill Turner



"A 58-year-old patient (smoker) admitted to the Emergency Department by his GP with severe right-sided loin pain radiating into the right lower abdomen."
Patient Management.

"A 58-year-old patient (smoker) admitted to the Emergency Department by his GP with severe right-sided loin pain radiating into the right lower abdomen."

Learning objectives

- Awareness of GP referral pathways in urological emergencies.
- Recognition of the acutely ill and the initial assessment.
- Diagnostic features and appropriate investigation of ureteric colic.
- Practicalities in the medical management of urinary tract stones.
- Indications and options in surgical management of urinary tract stones.
- Discharge planning and outpatient follow-up for urinary tract stones.

Introduction

Ureteric colic, traditionally referred to as renal colic, is a commonly presenting urological emergency. The estimated lifetime risk of developing a kidney stone is around 12% with a lifetime prevalence of 2-3%[1].

British Association of Urological Surgery (BAUS) guidelines are available for the acute management of first presentation of renal and ureteric lithiasis. More detailed information on the active removal of stones and appropriate follow-up can be found in the European Association of Urology (EAU) guidelines for urolithiasis. The discussion here aligns with these guidelines.

Clinical scenario

GP admission to the Emergency Department; a 58-year-old man with a previous medical history of stable angina who is otherwise well with no other medical or surgical history of note. The patient presents with a 2-day history of sudden onset, intermittent, excruciating right-sided loin pain radiating to the groin region. The patient has vomited twice in the last 24 hours and is feverish with rigors.

What is the most likely diagnosis and what are the differentials?

The patient is presenting with the classic symptoms of right-sided ureteric colic. Urological differentials include severe urinary tract infection and pyelonephritis. As for any acute surgical presentation it is useful to apply a surgical sieve in order to classify the differential diagnoses, as illustrated for ureteric colic in Table 1.

Category	Differential diagnoses
Traumatic	Urinary tract injury
Infectious	Pyelonephritis
	Appendicitis
	Pelvic inflammatory disease
	Urinary tract infection
	Abscess
	Sepsis
Inflammatory	Pancreatitis
	Diverticulitis
	Peptic ulcer disease
Vascular	Leaking aortic aneurysm
	Dissecting aortic aneurysm
	Pelvic thrombophlebitis
Psychosomatic	Somatization disorder
	Munchausen syndrome
Neoplastic	Ectopic pregnancy
	Bowel obstruction
	Bowel perforation

Table 1: Differential diagnoses for ureteric colic.

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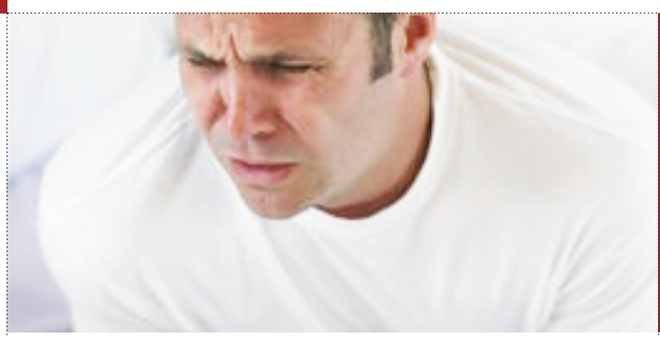


Patients in severe pain from ureteric colic may attend the Emergency Department (ED) directly without first seeing a General Practitioner (GP).
Patient Management.

Where should a GP refer a patient with this presentation?

Patients in severe pain from ureteric colic may attend the Emergency Department (ED) directly without first seeing a General Practitioner (GP). Patients referred by the GP with ureteric colic may be admitted to either the ED or a dedicated Urology Assessment Unit (UAU) if the service is available within the trust. GPs often refer patients with a clear-cut presentation of severely symptomatic ureteric colic accompanied by microscopic haematuria for direct admission to UAU.

In this particular case, the patient is a known arteriopath and it is important to consider other differential diagnoses including a leaking or dissecting aortic aneurysm. For this reason the GP has made a sensible decision to send this patient to the ED for full assessment prior to admission to the UAU. If there is any suspicion of aortic aneurysm pathology, for example, if there is hypotension, a pulsatile abdominal mass or pain radiating into the back, an urgent abdominal CT is indicated.

What information would you like to review on admission?

Timely review of admission observations including pulse rate, blood pressure, respiratory rate and temperature is a critical first step and will guide you as to whether the patient is acutely unwell requiring immediate resuscitation. If the patient is afebrile, haemodynamically stable and appears well it may be that the triage staff have assigned a lower priority to this patient. Although prioritisation at triage is a useful guide, it is important to use your own clinical judgement as patients can deteriorate quickly.

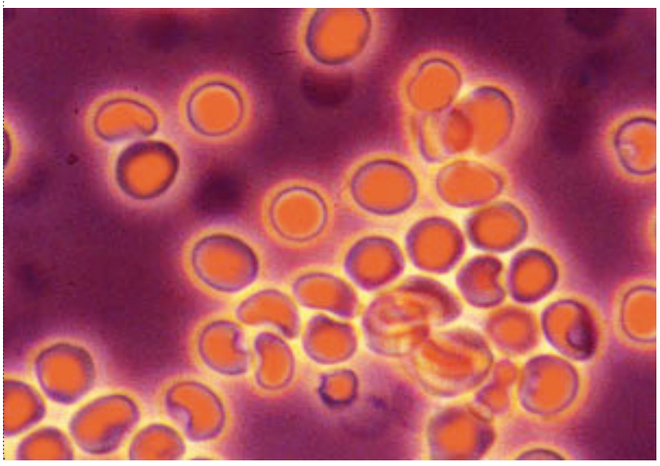
A tachycardia alone, or if combined with an elevated blood pressure, may be a physiological response to pain. A patient with significant pyrexia is more likely to have pyelonephritis than ureteric colic. A low blood pressure and tachycardia is more suggestive of sepsis of unknown origin or a dissecting aortic aneurysm. Remember that sepsis may present with a significantly reduced temperature as well as a spiking temperature.

As the receiving doctor in the ED it is prudent to efficiently review any other information available on admission. This may include a referral letter from the GP, documentation provided by the paramedics and admission information provided by the triage nurses. This may be particularly useful if the patient is in too much pain to give you a reliable history.

Often GPs will dip the patient's urine before admitting to the ED with a query ureteric colic and the results may be documented in the referral letter. Mid-stream urine dip is sensitive, however, not specific, in the diagnosis of ureteric colic; over 90% of positive presentations are accompanied by microscopic haematuria[2]. Thus a dip negative for haematuria does not exclude ureteric colic but makes the diagnosis less likely. A dip which is leukocyte and nitrite positive is indicative of urinary tract infection or pyelonephritis. Non-traumatic causes of macroscopic haematuria are often painless and although a stone may present this way it is more commonly due to malignancy of the renal tract. There are many differential diagnoses of haematuria and the most common causes are summarised in Table 2.

Causes of haematuria	
Urinary tract malignancy	Glomerular disease
Urinary tract stones	Renal vein thrombosis
Urinary tract infection	Alport's syndrome
Acute pyelonephritis	Thin basement membrane nephropathy
Bleeding diathesis	Sickle cell disease / trait
Trauma	Severe exercise
Haemorrhagic cystitis	Schistosomiasis
Polycystic kidneys	Angiomyolipoma

Table 2: The common differential diagnoses for both microscopic and macrosopic haematuria.



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What is your initial assessment of the patient?

The patient should be assessed using the airway, breathing and circulation (ABC) principles and resuscitated where appropriate. Every patient admitted to the ED requires a focused history and examination, which must be appropriately documented. A detailed pain history is crucial and may lead you to suspect other differentials. In particular ask about previous stones or urological interventions, does the patient have known renal impairment or a solitary kidney? On examination, is the patient lying completely still, suggestive of peritonism or writhing, classically associated with ureteric colic. If the patient has ureteric colic you should expect a soft abdomen with flank tenderness. A presentation with abdominal guarding and rebound tenderness is more suggestive of abdominal pathology. Palpate for a suprapubic mass that is dull to percussion, indicative of a distended bladder in urinary retention.

What is the initial management and which investigations are required?

With a diagnosis of ureteric colic in mind it is mandatory to take full blood count, urea and electrolytes. A fresh admission MSU is also essential. If abnormalities are found on the dip, it should be sent for microscopy, culture and sensitivity, which will guide antibiotic cover in the case of urinary tract infection or pyelonephritis. It is important to keep an open mind as the patient may present a different picture to the diagnosis the GP was suspecting. Further blood tests and investigations may be ordered as necessary. Always remember to check urine or serum beta-HCG in a woman of childbearing age to exclude ectopic pregnancy.

In this presentation, the observations were stable and the patient was afebrile. A full blood count revealed haemoglobin of 14.2 g/dl and a white blood cell count of 13.7×10^9/L. Blood chemistry revealed sodium of 136 and a potassium of 3.8mmol/L, a urea of 7.3 and a creatinine of 114µmol/L. Urine dip was positive for microscopic haematuria, nitrites and leucocytes.

What imaging does the patient require?

The patient should be imaged according to the BAUS Guidelines. First, a kidney, ureter and bladder (KUB) radiograph should be arranged, as despite its limitations, 60% of stones are visible on plain film. If a stone is visualised a non-contrast CT (NCCT KUB) is the gold standard imaging, as this will confirm the diagnosis and assist in planning of surgical intervention. CT has the advantages of more detailed anatomical resolution allowing for detection of alternative diagnoses and the ability to visualise radiolucent (uric acid and xanthine) stones.

Intravenous urogram (IVU) was previously the gold standard and is still used first line in some centres where CT is impractical, provided the patient does not have any contraindications, as shown in Table 3. IVUs carry the risk of contrast reaction (anaphylaxis) and are more time consuming to perform than CT. In practice, where creatinine is over 150 most centres would opt for CT in preference to IVU as when there is impaired renal function contrast excretion is impaired resulting in IVUas in renal impairment contrast excretion is impaired resulting in IVU images which are difficult to interpret.

Relative contraindications	Absolute contraindications
Diabetic on metformin	Contrast allergy
Serum creatinine over 200mol/L	
Myelomatosis	
Pregnancy	
Untreated hyperthyroidism	

Table 3: Relative and absolute contraindications for IVU.

In the case of impaired renal function, N-Acetylcysteine combined with intravenous fluids may be used to help protect the kidneys against contrast-induced nephropathy. There is increased risk of contrast-induced nephropathy in diabetic patients even with normal renal function[3]. There is a further risk of lactic acidosis in diabetics on metformin[3]. This is because metformin is exclusively renally excreted and worsened renal impairment may lead to its accumulation. Consequently, it is recommended to withold metformin for 48 hours before and after the administration of contrast where possible.

In pregnancy in order to minimise radiation dosage for the foetus, renal tract ultrasound is the imaging modality of choice and IVU or NCCT are reserved for the more complex cases.

At the time of presentation, the hospital policy for query renal colic indicated IVUs as the first line imaging modality. The radiological series reveals an obstructed right ureter with mild dilation of the right pelvicalyceal collecting system secondary to a 6mm radio-opaque stone at the right pelvi-ureteric junction, as shown in Image 1. The stone is difficult to see without magnification, however, the dilatation and paucity of contrast in the right ureter are easy to visualise. Also be aware to check for an increasingly dense nephrogram, a hallmark of high-grade obstruction. In a dense nephrogram a high concentration of contrast can be seen confined to the renal cortex and medulla as the passage of urine to the pelvicalyceal collecting system is delayed, even after a significant interval post contrast injection. Image 2 shows an example of a dense nephrogram taken from a different subject.

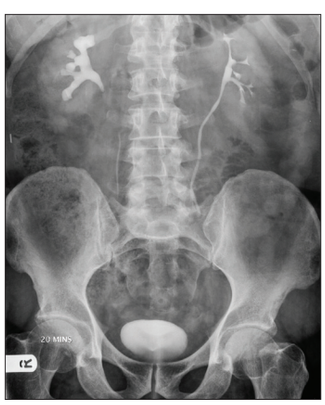

Image 1: Twenty-minute IVU illustrating a 6mm proximal right ureteric stone at the PUJ causing a moderate grade of obstruction and free drainage on the left.

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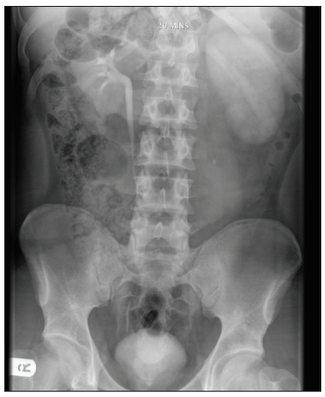


Image 2: Twenty-minute IVU illustrating a left-sided dense nephrogram secondary to high-grade obstruction with free drainage on the right.

In summary, investigations have revealed that the patient has a stone obstructing the left upper urinary tract, complicated by urinary tract infection.

Given the diagnosis, does this patient require admission?

Patients presenting to the ED with ureteric colic and confirmed urolithiasis may be discharged for conservative (expectant) management in the outpatient setting provided they are pain controlled with oral analgesia and have normal observations and bloods. These patients should be asked to return to the ED if there is any deterioration in their clinical condition. Follow-up for these patients will typically consist of outpatient IVU, NCCT or renal tract ultrasound before urology outpatient review.

However, confirmed cases of ureteric colic complicated by obstruction and/or infection, as in this case, require urgent referral to the urologists for admission. The indications for admission of patients presenting with suspected ureteric colic are shown in Table 4. In particular, note that a leaking abdominal aortic aneurysm may present with similar symptoms to left-sided ureteric colic and consequently men over the age of 60 should be admitted due to the risk of a misdiagnosis.

Indications for admission	
Sepsis, urinary infection or pyrexia	Severe symptoms
Urinary tract obstruction	Pain unresponsive to analgesia
Age over 60 (especially if male)	Solitary or transplanted kidney
Pre-existing renal impairment	Bilateral stones
Uncertainty of diagnosis	No reliable social support
Pregnancy	Patient preference
Dehydration secondary to vomiting	Delayed menstrual period

Table 4: Indications for admission in suspected ureteric colic.

How should this patient be medically managed?

Given that the pain is severe enough to induce vomiting this patient requires analgesia at the time of presentation, before confirming the diagnosis with imaging. In the absence of any contraindications, non-steroidals (NSAID), such as diclofenac, would be the first choice of analgesia in ureteric colic due to the secondary effect of reducing ureteral oedema. NSAID are nephrotoxic and so ensure renal function is checked before prescribing. They would usually be combined with regular paracetamol. In addition, opiates (morphine) may be used in severe cases.

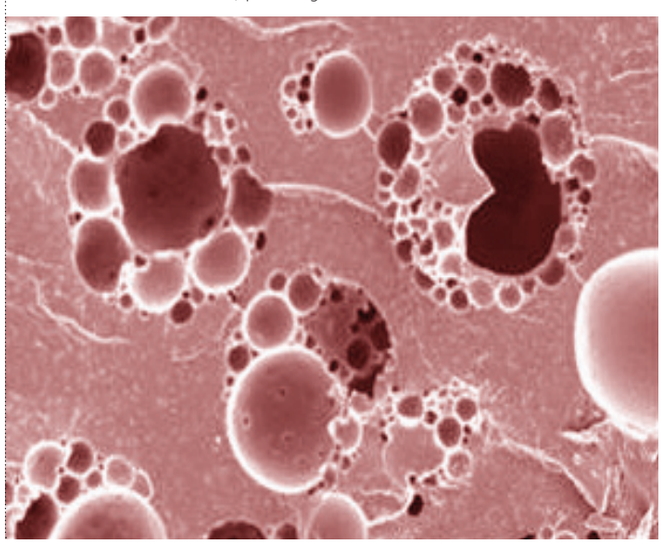
Anti-emetics are of benefit in reducing morbidity for the patient and avoiding electrolyte disturbance and dehydration, and are especially important if opiates have been administered.

High intake of oral fluids should be encouraged to rehydrate the patient, however, this may prove difficult if there is vomiting or anorexia. If the patient's oral intake of fluids is poor for any reason then an intravenous infusion of fluids may be considered appropriate. As always, care must be taken when giving fluids in patients with congestive cardiac failure.

If the patient is demonstrating signs of infection or sepsis, for example, a deteriorating clinical condition, pyrexia, raised white cells or positive urine dip then appropriate antibiotics are indicated as per trust guidelines.

The use of an alpha1 adrenergic antagonist ("alpha-blocker"), such as tamsulosin, has been shown to be effective for ureteric colic[4]. In particular this class of drugs act at the distal ureter to reduce ureteral contractions and spasm hence allowing lower ureteral stones to pass. There is currently no published evidence that this class of drug is effective for more proximal stones, as in this case.

It is important to consider thromboprophylaxis for any inpatient who is largely confined to bed rest, as per local trust guidelines. Thromboembolic deterrent stockings and subcutaneous injections of low molecular weight heparin are the standard interventions, providing there are no contraindications.



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What are the indications for active stone removal?

Although passage of renal stones is notoriously difficult to predict, Table 5 provides a rough rule of thumb for predicting the chance of passing a ureteric stone spontaneously[5].

Size	Chance of spontaneous passage
<4mm	80%
5–7mm	50%
>7mm	Unlikely

Table 5: Approximate percentage of ureteric stones of given size passing spontaneously.

The indications for active intervention, either stone removal or decompression of obstruction via stent or nephrostomy, are listed in Table 6.

Indication for active intervention	
Adequate analgesia not achieved	Obstructed solitary kidney
Obstruction associated with infection	Bilateral obstruction
Risk of pyelonephritis or urosepsis	Size >7mm

Table 6: Indications for active intervention in renal tract stones.

In practice, clinically stable patients with a newly diagnosed ureteric stone of diameter <10mm should undergo an initial period of observation provided their symptoms are controlled. These patients should be treated medically before proceeding to surgical intervention, as there is a reasonable chance for spontaneous passage.

In this case the patient has an approximately 50% chance of passing the stone and expectant management is indicated.

What are the surgical treatment options for renal tract stones?

In a febrile patient with a leucocytosis who responds poorly to intravenous antibiotics, there may be a pyonephrosis requiring emergency decompression via a percutaneous nephrostomy or ureteric stent insertion. Stone removal can be performed electively following decompression once the infection has resolved.

The advent of endoscopic techniques and non-invasive technologies has meant that stone removal has become safer and a more widely available treatment option.

In the case presented here the stone would be accessible endoscopically and the primary treatment would be ureteroscopy and lasertripsy. In this procedure a ureteroscope is passed and holmium: YAG laser energy is used to fragment the stone, with the intention that the fragments will spontaneously pass through the urinary tract over the following few days.

Extracorporeal shock wave lithotripsy (ESWL) can be used for stones in the kidney or ureter and if facilities were available it could be used as an alternative primary intervention in this case. ESWL involves focusing an externally-generated shockwave onto the stone in order to fragment it. Fluoroscopy or ultrasound scanning may be used to locate the stone. The ESWL technology is less effective for cystine stones.

In rare cases where the less invasive techniques fail, laparoscopic or open surgery for stone removal may be considered, but both have respectively increased complication rates and require more stringent patient selection[6].

What is the long-term management and outpatient follow-up?

Continuation of oral antibiotics may be required in the community in accordance with local trust guidelines and are especially important in triple phosphate stones which are formed by urease-producing micro-organisms.

Stones should be captured for composition analysis when possible, for example, if passed spontaneously or if removed surgically. Serum markers for renal stones are required if not already available and include calcium, phosphate and urate. Any metabolic abnormalities require appropriate investigation, including urinalysis, and treatment where indicated.

Repeat urea and electrolytes and renal tract imaging as an outpatient may be indicated to ensure renal function is restored, which is particularly important if the patient has had an obstructive uropathy, as in this case. Results should be reviewed in a urology outpatient clinic, which provides an opportunity to arrange further interventions if necessary, such as stent removal. If obstructive symptoms persist decisive operative management should be carried out urgently.

Increased intake of oral fluids can be used as a prophylactic measure in known stone formers of any type and may be the only intervention required in those at low risk of recurrence. Urinary alkalisation may be used for uric acid and cystine stones and can be achieved with potassium citrate or sodium bicarbonate. Urinary acidification may be used in triple phosphate stones and can be achieved with ammonium chloride or methionine. In the case of calcium oxalate and phosphate stones, dietary restrictions of animal protein and oxalate may be beneficial. High levels of oxalate are found in tea, chocolate, rhubarb and spinach. Interestingly, note that calcium intake should not be restricted for recurrent calcium stones as there is an inverse relationship between calcium intake and stone formation[7].

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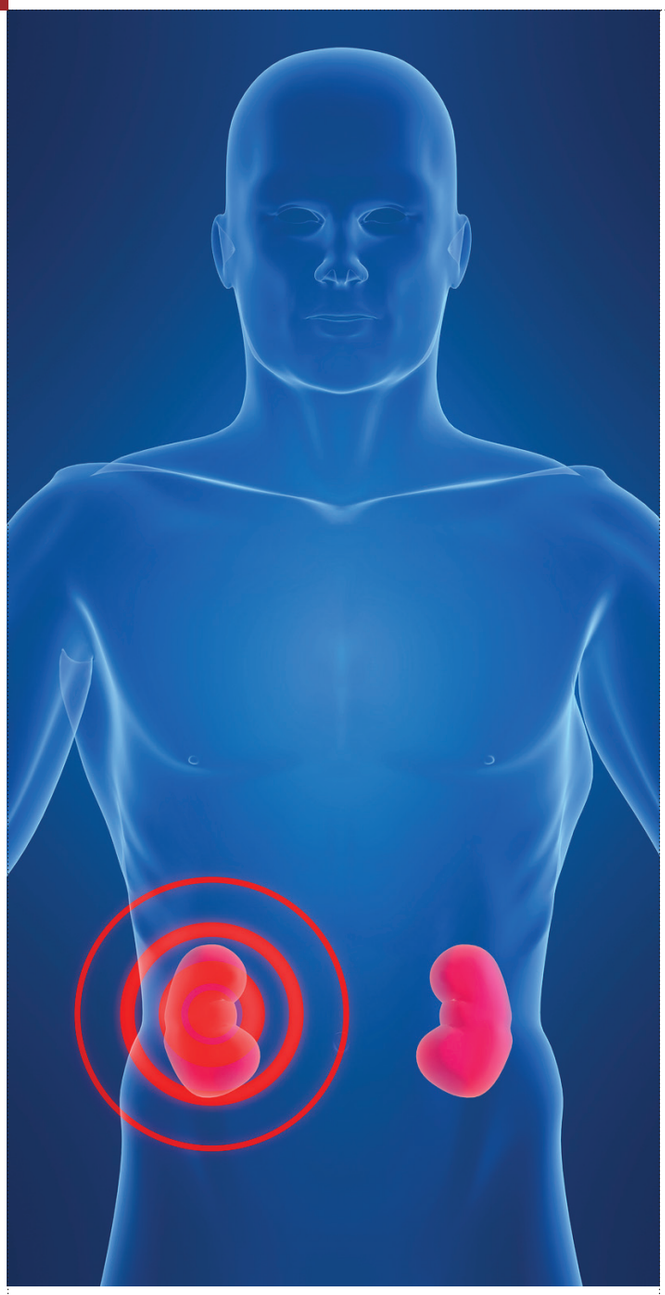

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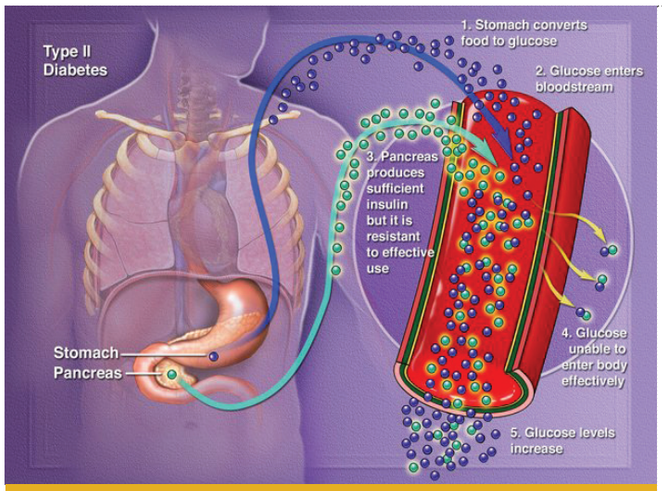
DIABETIC HYPERGLYCAEMIC CRISES – A NEWLY-DIAGNOSED DIABETIC PRESENTING WITH MIXED DIABETIC KETOACIDOSIS AND HYPERGLYCAEMIC HYPEROSMOLAR SYNDROME

Joanna Wilson and Barbara Philips



Type 2 diabetes mellitus is increasing in prevalence and patients may present in hyperglycaemic crisis. Good Clinical Care.

Abstract

Introduction: type 2 diabetes mellitus is increasing in prevalence and patients may present in hyperglycaemic crisis. We present a case of mixed Diabetic Ketoacidosis (DKA) and Hyperglycaemic Hyperosmolar syndrome (HHS) in a previously unknown diabetic.

Case Presentation: a 49-year-old man presented to the A&E department with polydipsia, polyuria, weight loss and confusion. Biochemical testing revealed evidence of both DKA and HHS. He was transferred to the intensive care unit for further management.

Discussion: it is essential to distinguish between DKA and HHS because of differences in management, with outcome implications. HHS patients are at high risk of cerebral complications and mortality of up to 50% has been reported. Regimens aimed at treating patients with DKA, may exacerbate risk to patients with HHS who should be managed to minimise the risk of cerebral oedema.

Case Presentation

A 49-year-old white British man was admitted to the A&E department following a 2-week history of polydipsia, polyuria and weight loss. He had a past medical history of hypertension, personality disorder and substance misuse (alcohol, benzodiazepines and cannabis).

On examination he had a heart rate (HR) of 120 beats per minute, respiratory rate (RR) of 28 breaths per minute, oxygen saturation (SpO2) of 91% breathing air and his blood pressure (BP) was 145/100mmHg. He had a Glasgow Coma Score (GCS) of 12/15 but no focal neurology was detected. Examination of his chest and abdomen was unremarkable. Table 1 shows the biochemical investigations from admission to day four.

	Admission	8 hours	Day two
Glucose mmol/L	50.7	19.7	9.3
Sodium (Na+) mmol/L	145	154	157
Potassium (K+) mmol/L	4.1	4.4	3.9
Chloride (Cl-) mmol/L	97	114	121
Bicarbonate mmol/L	18	24	22
Urea (U) mmol/L	16.6	12.9	13.0
Creatinine (Creat) mmol/L	224	158	202
C Reactive Protein (CRP) mg/L	47.4	43.8	74.9
Haemoglobin (Hb) g/dl	18.3	15.6	15.2
White Cell Count (wcc) x 10^9/L	13	13.2	12.7
Effective Serum Osmolality mOsm/Kg	340	328	327

Table 1: Trend in biochemistry values from admission to day four.

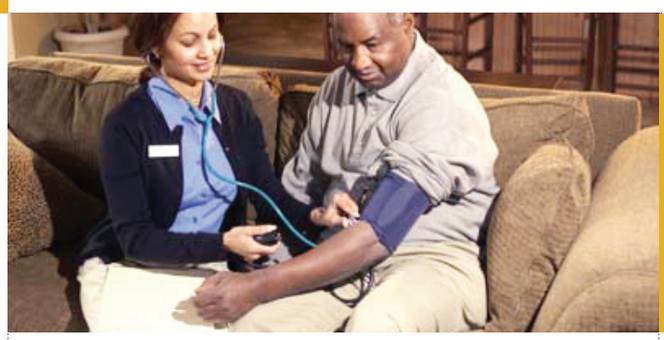
Liver enzymes were mildly deranged. Urinalysis was strongly positive for ketones and glucose. He had a compensated metabolic acidosis pH 7.31, lactate of 2.3, arterial partial pressure of carbon dioxide ($PaCO_2$) of 2.5kPa and base excess (be) of -9.4. His chest X-ray was unremarkable.

He was given a bolus of 10 units of short-acting insulin (Actrapid) followed by an infusion of short-acting insulin at 6 units per hour (U/h). Three litres of intravenous 0.9% sodium chloride (NaCl 0.9%) were infused over 3 hours and within 6 hours, his serum glucose concentration had decreased to 19mmol/L. Although initially, he made a clinical improvement he later became increasingly agitated and his GCS decreased to 10/15. He was intubated and transferred to the intensive care unit (ICU).

In ICU fluid resuscitation was continued with NaCl 0.9%. An insulin infusion was continued at a minimum rate of 2U/h but to prevent further rapid decrease in his serum glucose concentration, 10% dextrose was administered to maintain a glucose concentration of approximately 18mmol/L. Potassium (K+) was given as required to maintain K+ >4.0mmol/L. He developed a pyrexia (temperature >38°C) and a repeat chest X-ray revealed possible consolidation of his left lower lobe and antibiotics were started for a presumed chest infection.

DIABETIC HYPERGLYCAEMIC CRISES – A NEWLY-DIAGNOSED DIABETIC PRESENTING WITH MIXED DIABETIC KETOACIDOSIS AND HYPERGLYCAEMIC HYPEROSMOLAR SYNDROME

Joanna Wilson and Barbara Philips



Diabetic hyperglycaemic crisis, accounted for 0.2% of emergency admissions to A&E in 2005–2006 in England[2].
Good Clinical Care.

His sodium increased from 145mmol/L to 157mmol/L over the next 2 days (see Table 1). He developed an acute kidney injury (AKI) (peak urea and creatinine, 24mmol/L and 481µmol/L respectively) which was attributed to both severe dehydration and sepsis. The AKI resolved as he recovered from his critical illness. His liver function tests deteriorated in a similar fashion again improving as he recovered.

He required artificial ventilation for 4 days. Initial attempts to extubate him failed because of agitation. A computed tomography (CT) scan of his head on day three excluded obvious intracranial pathology but his agitation continued and eventually he was given a percutaneous tracheostomy which allowed him to be successfully weaned from the ventilator. He finally settled as his electrolytes normalised and he was transferred to a medical ward to be followed-up by the diabetologists with regular reviews by the diabetic nurse specialist and dietician. He is now being followed-up by the diabetic team as an outpatient and his diabetes mellitus is being managed using oral hypoglycaemic agents.

Discussion

Diabetic hyperglycaemic crisis, accounted for 0.2% of emergency admissions to A&E in 2005–2006 in England[2]. Mortality in experienced centres should be <5% for adults with DKA but may be as great as 10–50% for HHS[1, 3].

DKA and HHS are recognised complications of diabetes mellitus but rather than being entirely separate conditions, they may be considered as the two ends of a spectrum of illness, between total insulin deficiency (DKA) and severe insulin resistance (HHS)[4]. Patients can present with a mixed DKA/HHS picture and although this may account for as many as 30% of diabetic emergencies[5,6], it is poorly recognised. Differentiating between the conditions is important. Patients with HHS who are treated with DKA regimens are at risk of severe and potentially fatal complications[1, 5-6]. Patients with a mixed DKA/HHS presentation should be treated in a similar manner to patients with HHS. This is discussed below.

DKA has a rapid onset (1–3 days) and patients present with polyuria, polydipsia, weight loss and weakness. Patients are dehydrated with dry mucosa, lax skin turgor, tachycardia and tachypnoea. Kussmaul-Kien respiration (rapid and deep breathing) is classical in DKA and exhaled breath may have the typical sweet, fruity odour of ketones. They may be hypotensive or even in shock and signs and symptoms of associated underlying conditions (e.g. infection) or co-morbidities should be sought[1, 4, 6–9].

HHS is generally associated with older patients with type 2 diabetes mellitus and thus patients with HHS often have significant co-morbidities. Traditionally it is not considered as a diagnosis of young patients but HHS is increasing in frequency in all age groups (including children) and this has been attributed to the increasing prevalence of type 2 diabetes associated with obesity. Since it is caused by a resistance to insulin rather than a total insulin deficiency, HHS usually develops over a number of days. Patients again complain of polydipsia and polyuria and may present with signs of dehydration and shock, but Kussmaul-Kein breathing is not observed and patients may present very confused or even in coma[1, 4, 6–9].

Typically patients with a mixed DKA/HHS are young obese patients with type 2 diabetes mellitus[4]. Presentation has features of both DKA and HHS. The differentiation between and diagnosis of DKA, HHS and mixed DKA/HHS is biochemical (see Table 2) and patients may exhibit abnormalities anywhere along the spectrum between pure DKA and pure HHS. Important variations include mixed DKA/HHS and ketosis-prone type 2 diabetes mellitus. In this latter condition a patient with type 2 diabetes mellitus may present with the features of DKA, whether or not it is considered a mixed DKA/HHS syndrome depends on the serum glucose and the consequent serum osmolality. Why some patients develop a ketoacidosis and some do not, is not fully understood but thought to be related to a relative or absolute lack of insulin and the concentration of circulating free fatty acids[1].

DIABETIC HYPERGLYCAEMIC CRISES – A NEWLY-DIAGNOSED DIABETIC PRESENTING WITH MIXED DIABETIC KETOACIDOSIS AND HYPERGLYCAEMIC HYPEROSMOLAR SYNDROME

Joanna Wilson and Barbara Philips

	DKA	DKA/HHS mixed	HHS
Plasma Glucose (mmol/L)	>14.0<33	>33	>33
Arterial pH	<7.3	<7.3	>7.3
Serum Bicarbonate (mmol/L)	<18	<18	>15
Plasma Ketones	+++	+/++	Negative
Urine Ketones	++/+++	++/+++	Negative
Effective Serum	Variable	>320	>320
Osmolality (mOsm/kg)			
Anion Gap	>10	>10	<12
Conscious level	variable	confusion	Depressed/ coma

Table 2: Biochemical diagnosis of the diabetic hyperglycaemic crisis[4].

The importance of distinguishing between the conditions lies in differences in management with direct impact on outcome. Patients presenting with a mixed DKA/HHS are often mistaken and treated as DKA[4]. Fluid resuscitation and glucose correction is too aggressive causing rapid fluid shifts between the extracellular and intracellular compartments, thus increasing risk of complications, including cerebral oedema[1, 3, 5]. If in doubt, it is advised to treat patients as if they have HHS[4].

Patients with HHS are much more dehydrated than patients with DKA. In DKA, due to rapid onset of ketone production, a severe metabolic acidosis develops and patients seek help earlier. The period of hyperglycaemia prior to admission is shorter and the blood glucose rarely exceeds 33mmol/L. Although 33mmol/L is an arbitrary limit, the majority of patients with DKA will have a lower glucose concentration than this and if greater, osmolality issues arise and the patient should be classified as a mixed DKA/HHS. In HHS the pathophysiology is one of insulin resistance and the onset of symptoms is more insidious. The patient has sufficient endogenous insulin to prevent ketone production and presents with a 1–2-week history. This results in profound dehydration due to the prolonged hyperglycaemic period. For patients on the spectrum between DKA and HHS, the serum osmolality is determined by the serum glucose concentration, and the acid-base balance is determined by ketone and lactate production within the context of the patient's ability to develop a respiratory compensation and their acute renal function. Patients with a pure DKA rarely have a markedly increased lactate and its generation in HHS may be considered a reflection of the profound dehydration consistent with a hyperosmolar syndrome. The patient described, presented with pH 7.31 which might be considered high given increased ketones and lactate (2.4mmol/L) and a base excess of 9.4. However, having normal lungs, he was able to partially compensate by hyperventilation. In this case, arterial blood samples were measured but venous blood samples may be used; pH measurements are on average 0.04 units lower on venous compared with arterial sampling, a figure very unlikely to have clinical significance[10]. The interpretation of venous lactate concentrations are, however, a little more problematic. The lactate sampled peripherally is a little more dependent on the conditions of the local vascular bed being drained than pH.

Patients have a total body deficit of water, sodium and potassium in all hyperglycaemic crises, but this is much more marked in HHS than DKA (see Table 3). Insulin deficiency or resistance prevents glucose and potassium (and ketones if insulin deficient) entering cells. Consequently, the plasma concentration of glucose increases and water moves by osmosis out of the cells. Glucose is freely filtered by the kidneys and accumulates once reabsorption reaches its threshold (estimated to be 12mmol/L)[4, 7]. An osmotic diuresis ensues and water and electrolytes are lost. To exacerbate matters, the sensation of thirst may make the patient desire drinks with a high concentration of glucose and not infrequently HHS is associated with the ingestion of high energy drinks[4, 7].

Patients with a hyperglycaemic crisis, but in particular HHS may present with hyponatraemia. This is a true finding and all arterial blood gas machines and most modern laboratories now measure sodium by a direct method and are therefore accurate. In DKA pseudohyponatraemia is reported, this may occur if the patient has raised serum triglycerides and a laboratory sample is assayed using one of the indirect methods of sodium analysis. Hyponatraemia in HHS and mixed DKA/HHS, is true. It is caused by the movement of water out of the cells into the extracellular fluids secondary to the severe hyperglycaemia. The patient has a total sodium deficit (osmotic diuresis) and an acute dilution of the extracellular sodium[4, 6–8].

Deficits	DKA	HHS
Total water (L)	7	7–14
Water (ml/kg)	100	100–200
Sodium (mmol/L/kg)	7–10	5–13
Potassium (mmol/L/kg)	3–5	4–6

Table 3: Fluid and electrolyte losses for DKA and HHS in a 70kg man[4, 7].

Immediately insulin is given, glucose moves back into cells and becomes an ineffective osmolyte. Water moves rapidly down the osmotic gradient into cells. The sudden contraction of the extracellular water volume causes an apparent hypernatraemia, although there is actually no immediate change in total body sodium. Many texts suggest using NaCl 0.45% if high sodium concentrations are measured[9, 11]. This is wrong, hypernatraemia should not be actively managed. Total body sodium is decreased and there is evidence that this apparent hypernatraemia is protective against development of cerebral oedema[4]. It maintains the high osmolality as the glucose is corrected and therefore slows the movement of water back into cells[4, 12]. In the case presented, the patient had a normal sodium concentration on admission, which increased on initiation of treatment and for the next 2 days. Once his fluid compartments were fully resuscitated, time had been given to allow equilibrium between intracellular and extracellular compartments and electrolytes had been replaced, it was possible to give alternative intravenous fluids safely. His sodium concentrations decreased to normal over a period of 5 days.

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The hyperglycaemia of HHS or mixed DKA/HHS should be corrected cautiously. It is hypothesised that in severe dehydration, cells generate osmotically charged molecules which aim to slow water loss. If the osmolality of the external environment is changed rapidly, the cells are then vulnerable to swelling. Glucose concentrations should be decreased cautiously. A short-acting insulin should be administered by infusion only (no bolus) and glucose given to prevent the serum glucose decreasing below 18mmol/L until adequate volume resuscitation has been achieved. This may take many hours and patients should be managed in a critical care area[4, 6, 12].

Early rapid fluid resuscitation and bolus of insulin in this patient produced an initial apparent improvement but was followed by a decline in GCS, worsening metabolic acidosis and the need for respiratory support. A number of factors in the patients' lifestyle may have contributed to his depressed conscious level but this pattern is consistent with the cerebral complications of HHS. The patient had a CT scan of his head to exclude other pathologies but nothing else was found and he gradually regained a GCS of 15/15 together with his lucidity.

Potassium should be measured every 2 hours and replaced as necessary. Total body potassium deficit is large from losses in the osmotic diuresis and on initiation of insulin therapy, potassium it is taken up by cells. Regular monitoring of other electrolytes is also required (e.g. magnesium, calcium and phosphate)[4, 6, 7].

The cause of this patients' hyperglycaemic crisis was likely to be a chest infection and early recognition and appropriate antibiotic treatment was important. Sepsis is a common cause of hyperglycaemic crisis, other possible precipitants include insulin omission, cerebral vascular events, myocardial infarction, acute abdomen and various drugs[7, 9, 11, 13].

This patient developed an acute kidney injury on a background of previously normal renal function. This was probably pre-renal due to the intravascular depletion caused by extreme dehydration and then exacerbated by his septic episode[6, 7].

Patients should be managed in an Intensive care environment and the distinction between each condition should be made as early as possible so that the correct management can be initiated. This case illustrates the importance of differentiating between these conditions and that failure to do so can lead to serious complications, such as cerebral oedema and death[1, 4, 5].

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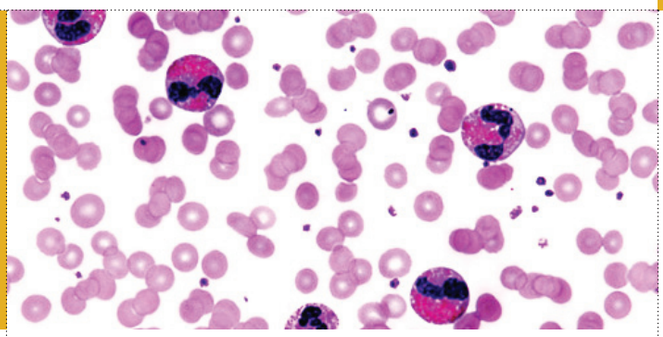
PROCALCITONIN: THE END OF C-REACTIVE PROTEIN?

Lucy Jackson, Parag Gajendragadkar, Carin Swanevelder and Andreas Karas

Initially trialled in the late 1990s, serum procalcitonin (PCT) is a relatively new marker of infection, which is being increasingly used in the critical care setting. It has a shorter half-life and is more specific and sensitive for bacterial infection than the white cell count (WCC) and C-reactive protein (CRP). Good Clinical Care.



Abstract

Initially trialled in the late 1990s, serum procalcitonin (PCT) is a relatively new marker of infection, which is being increasingly used in the critical care setting. It has a shorter half-life and is more specific and sensitive for bacterial infection than the white cell count (WCC) and C-reactive protein (CRP). There has, therefore, been great interest in the use of PCT as a tool to detect sepsis at an earlier stage, exclude patients that do not have bacterial infection, gauge the effectiveness of medical intervention and also as a prognostic marker. This review looks at the basic physiology of PCT and discusses its uses in various clinical settings, highlighting differences with other frequently used markers of inflammation and sepsis. By reviewing the current literature this review aims to discuss the benefits of introducing PCT into routine clinical practice as an adjunct to other markers of sepsis and to guide and limit the use of antibiotic therapy.

Background

A product of the CALC[1] gene on chromosome 11, procalcitonin (PCT) is the 116 amino acid protein precursor to the 32 amino acid protein responsible for calcium regulation, calcitonin. In a normal healthy adult, hormonally active calcitonin is produced in the parafollicular cells (C-cells) of the thyroid gland from PCT in response to high calcium levels in the body. In health, PCT levels in the circulation are kept very low, at levels less than 0.05ng/ml. The in vivo half-life of serum PCT is approximately 24 hours[1].

Quantitative analysis of PCT during bacterial infection has demonstrated its production in a variety of cells throughout the body. These include liver, lung, kidney, muscle cells and adipocytes[2]. Figure 1 demonstrates the variety of cells that produce PCT as a result of bacterial induction of PCT synthesis. The result is a significant increase in the serum concentration of PCT in infected patients as a result of its release from these tissues. Serum levels can reach up to 1000ng/ml in patients with severe bacterial infection/sepsis[3]. The absolute level of PCT concentration has been demonstrated to increase with the increasing severity of disease[4, 5].

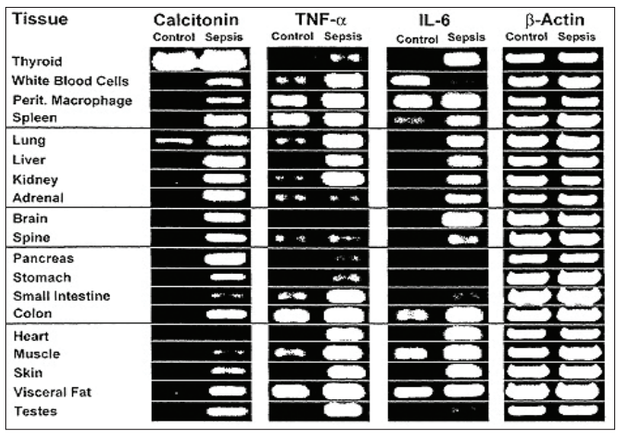


Figure 1: Quantitative Analysis of PCT mRNA Expression by Taq-Man PCR technology shows a significant induction in all tissues during bacterial infection. Parenchymal cells (including liver, lung, kidney, adipocytes and muscle) provide the largest tissue mass and principal source of circulating PCT in sepsis. Compared to classical cytokines, the transcriptional expression of Calcitonin-mRNA seemed more uniformly up-regulated in sepsis[2].

The release of PCT has been shown to be induced both directly and indirectly. Direct stimuli include microbial toxins, while indirect stimuli are via the humoral or cell-mediated host response[3]. However, the precise mechanisms of induction are currently not known. This PCT response occurs within 2–3 hours after induction and has been shown to occur as a rapid rise in concentration that plateaus after 6–12 hours. As infection resolves PCT levels decrease rapidly back to normal values, usually within 48 hours (t1/2 = 20–24 hours) and this response indicates a favourable prognosis[6]. Thus the success or ineffectiveness of specific treatments can be monitored by dynamic changes in the PCT value. PCT values can also be used as an indicator of poor prognosis as progressively increasing levels are directly linked to increasing mortality[6]. The specificity of the PCT response to bacterial infection has been shown to be as a result of attenuation of PCT production by cytokines released in response to viral infection, such as interferon-gamma[7]. The optimal range of cut-off values of PCT are variable and dependent on a number a factors including the aetiology, site and extent of the infection (lower respiratory tract infection (LRTI) or sepsis) and patient co-morbidities (e.g. immunosupression)[8]. Tables 1 and 2 demonstrate the different cut-off levels used for PCT in sepsis and LRTIs.

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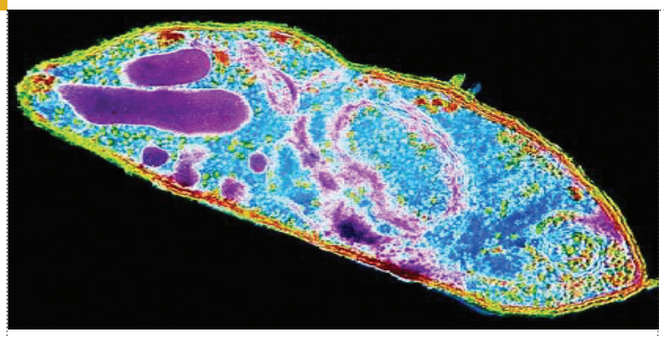

PCT value (ng/ml)	Interpretation in Sepsis
<0.05	Within normal range.
<0.5	Systemic infection (sepsis) is not likely. There is low risk for progression to severe systemic infection (severe sepsis). However, local bacterial infection is possible.
0.5–1.99	Systemic infection (sepsis) is possible and there is a moderate risk for progression to severe systemic infection.
2–9.99	Systemic infection (sepsis) is likely, unless other causes are known. There is high risk for progression to severe systemic infection (severe sepsis).
>10	Significant systemic inflammatory response, almost exclusively due to severe bacterial sepsis or septic shock.

Table 1: Procalcitonin (PCT) values in sepsis and their interpretation[4, 5, 9].

PCT value (ng/ml)	Interpretation in LRTIs
<0.05	Within normal range.
<0.1	Indicative of the absence of bacterial infection and therefore the use of antibiotics is strongly discouraged.
0.1 - 0.24	Bacterial infection is unlikely and the use of antibiotics is discouraged.
0.25 - 0.49	Bacterial infection is possible and it is advised to initiate antibiotic therapy.
>0.5	Suggestive of the presence of bacterial infection, this antibiotic treatment is strongly recommended.

Table 2: Procalcitonin (PCT) values in Lower Respiratory Tract Infections (LRTIs) and their interpretation[16].

Increased PCT levels have also been found in patients with invasive fungal infections and acute attacks of *Plasmodium falciparum* malaria and therefore may also be useful in this setting[8, 10]. PCT levels can be increased by non-infective causes, for example, in the first few days after a major insult such as trauma, major surgery, severe burns, severe cardiogenic shock or prolonged severe organ perfusion anomalies[8]. Neonates have also been shown to have a physiological elevation of PCT levels in the first 48 hours of life[9]. Finally, patients with small cell lung cancer or medullary C-cell carcinoma of the thyroid have also demonstrated elevated PCT levels[11]. Low PCT levels may be present in bacterial infection, such as in the early course of infection (less than 6 hours)[8] and thus levels should be repeated within 6 to 24 hours to exclude a false negative result. Falsely low PCT levels in the presence of infection have also been shown in localised infection[12] and subacute infective endocarditis[13].

Procalcitonin, sepsis and the critical care setting

Early detection of sepsis and ultimately the subsequent clinical interventions used at this stage are crucial for the improved outcome of patients with sepsis. However, sepsis can be difficult to distinguish from the systemic inflammatory response (SIRS), a response to stress which is found in a number of critically ill patients. Although SIRS is often due to infection, PCT is a potentially valuable tool for early and specific detection of bacterial infection/sepsis and differentiation from other causes of SIRS, such as trauma, burns, pancreatitis, ischaemia or haemorrhage. This is especially relevant when compared to markers of infection currently used in the clinical setting, such as C-reactive protein (CRP), white cell count (WCC) and interleukins 6 and 8 (IL-6 and IL-8)[6]. The result is the ability to decide on the appropriate therapeutic measures for each patient on a more informed case-by-case basis.

In septic patients PCT reliably and rapidly returns to values below 0.5ng/mL as their infection resolves[6]. Therefore, the PCT serum levels can be utilised to monitor the progression of life-threatening systemic bacterial infections and ultimately tailor therapeutic interventions to individual patients. This has been demonstrated for the monitoring of patients with ventilator-associated pneumonia (VAP)[14]. Consequently this results in reduced antibiotic exposures in length of treatment, as there is a definitive marker at which to stop antibiotics (i.e. a set PCT value). This also decreases unnecessary changes in antibiotic regimen, as physicians have a marker of change within 24 hours as opposed to the 48 hours currently provided by CRP thus reducing the pressure to change antibiotics when after 24 hours of treatment there is no change in the patients clinical condition despite underlying biological improvement[15]. In addition, a reduced length of stay in critical care has been shown[15]. Furthermore, changes in serum PCT levels have been demonstrated to allow prediction of outcomes in this setting. Jensen et al. demonstrated that increasing levels of serum PCT on consecutive days in critical care were directly associated with increasing mortality[6]. Ultimately this means significant reductions in clinical costs and improved patient management.

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Procalcitonin and LRTI

Research has shown that due to the high specificity of PCT for bacterial infection, PCT measurement at low concentrations can help to differentiate patients with clinically relevant LRTI who require antibiotic therapy from those who do not require antibiotic treatment[16]. Christ-Crain et al. used the PCT algorithm depicted in Table 2 as a guide to antibiotic treatment in patients with LRTI admitted through the emergency department in a large teaching hospital in Switzerland. In this randomised intervention trial, they showed that by using a PCT-based decision algorithm, antibiotic prescription potentially could be reduced to 50% of cases with LRTI. Importantly, when compared to the control group of patients who were treated according to usual empirical medical practices there was no difference in clinical outcome between the two groups. This algorithm assisted the identification of patients with infection of viral aetiology or self-limiting disease where antibiotics would not be clinically useful[16].

As in the critical care setting, serial PCT measurements in LRTI patients have demonstrated that the length of antibiotic therapy can be tailored to the individual need of the patient. Where previously standard practice was to empirically treat a patient with community-acquired pneumonia (CAP) with 10–14 days of antibiotic, by using the serum PCT level to guide withdrawal of antibiotic treatment the median length of treatment was reduced from 12 to 5 days[17]. Again there was no change to the ultimate outcomes of these patients when compared to those treated under the empirical system. It should be noted that current British Thoracic Society (BTS) guidelines suggest standard antibiotic therapy for non-complicated CAP is 7 days[18], however, this is still an additional 2 days of antibiotic treatment than when compared to patients whose PCT levels were monitored in the community[17]. These results have led to the initiation of a large prospective multi-centre randomised trial, the goal of which is to compare clinical outcomes, use of antibiotics and hospital resources in a strategy based on enforced evidence-based guidelines versus PCT guided antibiotic therapy in inpatients with LRTIs[19]. It will be interesting to see if future studies are performed that assess the practicalities and costs of measuring serum PCT values of CAP patients in the community and if this a realistic option. The above studies demonstrate that the integration of PCT into diagnostic and treatment algorithms allows both targeted treatment and treatment duration tailored to the individual clinical situation of each patient. This has been shown to reduce total antibiotic usage which ultimately results in reduced expenditure and exposure of unwell patients to antibiotics. This reduction in antibiotic usage could in turn reduce antibiotic-related illnesses, such as Clostridium difficile infection and antibiotic resistance.

Procalcitonin and surgery

Given the use of PCT in differentiating bacterial infections from systemic inflammation, its use has been investigated in patients who have undergone surgery. There has been some debate as to the use of PCT in post-surgical patients as small rises post-operatively are well documented. However, after 24 hours these modest increases are followed by a rapid reduction to baseline levels. As the absolute PCT levels are less important than change in levels there is no unique "cut-off" point to differentiate post-surgical rises in PCT from ongoing infection. Nevertheless, based on a number of observational studies, literature provided by the company owning PCT suggests that retention of serum levels >2ng/ml or a value that does not fall appropriately implies ongoing bacterial infection. In a prospective study looking at the ability of PCT to diagnose post-cardiac surgery wound infection the data suggested that any value of PCT above 1.5ng/ml 48-hours post-surgery diagnosed infection with a sensitivity of 93% and a specificity of 80%[20]. The authors found no differences in post-operative changes in C-reactive protein (CRP) and white cell count between those who developed infection and those who did not, highlighting the superiority of PCT in this setting.

In patients with acute severe pancreatitis, infection of the pancreatic necrotic tissue can be common and is associated with a significant increase in morbidity and mortality. While those with sterile necrosis may be managed conservatively, proven pancreatic infections with signs of sepsis may need interventional or surgical therapy. A prospective multi-centre trial found that a PCT level ≥3.5ng/ml on 2 consecutive days was superior to CRP for the assessment of infection associated with tissue necrosis and as a predictor of mortality[21].

A meta-analysis of patients in the intensive care unit who had undergone surgery or trauma found that PCT was superior to CRP as a diagnostic marker for sepsis. The authors concluded that PCT should be included in diagnostic guidelines for sepsis and in clinical practice in intensive care units[22].

In summary, while PCT levels do go up to some extent following systemic inflammation, trauma or surgery, a persistent elevation over a few days above 2–3ng/ml has a high sensitivity and specificity for detecting serious bacterial infection.

Summary

PCT is a sensitive and specific marker for bacterial infections and sepsis with a short half-life making it an excellent marker of infection when compared to currently used biomarkers. Figure 2 demonstrates typical patterns of PCT and CRP responses in patients with bacterial infection (data from study by authors). Development of simple laboratory assays means that this marker is being used routinely in some critical care centres with increasing frequency. There is a large amount of clinical data to show its usefulness but clinicians must be aware of its specific limitations (e.g. localised infection) and not use PCT as a substitute for clinical judgement but rather as an adjunct, albeit a powerful one. In order for PCT to become more widely adopted further research concentrating on cost-effectiveness needs to be carried out. This is likely to have the most benefit in the critical care setting, however, its use in patients with LRTI on the general ward may result in a reduced length of antibiotic treatment and with that a reduction in side effects in particular Clostridium Difficile infection.

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PROCALCITONIN: THE END OF C-REACTIVE PROTEIN?

Lucy Jackson, Parag Gajendragadkar, Carin Swanevelder and Andreas Karas

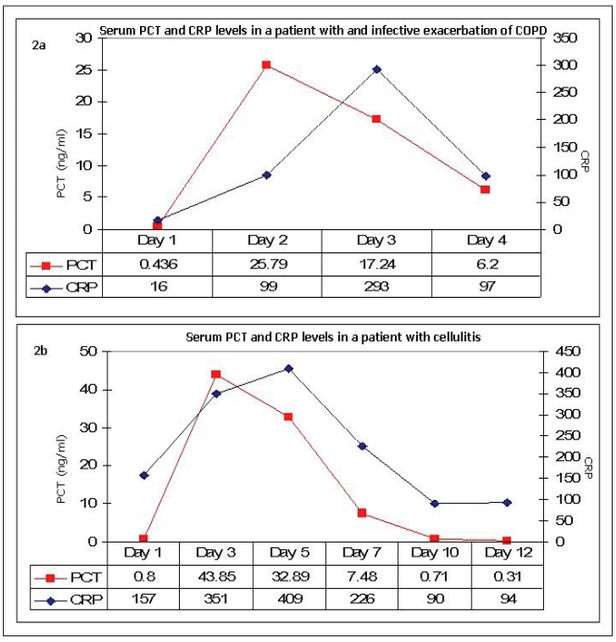

Figure 2: Figures 2a and 2b depict the serum PCT and CRP values in two different patients. Figure 2a was a patient with an infective exacerbation of chronic obstructive pulmonary disease (COPD). The graph shows the faster and more definitive response of PCT to the presence of infection and then the treatment with antibiotics, which started on day one. Figure 2b was a patient with cellulitis and again shows the quicker response of PCT when compared to CRP once antibiotics were started on day two.

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ETHICAL DILEMMAS IN ACUTE CARE - WHAT IS MEDICAL ETHICS?

Karl Brennan and Daniele Bryden

Medical ethics is about matters of right and wrong in medicine: "It is specifically concerned with moral principles and decisions in the context of medical practice, policy and resources."[1]
Good Clinical Care.

Medical ethics is about matters of right and wrong in medicine: "It is specifically concerned with moral principles and decisions in the context of medical practice, policy and resources."[1] Medical ethics has its roots in theology and philosophy, and key principles have developed helping to guide our decision making. These are respect for autonomy, non-maleficence, beneficence and justice.

General Ethical principles

1. Respect for autonomy refers to the right of an individual to make their own decisions and requires the individual to have both the capacity to make an informed decision, and that it is also made free from any external influences. This is the basis of valid patient consent.

2. Non-maleficence refers to the principle of "first, do no harm". It is our responsibility as clinicians not to inflict harm, but in practice, this can cause conflict with our desire to do good, such as when we know a treatment we offer may have unpleasant side effects (e.g. chemotherapy for cancer). It is part of the basis for decisions regarding withholding and withdrawing treatment.

3. Beneficence refers to a desire to do good. It is our obligation to contribute positively to the welfare of an individual and includes attempts to prevent harm from occurring, removing harm where it exists and promoting good. This is the ethical basis for clinical governance and a supporting idea behind the best interests test.

4. Distributive justice, refers to the need to treat all individuals fairly, with equal access to resources. In a publicly funded health service like the NHS where resources may be limited, it can sometimes come into conflict with autonomy and the desires of the individual. It is most often seen in acute care areas where there are limited numbers of beds and decisions regarding care are made on the basis of greatest need and likelihood of benefit.

This article will examine how these principles operate within acute care environments.

Ethics, law and the GMC

Legal requirements also influence a doctor's actions. The law considers rules of conduct/behaviour established by an authority and enforced through the judiciary. Ideally, it produces regulations that can be easily followed or broken. In contrast, ethics is based on principles which help us make the right moral decisions, but is not as prescriptive as the law. The GMC identifies that it is every doctor's duty to uphold both the law and medical ethical principles and in acute care practice this can present many challenges[1].

Scenario one

A 21-year-old student is brought into hospital by her friends having collapsed in the toilets at a club. She has drunk 3 bottles of beer and is refusing to have any blood samples taken for paracetamol and salicylate levels.

Does she have the capacity to make a decision?

She should be presumed to have capacity until demonstrated otherwise (i.e. having reportedly drunk the beer does not automatically mean she is inebriated and lacks capacity).

Furthermore, you must not assume that if the patient lacks the capacity to make a decision in relation to one matter, she lacks the capacity to make any decisions at all. She must be able to comprehend the information about the need for testing; must be able to retain that information long enough to make a decision; must be able to use and weigh it prior to making a choice; and must be in a position to communicate the decision. If necessary she must be helped to communicate her decision.

If there is doubt regarding a patient's capacity, advice should be sought from other health professionals including nursing staff, principal carers, etc. If doubt remains, legal advice must be sought with a view to a court deciding on capacity, but this is very rarely required in such a scenario.

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The Mental Capacity Act 2005 does not define the term but says treatment decisions "must not be based on a patient's age, appearance or unjustified assumptions based on the person's condition or behaviour"[3].
Good Clinical Care.

Does she have to consent?

If a person has capacity, consent must be obtained before any intervention. Failure to do so is unlawful and risks a charge of assault. In order for consent to be valid, the patient must have full disclosure of information; have the capacity to make a decision; and be given the freedom to do so[2]. Full disclosure of information includes: sharing the diagnosis or likely diagnosis; the prognosis; and all options available, explaining the potential benefits and risks of treatment and their likely success. Patients should be given this information in the environment in which they are most likely to understand and retain it. In this case it would be appropriate to talk to the patient privately without her friends and explain why there is a need to do these tests, and the consequences to her of not receiving treatment for potential paracetamol or salicylate overdose. Even if the refusal to have blood tests performed seems irrational, provided the patient has capacity, her decision must be respected.

In acute situations, however, the law allows for treatment without consent where a patient is incapacitated, provided it is immediately necessary to save a patient's life or to prevent a serious deterioration in their condition[3]. When the patient recovers capacity you should tell them what has occurred. In this case, if she was brought in unconscious, it would be appropriate to do the blood tests at that point, but when she recovered consciousness explain what had happened.

Scenario 2

A 55-year-old man with multiple sclerosis (MS) who is wheelchair bound is referred to the intensive care unit with pneumonia. He is confused and has oxygen saturations of 88% on 40% oxygen. On what basis can decisions be made regarding his treatment?

Critical care involves the provision of complex, often invasive and potentially unpleasant treatments for patients who commonly lack capacity to consent. This is usually due to reduced consciousness or mental function being compromised by severe illness or injury. Therefore, treatments are often initiated based on a clinician determining what they think to be in the patient's best interest.

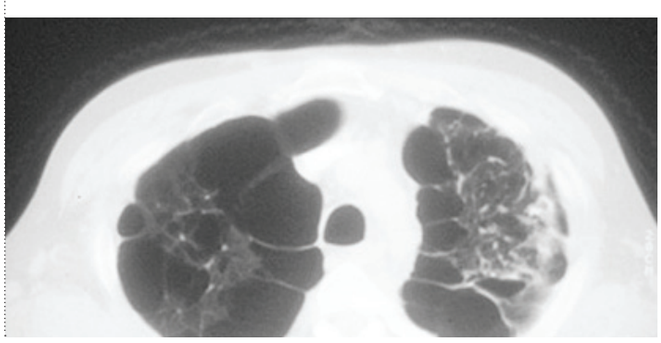
What is "best interest"?

The Mental Capacity Act 2005 does not define the term but says treatment decisions "must not be based on a patient's age, appearance or unjustified assumptions based on the person's condition or behaviour"[3]. In this example, it would be unacceptable to assume that as this man has a physical disability, he does not have a good quality of life and therefore treatment should not be given. An objective assessment would consider all the relevant factors and circumstances equally (e.g. obtaining information from his family, close friends or carers) as to his usual wishes regarding medical treatment, the type of life he is able to lead and whether this is to his satisfaction, to help reach a balanced decision.

The decision must also consider whether this man's lack of capacity is temporary (e.g. due to sepsis or permanent: related to his MS). If it is temporary, the clinician must make a judgement as to whether a decision regarding treatment could wait until the patient could decide on his treatment wishes, or to institute treatment based on his best interests. It is important that where more than one treatment option exists, consideration is given to the option least restrictive of the patients future options. In acute care, the severity of illness is often so great that recovery to a point where capacity is regained can be a long time, and treatments are often needed so acutely that waiting for capacity to be regained is rarely an option. It is therefore an appropriate professional practice for the health care team to consider all these factors in deciding on the extent of this man's treatment.

Scenario 3

A 63-year-old lady with severe chronic obstructive pulmonary disease (COPD) is admitted to hospital with a further exacerbation of COPD. Her daughter says she has an advance directive in her notes stipulating that if she has further exacerbations of COPD she would want care to continue up to but not including invasive ventilation for her COPD.



ETHICAL DILEMMAS IN ACUTE CARE - WHAT IS MEDICAL ETHICS?

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What is an Advance Directive?

Where any previously written statements exist they should be considered regarding decisions about further care but are not absolutely binding. If an Advance Directive exists, which is signed, witnessed and relates exactly to the circumstances under consideration as in this case, management decisions should be based on the stated wishes of the individual. The health care team should continue to treat this lady actively including giving her non-invasive ventilation for her COPD, if needed, but would not refer her to ICU for invasive ventilation if they reasonably believe the Advance Directive applies to this illness. An Advance Directive can, however, be revoked at any time by the patient (e.g. this lady could verbally revoke this decision on her admission if she has capacity) in which case the team should refer her to ICU, if necessary. Also if the Advance Directive is unavailable or doubt exists about its validity, there is a presumption in favour of providing clinically appropriate treatment.

Scenario 4

A 56-year-old man has developed multi-organ failure on ICU from severe pancreatitis. Despite receiving maximal organ support his clinical condition has continued to deteriorate and the surgeons feel there is nothing more they can do to treat the pancreatitis. The ICU team feel that after consultation with the family, it would be appropriate to stop treatment.

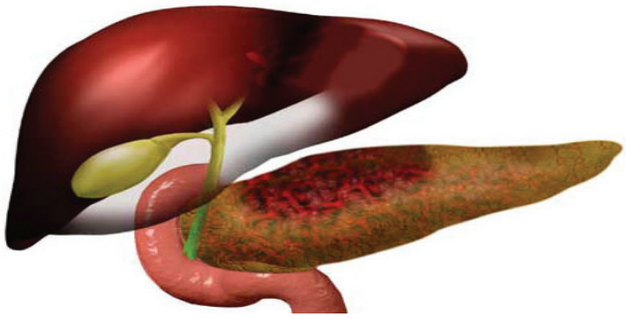
Having started ICU life support can it be stopped?

The decision of when to withhold or withdraw treatment is arguably the most difficult decision any doctor has to face. The majority of intensive care patients are not in a position to state their wishes and it must be remembered that intensive care treatment is rarely curative in its own right but rather provides supportive care while other treatments and time facilitate recovery.

Generally, doctors initiate and continue treatment until a decision is made that further treatment is no longer required or futile. Futility must be determined on an individual basis (e.g. if a patient were to survive, would the quality of life be so poor as to render further treatment unacceptable?). For example, a patient who is already bed bound due to frailty or disability due to chronic disease that is not improvable, is extremely unlikely to survive a prolonged intensive care stay. Therefore, even if the presenting complaint is deemed reversible (e.g. pneumonia), invasive treatment like artificial ventilation is usually deemed futile as it would not return the patient back to their previous quality of life. This would be a decision to withhold treatment. In practice, it is again essential to try and elicit any knowledge as to the patient's likely wishes.

This will usually require further discussion with family members and close friends but very occasionally a patient may have explicit written guidance regarding treatments they would find tolerable and those they would not.

So how do we decide at what point treatment should be discontinued when faced with a patient who is failing to respond? In this case, all therapeutic options have been exhausted and the patient is deteriorating despite maximal therapy. It is arguable that it is not in the patient's best interests to have death prolonged through artificial means, exposing him to additional discomfort, pain, loss of dignity and loss of autonomy.



What then is the best way to proceed?

Ethically there is no distinction between withholding and withdrawing treatments in end of life care. However, doctors have an ethical duty to uphold life wherever possible and a legal duty not to do any act with the intention of hastening death[4]. The law does not consider withdrawal of treatment as positive acts to hasten death, and so providing the health care team and the relatives were in agreement that this, was the best course of action. Intensive care interventions could be withdrawn on the grounds of futility (e.g. stopping inotropes). In those circumstances, the natural underlying course of the disease would mean that it is very likely that the patient would die of pancreatitis.

Conclusion

Ethical dilemmas in an acute care setting are numerous and complex. There are many factors involved in the difficult decision making that often occurs. Patients may often lack capacity or require treatment based on judgements of their best interests as they are unable to take part in a process of consent. Decisions regarding the withholding and withdrawal of treatment are frequently made by the multidisciplinary team and the Foundation doctors can have a valuable contribution in terms of prior knowledge of the patient, family and any expressed wishes.

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AN SHOS EXPERIENCE OF WORKING IN THE A&E

Faraz Imran and Asgharali Wain

Accident and Emergency (A&E) is an important and integral part of every hospital in the NHS (national health service). It is the central hub of the hospital through which a significant proportion of patients enter a hospital as inpatients.
Good Clinical Care.

Introduction

Accident and Emergency (A&E) is an important and integral part of every hospital in the NHS (national health service). It is the central hub of the hospital through which a significant proportion of patients enter a hospital as inpatients. A thriving, well-resourced, forward looking and innovative A&E is the key to the success of a hospital.

Generally a 4-month rotatory posting in the A&E is achieved in the 2nd year of the Foundation programme (senior house officer level).

Working in the A&E for a junior doctor is a bit scary in the beginning, however, it remains an integral part of training and an invaluable experience for the junior doctor whatever specialty they choose to pursue in the future.

Organisation

Medical Staff
- consultants
- associate specialists
- specialist registrars
- clinical fellow/staff grade
- senior house officers

Nursing Staff
- matron/in charge
- staff nurse
- emergency nurse practitioners
- emergency care technician

What to Expect

1. Clerk the patients after they are triaged with initial assessment
2. Decision making in patient diagnosis and management
3. Make referral to the appropriate specialty
4. Participate in teaching, audit and learning activities

Decision making is the key to A&E practice. As a new doctor I was independently making decisions about the patient I saw. Initially this felt a bit daunting, but my seniors were very supportive and helpful especially in the early days and I settled in quickly. I never felt embarrassed asking nurses or even radiographers for help as some of them were very senior At times, however, it is practically very difficult to consult a senior for every patient especially when the A&E is very busy, which is not unusual. It is therefore advisable to seek help with complex cases earlier before the pressure to take a decision builds up in order to comply with the 4-hour breech rule. Also decisions taken in haste could be detrimental. I learned that doctor should use their clinical knowledge and experience in the best way to treat the patient and rule out any serious pathology. They should also positively and meticulously document all their findings on the patient notes before taking the final decision. This is a learning process that will gradually make the trainee more confident. I have discovered that when the A&E is very busy (and so are the seniors) calling up the respective specialty and asking the on-call registrar for advice (rather than a referral) can be very helpful in making decisions about safe discharge of a patient you are concerned about. If not, then this turns out to be a referral which the registrar would be obliged to accept if he or she is concerned.

Work Rota

One of the drawbacks of an A&E rotation that doctors fear is its tight and unsocial working schedule that may include different combinations of early, day, midday, evening/late and whole night shifts from 8–10 hours. Although the total working hours per week would not exceed the European working time Directives. The junior doctors would have to develop the flexibility and work their social lives around this schedule to fulfil the demands and obligations of the rota. Again it is a matter of getting used to the system. Once you get used to the sync it would be quite enjoyable with lots of opportunities and time off especially on weekdays (something colleagues in other specialty's miss) that may be very helpful to do courses and official works.

AN SHOS EXPERIENCE OF WORKING IN THE A&E

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Triage

Every patient in A&E is triaged before admission and based on the decision transferred to minors, majors or the resuscitation area. Patients may be triaged and brought in to the respective department by emergency ambulances as well.

Common presentations:

- minors
- fracture and dislocation
- lacerations and abrasions
- soft tissue injuries/sprains
- foreign body/abrasion in the eye
- epistaxis.

The minors department is generally quite a busy part of the A&E. Unlike its name it often requires a more senior and experienced doctor as it may require interpreting fractures (that can be quite tricky) and manual fracture reduction and plastering. A devoted ENP (emergency nurse practitioner) would ideally be there to carry out the plans and help the doctor and even clerk patients independently. I found it a good experience to spend some time daily in the minors area with the senior doctor to learn how to order and interpret X-rays and manage patients with various fractures and dislocations.

Majors:

- chest pain
- difficulty in breathing
- abdominal pain
- fractures
- cerebrovascular event/meningitis
- burns
- sepsis
- bleeds (gastrointestinal/per vaginal).

Unlike the frightening perception I had about majors, I found it quite a familiar exercise to clerk patients in the majors area. They basically involved the same kind of emergency admissions whenever in doubt that were witnessed by me while I was a house officer in the acute medical and surgical specialties. However, the main concern is not to miss an atypical presentation of a common disease which definitely requires the expertise of an experienced senior. Hence, one should try to get a senior review or a second opinion as much as possible. For example, an atypical abdomen pain should alert you to rule out the possibilities of pancreatitis (serum amylase), mesenteric ischemia (serum lactate) or an aortic dissection (ultrasound); likewise other rescue investigations being a troponin for a query silent MI (myocardial infarction) in an elderly/diabetic patient and a beta HCG (human chorionic gonadotropin) in a female in the reproductive age group with blood per vaginum. If the investigations are normal but the patient is still clinically unwell then the patient should be reassessed by a senior colleague, observed for some time (especially pediatric patients) either in the A&E or under the respective specialty after referral (this might have to be on a senior level on clinical concerns with a view to repeat investigations). The bottom line is do not discharge a patient if you are concerned.

Resuscitation:

- Major Trauma
- Cardio-Respiratory Arrest
- Shock

The resuscitation department is often the most feared area for the junior doctors in the beginning and I was no exception to this. But I found out that this can be overcome by visiting any blue light calls if you are not clerking any patient and seeing how the other doctors manage and resuscitate the patient. In fact keeping in mind that the fundamental aim of the A&E doctor being to refer the patient to the appropriate specialty as soon as possible after identifying and stabilising the patient (airway, breathing, circulation, disability, exposure) this is a very straightforward exercise once you overcome your nerves! Doing an ALS/ATLS course may be very helpful in this regard.

The specific Skills required or desirable for the SHO to deal with the above variety of cases are knowledge and competencies in the following:

Required skills:

- basic and advanced life support
- fracture diagnosis with X-ray interpretation
- wound management including local anaesthesia and suturing
- catheterisation
- placing collar and cuff support
- ECG interpretation and management of ACS (acute coronary syndrome)
- use of otoscope, opthalmoscope
- removing foreign body from the eye
- anterior nasal packing
- early identification and assessment of suspected stroke patients (may need urgent CT/thrombolysis).

Desirable skills:

- advanced trauma life support
- cricothyroidotomy
- ring block, haematoma block
- venous cut down/femoral or central line
- needle thoracocentesis/chest drain insertion
- reduction of common fractures
- needle aspiration from a joint
- use of FAST (focused assessment sonography in trauma)
- use of a slit lamp
- thrombolysis.

AN SHOS EXPERIENCE OF WORKING IN THE A&E

Faraz Imran and Asgharali Wain

The change in the policies on junior doctor working time and a shorter 4-month rotation instead of 6 has made it more difficult to inculcate the above skills and expertise during the A&E rotation at the Foundation level. Good Clinical Care.

The change in the policies of junior doctors' working time and a shorter 4-month rotation instead of 6 has made it more difficult to inculcate the above skills and expertise during the A&E rotation at the Foundation level. Nevertheless, instead of scaring the novice away, the above list I believe should be used as a yardstick to assess individual level of skill and competencies. One may overcome the time constraints through focused training and practice with seniors in hospital settings and pre-planning and attending to various courses (advanced life support, advanced trauma life support, accident and emergency course/invasive medical procedure courses). Having a good reference book along, like the *Oxford Handbook of Emergency Medicine* would be much advisable.

Referral

Apart from management of acute cases, referral is the prime function of the A&E department. Referral are all about communication. The doctor should pay special attention to this and learn to communicate effectively, courteously and to the point. I have felt that in the hospital culture no one likes to accept a patient unless you can convince and satisfy them. I believe that referral is not only about being able to "sell" your case (a term used in hospital medicine) it's about your concern about the correct management of your patient and which department it could be most effectively realised in. If you are not able to get your patient accepted by a particular specialty and you are genuinely worried ask your registrar or consultant to review the patient and have them refer the patient at a senior level. Remember the patient's safety comes first. Again, I would emphasize document everything clearly in your notes. A point of note that I found particularly helpful in this regard is being aware of the various locations, emergency contact numbers and bleeps of the specialties that accept referral from your trust as per the trust policy.

Note keeping

Medico-legally, the A&E record is the prime source of evidence in medical negligence cases. And the A&E doctor is often the most labile in terms of medico-legal litigations. A well documented and nicely presented record (like it or not) reflects the attitude of the doctor towards his patients whether sincere or casual. This requires devoting some time to your notes which of course needs to be balanced between clerking the patient thoroughly and respecting the 4-hour breech rule as well. If time is not unnecessarily wasted this balance is usually not difficult to accomplish.

Casualty Card Layout:

- Presenting complaint – indicate source of history.
- History of presenting complaint – indicate the mechanism of injury, the speed of the vehicle in trauma patients or acuteness of symptoms.
- Previous relevant history – including recent A&E admission, family and social history.
- Current medications and allergies – including non-prescribed drugs.
- Examination findings – document negative findings as well, such as absence of neck stiffness in patients with headache. Document the side of injury and the dominant limb (whether left- or right-sided). Always mention the neurovascular status of the injured limb.
- Investigations – record clearly. Mention key investigations, such as amylase to rule out pancreatitis; or troponin to rule out ACS.
- Working diagnosis – a differential diagnosis list.
- Treatment given – note the drug, dose, route of administration. Note the number and type of sutures/staples used for wound closure. Advice sheet and follow-up instructions should be clearly mentioned. Indicate when patient needs to be reviewed by the GP and send discharge letters if appropriate.

Basic Rules of Documentation:

- Always write legibly in black ballpoint pen preferably as it is better for photocopies.
- Always date and time the notes.
- Sign your notes and print your name and status below.
- Use either pre-printed sheets or simple line drawing for wound descriptions.
- Avoid abbreviations.
- Never make judgemental comments.
- Keep the GP informed with a written letter.

AN SHOS EXPERIENCE OF WORKING IN THE A&E

Faraz Imran and Asgharali Wain

Interpersonal relationship with other colleagues and staff

The A&E perhaps enjoys a very unique place in the hospital where a closely knit social atmosphere between the members of the MDT (multidisciplinary team) exists, mostly due to the close nature of the working environment between doctors and nurses. One should respect this and be wary that it is as easy to get infamous as is to get along well with others. An unfriendly environment may make working very difficult, harming both the patient and the doctor. The thing to bear in mind is to keep to the principles of good communication and avoid undue confrontations. The nursing staff play a pivotal role in the A&E. They have been working for many years and are quite experienced and if they disagree with your plan or views one should try to listen to their concerns and explore a common solution or if that is not possible consult a senior A&E staff who would know how to deal with the situation.

Some common pitfalls and advice for junior doctors

Be on time and leave on time. Try not to be late for your shifts no matter how awkward it may be, and try to pack up and hand over the patient clerking and other details thoroughly and clearly once you have finished off a shift without having to over stay too long, as it may interfere with your next shift. Take a break. Nurses are usually more organised in this than doctors. Your body needs hydration and your bladder needs to be emptied – don't forget them as it will reduce your efficiency and make you more prone to errors.

The A&E was renamed from its previous name "casualty" to highlight the nature of this department and to discourage casual attendees. The young doctor should realise that the A&E is a busy department and requires early decision making and appropriate referral to the respective department if it is to abide by the national 4-hour target which is again in the interest of the patient eventually. The junior doctors usually have a habit of detailed history taking and examination of all the systems meticulously from their previous attachments in medical and surgical specialties. Whereas doing a proper evaluation is never discouraged. The point I wish to highlight here is "focused history taking and relevant examination and management of the patient" to stabilise acute conditions leaving the non-acute or chronic management for the general practitioner (either through a discharge letter or advise to the patient to see their GP) or the respective specialty referred to.

It is discouraged to "swap patients" or pick up another casualty card. In fact one should be prepared for everything that comes through the door! Although it sounds a bit scary at first, developing and sticking to a structured management protocol, such as the airway, breathing, circulation, disability and exposure (ABCDE) and taking senior help as and when necessary should make one more confident and experienced gradually.

Failing to check baseline visual acuity in both the eyes could have medico-legal implications if the patient claims to have lost his visual acuity post any intervention in the A&E. Steroids should not be prescribed in the A&E for eye conditions rather the patient should be referred to the ophthalmologists.

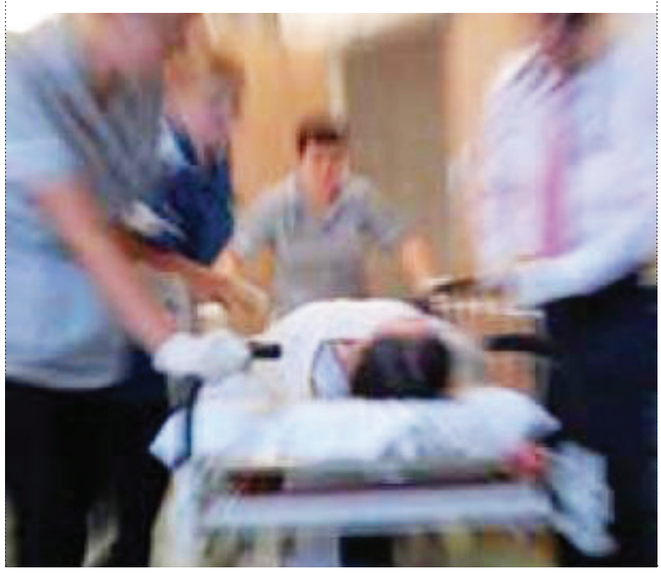
A chaperone should be used in all invasive examinations. The doctor should not forget to take consent from the patient in all possible cases unless they are unconscious. Prescription errors especially involving lifesaving drugs could be catastrophic. The junior doctors should make it a habit to consult the BNF and/or take senior opinion for the same.

Stick to the ALS/ATLS guidelines (ABCD approach), the NICE guidelines and the local hospital/trust protocol while managing patients. And ask for help early in complex cases to prevent the 4-hour breech. No decision of discharge should be taken by the junior doctor without a senior review if there is any doubt about the underlying pathology, especially in cases concerned with abdominal pain, chest pain, shortness of breath, paediatric patients, and so on. Bone injury including subtle growth plate fractures in children should always be reviewed by more senior staff.

Keep a note of any interesting case, and try to follow them up. This will make up for the lack of a fuller picture of the pathology and treatment that the A&E doctors usually miss out on due to referral to other specialties. Read up as you see cases, between seeing patients, this is a very good way of learning in the A&E. Keep the Oxford Handbook of Emergency Medicine in your reach.

For abusive and uncooperative patients, who are rare, you have a right to report it to the Matron or the Sister in charge and they can be dealt with by security or even refused treatment. Similarly patients with psychiatric emergencies (suicidal) may be sectioned under the appropriate clause until seen by the doctor if they are uncooperative. You might at times be requested to write a police report for a patient you had clerked in earlier. Take help from seniors and/or photocopies of previous police reports filled up by other doctors that could be obtained from the secretaries.

There may be some "regular attendees" who develop an attachment to the A&E, your seniors and the nurses would know how to deal with them so follow their advice but beware of missing out on important and obvious pathology that requires treatment.



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AN SHOS EXPERIENCE OF WORKING IN THE A&E

Faraz Imran and Asgharali Wain

Conclusion

Working in the A&E has its pros and cons. Nevertheless, it is one of the most important attachments for a junior doctor and most sought-after job experience in the CV (curriculum vitae) when applying for jobs in any specialty. Surviving A&E is not as difficult as it looks from the outside. It requires adhering to the principles of basic and advanced life support, good communication skills and effective time management. For most junior doctors it is the most enjoyable rotation ever, mostly due to the close social and emotional bond developed between the multidisciplinary team due to the close nature and the team work involved. I would conclude that a little bit of careful planning before starting your A&E job and focused learning could make you feel quite confident in assessing and managing acutely unwell patients at the end of your attachment.

Key Words

- accident and emergency (A&E)
- 4-hour breech
- minors, majors and resuscitation departments
- medico-legal issues and documentation
- skills and competencies.

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PRACTICAL PROCEDURES INVOLVED IN MANAGING EPISTAXIS

Mark Pais, Yim Lun Wong, William Cheung and Mudit Jindal

A 65-year-old female presented to the A&E department with epistaxis from her left nostril lasting for 45 minutes. She had tried to control the bleeding by pinching the bridge of her nose but to no avail.
Practical Procedures.

This material has never been published and is not currently under evaluation in any other peer-reviewed publication.

History

A 65-year-old female presented to the A&E department with epistaxis from her left nostril lasting for 45 minutes. She had tried to control the bleeding by pinching the bridge of her nose but to no avail. On examination she was haemodynamically stable but was very anxious. The attending doctor immediately inserted an anterior pack to control the bleeding. The bleeding resolved and the Ear Nose and Throat (ENT) team were informed. After 30 minutes, the patient noticed blood trickling down the back of her throat and bleeding from the contra-lateral nostril. A Foley catheter was inserted by the ENT registrar into the left nostril which controlled the bleeding. The patient was transferred to ENT ward.

Epistaxis is by far the most common potentially life-threatening ENT emergency with an incidence of around 108:100,000 population per year[1]. This condition is commonly seen in A&E and junior doctors are often expected to manage this condition in the initial stages[2].

Relevant anatomy

Epistaxis is often categorised into anterior and posterior bleeds. This is of clinical relevance as anterior epistaxis exhibits unilateral, steady bleeding, while posterior epistaxis can be more difficult to control and can be bilateral. Bleeding of 90% is anterior and originates from Kiesselbach's plexus - an anastamosis of the sphenopalatine artery (from maxillary), greater palatine (maxillary), anterior/posterior ethmoidal (ophthalmic) and superior labial (facial) arteries. This region is known as Little's area. Posterior bleeds are less common and are due to leakage of the sphenopalatine artery or one of its branches located in the nasopharynx.

With all unstable patients initial management begins by assessing airway, breathing and circulation.

1. First aid

This encompasses the treatment patients use to stop the bleed prior to arriving at hospital, the initial management used in the A&E department and the recommendations given to the patient to avoid a further admission. You should be wearing appropriate protective clothing (e.g. aprons, gloves and eye protection) as epistaxis can cause marked blood splattering and spillage. If available, patients can also wear a surgical face mask covering their mouth. This prevents patients coughing and spraying blood towards the attending doctor! A benzodiazepine can be used if the patient is overly anxious. Advise the patient to pinch the alar of both nostrils firmly for 10–15 minutes, WITHOUT RELEASING THE PRESSURE and breath through their mouths. Finally place a glove filled with ice pressed upon the bridge of nose to help limit the bleeding. If bleeding is controlled, proceed to nasal cauterisation, however, if it is not controlled consider anterior packing.

2. Nasal cauterisation

Nasal cautery involves the application of a caustic agent, such as silver nitrate, to stop the bleeding in the anterior part of the nasal septum. It is indicated whenever bleeding has stopped and can be used to stop bleeding if direct pressure has failed.

Before starting, explain the procedure to the patient. Serious complications related to nasal cautery are rare but include septal crusting, perforation and reaction to either the cautery agent or the local anaesthetic. Common side effects include: stinging during the procedure; itching; and reoccurrence. You should be careful when using silver nitrate as it cauterises anything it touches so do not touch the skin, nasal alar or nasal mucosa. Ensure that you do not cauterise both sides of the septum for fear of septal perforation.

Equipment needed include:

- silver nitrate stick
- Lignocaine and adrenaline-soaked pledget
- headlamp/lighting facility
- suctioning facilities
- Thuducum nasal speculum.

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It is essential to wear protective clothing (e.g. gloves and aprons) throughout this procedure. Practical Procedures.

It is essential to wear protective clothing (e.g. gloves and aprons) throughout this procedure. Get adequate lighting exposure and suction out any debris from the nose, then soak 2 small pledgets with the lignocaine spray and insert gently into nose. This not only helps stop the bleeding but anaesthetises the area. Use a nasal speculum to inspect the nostril of each side of the nose and look for bleeding points on the nasal septum (often looks like little dots). Paint the bleeding point and the area with a small radius of silver nitrate for 10 seconds[2]. Finally, dispose of the silver nitrate safely without touching yourself or the patient.

3. Anterior packing

Anterior packing involves inserting a pack into the anterior nose to obstruct the steady flow of blood and allow the body's haemostatic mechanisms to take hold. Packing is indicated when first aid and/or cautery has failed to stop the bleed but most authorities use it as a first line intervention.

The two commercially available products used to pack the nose in A&E include merocel tampon (Medtronic Inc., Minneapolis, USA) and rapid rhino anterior balloon pack (Arthrocare UK Ltd, Leicester, UK). Both merocel and rapid rhino are similar in their efficacy but it has been reported that rapid rhino affords better patient tolerance during insertion and removal of the pack3. It all depends upon availability in your department.

Equipment needed:

- merocel pack/rapid rhino
- lubricating jelly
- lighting facilities
- gauze and tape.

Ensure you are wearing protective clothing and have the patient sitting up, spitting any blood into a dish. Explain the procedure to the patient and warn them that it may be uncomfortable. Complications are rare but include septal necrosis, anosmia, sinusitis, toxic shock syndrome (if tampon is not removed) and failure to control bleeding. Contraindications include respiratory compromise which needs addressing before inserting the pack.

Lubricate the tampon quickly (it tends to swell and lose shape fast) and insert horizontally along the floor of the nasal cavity. Ensure that the whole pack is in the nose. For the rapid rhino, inflate the cuff after insertion to expand the balloon. Apply gauze and tape to hold pack in place and tape strings to the cheek. Advise the patient that they will have to be admitted to hospital to monitor their condition for a couple of days or so. Finally, inform the ENT team about the patient and arrange for transfer.

4. Posterior packing

Merocel or rapid rhino packs are effective for anterior epistaxis. Often bleeding from the contralateral nostril is an indication of posterior bleeding. This may require posterior nasal packing. Posterior packing involves inserting a pack into the nasopharynx and is performed when anterior packing is insufficient to control the bleeding. The physician is often alerted when there is dripping of blood from the other nostril or in the back of the throat despite the presence of an anterior pack.

It is advisable to discuss with a senior ENT colleague before posterior packing as this can be distressing for the patient particularly in the inexperienced hands.

Sometimes it may be possible to avoid posterior packing. Other measures, such as gentle packing with paraffin-soaked gauze or other haemostatic agents, may be better alternatives. If bleeding is not controlled with a posterior pack the options include: endoscope cauterisation; or sphenopalatine artery ligation.

There are many commercial balloon tampons available on the market but often a 10–14F Foley catheter will suffice.

Equipment Needed:

- 12ch Foley catheter
- lubricating jelly
- lighting facilities
- gauze and tape
- padded umbilical clip.

It is important to have an otolaryngology senior with you to supervise you. Ensure you are wearing protective clothing and have the patient sitting up, spitting any blood into a dish. Explain to the patient the procedure and warn them that it will be uncomfortable. Complications are similar to anterior packing. Contraindications include respiratory compromise which needs addressing before inserting the pack and extensive facial trauma. Appropriate sedation is advisable as the procedure is uncomfortable.

Gain adequate lighting and suck out any clots in the nose. Spray the nose with a topical local anaesthetic and insert a lubricated catheter into the nostril. Once the catheter tip can be visualised in the back of the throat inflate the balloon and gently pull the catheter back until it sits on the posterior nasal cavity. Maintain tension on the catheter and attach a padded umbilical clip across to prevent alar necrosis and maintain position. Inform the ENT team and arrange transfer.

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Discharge planning

Patients can be discharged once the bleeding has stopped although those with a pack should be monitored for at least 1 day. Before discharge it is necessary to cauterise any visible bleeding points and to prescribe a topical antibiotic to prevent infection and secondary haemorrhage.

To prevent reoccurrence advise the patient:

- To avoid picking the nose or traumatising it.
- To rest as much as possible and not to engage in any vigorous exercise.
- Avoid manoeuvres that increase intra-abdominal pressure (e.g. straining, provide a laxative if necessary, coughing, heavy lifting).
- Avoid sneezing through the nose. Instead occlude the nose and sneeze through the mouth.

For about 2 weeks

If a nose bleed occurs again explain correct technique for prevention and to return to the A&E department if it is not controlled after two attempts.

Best of 5 EMQs

1. A 50-year-old man presents to A&E with epistaxis for 10 minutes that has now resolved after first aid measures. This is his first nose bleed and he is very anxious. BP 190/110 Pulse 80bpm.

a. Insert anterior nasal pack.
b. Refer to ENT team.
c. Insert posterior nasal pack.
d. Observe and give a benzodiazepine if necessary.
e. Insert paraffin-soaked gauze.

2. A 80-year-old man presents to A&E having been bleeding from both nostrils for 90 minutes. He is orientated and conversational while his blood pressure is 80/60mmHg, his pulse is 90bpm sats are 98% on air.

a. Insert posterior pack.
b. Insert anterior pack.
c. Assess and manage airway breathing and circulation.
d. Call senior ENT member.
e. Book for emergency sphenopalatine artery ligation.

Answers

1. d. Notes. The bleed is likely to originate from the anterior nasal septum because these occur more frequently and are more likely to be controlled with first aid measures alone. Since the bleed has completely resolved there is little need for active management (e.g. insertion of anterior/posterior packs or paraffin-soaked gauze) all of which referral to ENT would be necessary. In this case, observation of blood pressure and benzodiazepine to control symptoms of anxiety is recommended. Ensure that the patient is well enough before discharge and that any bleeding points are cauterised. A 10-day course of topical antibiotics is administered and advice is given to prevent reoccurrence. In this case, follow-up with his GP regarding elevated blood pressure would be appropriate.

2. c. It is essential to initially assess and manage the airway, breathing and circulation in all patients who present with epistaxis. This should be done in A&E where facilities for resuscitation are available. Only when patients are haemodynamically stable should specific measures to control the bleed be started. In this patient, large bore IV access should be obtained (preferably in both arms) and blood taken for FBC, U&E, LFTs, INR/clotting (if on warfarin) and a "group and save" depending on severity. Suitable fluids (e.g. colloid/crystalloid) should be given to restore the blood pressure. It is essential to continually recheck ABCs until the patient is stable. Once he has been adequately resuscitated initiate measures to stop the bleeding.

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CAN THIS ADULT WHO HAS SUSTAINED A HEAD INJURY GO HOME?

Elizabeth Gillott and Ashis Banerjee

A 24-year-old man attended his local A&E department in the early hours of the morning after being assaulted.
Patient Management.

Case report

A 24-year-old man attended his local A&E department in the early hours of the morning after being assaulted. He was accompanied by his friend who had witnessed the incident. They gave a history of the patient being attacked while leaving a pub. He was knocked to the ground and received repeated blows to the face and head. His GCS was 14/15 on arrival in the A&E department. Prior to the assault, he was fit and well and was not taking any medication, prescribed or otherwise.

Questions

1. How would you clinically assess him?
2. What else do you need to know?
3. What specific investigations and assessment should be performed?
4. Does he need a CT scan?
5. Can he have analgesia?
6. Can he go home?

Answers

1. The purpose of clinical assessment is to ascertain the presence of primary brain injury, if any, and to determine the potential risk of secondary brain injury from delayed intracranial haematoma. Immediate assessment after evaluation of ABCs.

This comprises:

a. Determine the conscious level.

Eye Response:	Open spontaneously	4
	Open to verbal command	3
	Open to pain	2
	No response	1
Verbal Response:	Talking and orientated	5
	Confused/disorientated	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Motor Response:	Obeys commands	6
	Localises pain	5
	Flexion/withdrawal from pain	4
	Abnormal flexion	3
	Extension	2
	No response	1

Table 1: Glasgow Coma Scale (Adults)1.

i. Record the conscious level and always subdivide to the component scores. The score attributed should reflect the best response in each area of testing.
ii. Unconsciousness in trauma generally means no eye response and GCS≤8.
iii. An anaesthetist should be called if a patient has a GCS≤8 as the patient may need a definitive airway, but at the least, should have the airway monitored and supported.

b. Is there any possibility of cervical spine injury?

i. Cervical spine injury is not confined to "trauma-call" patients. Even those that walk into the department are at potential risk. The mode of injury should be considered in all patients.
ii. Patients receiving repeated blows to the head and face are at increased risk of cervical spine injury. If this is suspected, clinically in the presence of neck pain, the cervical spine should be immobilised and protected prior to radiological clearance.

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2. History

a. Had he consumed alcohol? Document how much had been drunk if the information is available. His GCS could be affected by alcohol (or drug) ingestion. Do not assume that a decreased GCS is attributable to alcohol.

b. Obtain an accurate history of events. It is important to ask exactly what happened at the time and since. Witnesses and ambulance crew should be quizzed as collateral history is extremely important in those patients who are unable to recount events. Find out whether the patient:

i. Lost consciousness?
ii. Vomited?
iii. Had any seizures?

c. Does the patient have other injuries?

d. What is the patient's pre-morbid medical state? Any allergies, medications (ask specifically about anticoagulation) and past medical history.

e. Patient's next of kin.

3. Physical examination

a. Vital signs. Record the respiratory rate and depth; the oxygen saturation; the pulse rate, character and rhythm; the blood pressure and temperature.

b. Regularly reassess GCS.

c. Eye signs. Document pupil size, response to light and accommodation. Unilateral dilatation may be due to a number of causes (traumatic mydriasis; oculomotor nerve compression leading to raised ICP – the latter is unlikely in the presence of a GCS of 14). Be aware of patients with pre-existing eye signs (e.g. from Horner's syndrome, a prosthetic eye or physiological anisocoria). Check for full range of eye movements and presence of nystagmus. Document any visual disturbance (e.g. diplopia, blurring of vision and visual field defect). Observe for "panda eyes" and subconjunctival haemorrhage.

d. Face, scalp and head. The patient received blows to his face and head. Consider the possibility that the patient has received facial injuries. Inspect the scalp, face, nose, eyes, ears and mouth for obvious injuries. Patients that have been assaulted may press charges against the assailant, and it is therefore even more important that the injuries are carefully documented at the time of presentation. Inspect the ear canal, looking specifically for haemotympanum or bleeding from the external auditory meatus (sign of a basal skull fracture). Note whether there is CSF otorrhoea or rhinorrhoea. Dropping fluid onto blotting paper would reveal a "double ring" caused by separation of fluid mixtures containing similar quantities of blood and CSF.

e. Limbs. Check tone, power, sensation and reflexes and observe for other injuries. Abnormalities may be detected that result from brain injury or an intracranial haematoma. An intracranial haematoma may require urgent surgical intervention, and therefore transfer to a specialist neurosurgical unit

f. Facial views should be obtained if facial injuries are suspected. Suspicion should be raised by the presence of facial bruising with bony tenderness, deformity and malocclusion.

4. CT scan

a. NICE guidance suggests that the following groups of patients should receive an immediate CT scan (within 1 hour).
• GCS less than 13 on initial assessment in the A&E department.
• GCS less than 15 at 2 hours after the injury on assessment in the A&E department.
• Suspected open or depressed skull fracture.
• Any sign of basal skull fracture (e.g. haemotympanum, "panda eyes", cerebrospinal fluid leakage from the ear or nose, Battle's sign).
• Post-traumatic seizure.
• Focal neurological deficit.
• More than 1 episode of vomiting.
• Amnesia for events more than 30 minutes before impact.

• GCS less than 13 on initial assessment in the A&E department.
• GCS less than 15 at 2 hours after the injury on assessment in the A&E department.
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• Any sign of basal skull fracture (e.g. haemotympanum, "panda eyes", cerebrospinal fluid leakage from the ear or nose, Battle's sign).
• Post-traumatic seizure.
• Focal neurological deficit.
• More than 1 episode of vomiting.
• Amnesia for events more than 30 minutes before impact.

Table 2: Criteria for immediate request for CT scan of the head (adults)[2].

b. For patients who need a CT scan but not within 1 hour, they can be admitted for overnight observation and scanned the next morning. These include patients who present out of hours and in addition to a period of loss of consciousness or amnesia, or have any of the following risk factors present:

i. Age 65 years or older.
ii. Coagulopathy (e.g. history of bleeding, clotting disorder, current treatment with warfarin).
iii. Dangerous mechanism of injury (e.g. a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 meter or 5 stairs).

c. CT scan should be requested immediately in the above groups of patients during normal working hours.

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5. Analgesia

a. In the first instance, patients can safely take simple analgesics, such as paracetamol, in the absence of any contraindications.

b. Pain should be managed effectively because it can lead to a rise in intracranial pressure. Reassurance and splintage of limb fractures are important. Evacuation of a full bladder will further reduce irritability. Severe pain should be treated with small doses of intravenous opioids titrated against clinical response and baseline vital signs.

6. Home

a. If CT is not indicated and the patient's risk of clinically important brain injury is felt to be low enough to warrant discharge, the patient should be assessed for other factors that might indicate hospital admission:

i. Drug or alcohol intoxication.
ii. Other injuries.
iii. Shock.
iv. Suspected non-accidental injury.
v. Cerebrospinal fluid leak.
vi. Lack of appropriate support structure in the community.

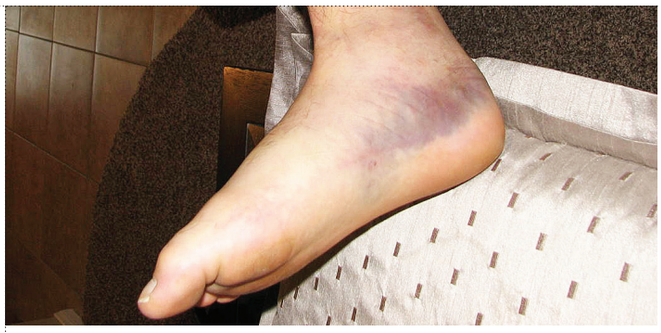
b. The patient should have someone suitable at home to supervise if, despite the head injury, safe discharge home is permissible. Those patients with no carer should only be discharged if suitable supervision can be arranged. Failing this, they should be kept in hospital until the risk of early complications is deemed negligible.

c. All patients being discharged should receive written head injury advice that indicates the "red flags" requiring further medical attention as well as safety advice.

Our patient lost consciousness at the scene during the attack for around a minute, but his GCS was 14/15 when the ambulance crew arrived (E3, M6, V5). He appeared drowsy which may have been a result of head injury, alcohol ingestion or a reflection of the time of day (4am) and his GCS improved to 15 and remained that way. He had not vomited or had a seizure, but had a period of anterograde amnesia. On later questioning, he was able to recall the events up until the assault, and after regaining consciousness.

He had left-sided facial bruising. He found it painful to bite down and open his jaw. He also had a painful swollen left foot and was unable to bear weight on the left lower limb. There was no neurovascular deficit in the foot. No focal neurological deficit could be found.

Facial views were obtained which revealed a zygomatic fracture. The maxillofacial team were contacted for further management.



Imaging of his foot revealed multiple tarsal fractures. He was admitted under the orthopaedic team for analgesia, high elevation and consideration of fracture fixation. He had several fractures, which caused the patient to experience significant pain. As he was an inpatient, he was able to receive opioid analgesia titrated to his pain level and had head injury observations for 24 hours. He did not develop any worrying symptoms or signs and did not proceed to CT scanning of the head.

Alcohol ingestion was a confounding factor. He was compliant and his GCS was 14 on arrival but 15 on later reassessment. Had he not had other injuries, he would have stayed for at least 6 hours in the A&E department observation ward and been discharged home in the care of a responsible adult with head injury advice, once the effects of the alcohol had worn off.

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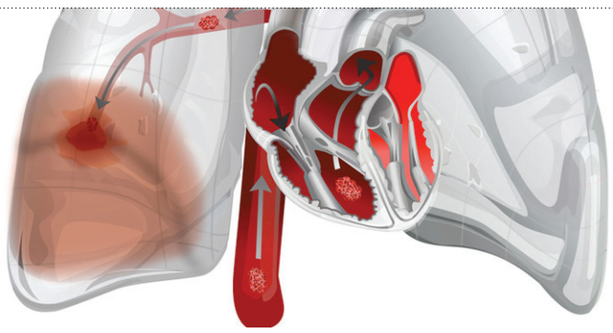
A CASE OF D-DIMER NEGATIVE PULMONARY EMBOLISM

Lawrence Li and Brodie Paterson

Pulmonary embolism (PE) presents a diagnostic challenge for the emergency physician.
Patient Management.



Abstract

Pulmonary embolism (PE) presents a diagnostic challenge for the emergency physician. Undiagnosed PE is associated with a mortality of up to 30% although more recent data suggests a lower mortality rate[1]. The advent of D-dimer testing looked to bring a sensitive "rule-out" test to EM. Reviews have shown the D-dimer testing is only accurate in the low-risk group[2]. This article highlights a case of a patient who despite having clinical signs of a pulmonary embolism had their diagnosis delayed due to a negative D-dimer result.

In conclusion it should be remembered that D-dimers are not sensitive enough to rule out PE in patients at high risk. Although the use of D-dimers initially promised to be very useful in ruling out PE subsequent reviews have suggested it is only accurate in low-risk patients.

Case history

A 54-year-old woman presented to the Emergency Department with sudden onset of left-sided chest pain lasting 1 hour that was described as sharp and worsened by inspiration. There was an associated cough productive of white sputum but no history of haemoptysis.

On further questioning, the patient mentioned two previous admissions to the same hospital with similar chest pain. The first episode was in 2006 and the second just 4 months prior to this presentation. She was diagnosed with chest infection on both occasions and discharged with a course of antibiotics. The patient was a former smoker and rarely drank alcohol. Of note there was no preceding history of prolonged bed rest, recent surgery or long-haul flights. She had a past medical history of pustular psoriasis and asthma. The only medication she took was hormone replacement therapy. On examination, the patient was in discomfort with intermittent chest pain. She was alert and orientated with a mildly raised temperature of 37.9°C. Although she subjectively denied any breathlessness on examination, her respiratory rate was raised at 28 breaths per minute with an oxygen saturation of 93% on air. She was also tachycardic at 120 beats per minute and her blood pressure was 146/80mmHg. Apart from some crepitations on auscultation at her left lung base, the remainder of her examination was unremarkable. There was no clinical evidence of deep vein thrombosis (DVT).

Her electrocardiograph showed sinus tachycardia and evidence of an s-wave in lead I but no other acute changes. Her arterial blood gas on high flow oxygen (15L/min) showed a pH of 7.38, $PaCO_2$ of 5.59kPa and a PaO_2 of 8.16kPa. The chest X-ray showed clear lung fields. A provisional diagnosis of pulmonary embolism was made in the Emergency Department and she was admitted to the medical assessment unit for further review and treatment.

Her admission blood tests showed both a raised white cell count of 14.9 x 10^9/ml (neutrophils count of 11.2 x 10^9/ml) and C-reactive protein (CRP) of 75mg/L. Her other biochemistry results were all within the normal ranges. Her coagulation screen was normal as was her D-dimer assay (20ng/ml). The local laboratory uses the reference value of 189ng/ml as cut-off for excluding venous thromboembolism. Due to the clinical symptoms and her recurrent presentation with pleuritic chest pain, a ventilation: perfusion (V:Q) scan was requested in spite of her low D-dimer score. This showed a combination of matched and mismatched perfusion defects in both lungs suggestive of multiple bilateral PE of varying ages. The patient was treated with low-molecular weight heparin and discharged with oral anticoagulation with warfarin. She was subsequently reviewed in the outpatient medical clinic. Interestingly, the patient's D-dimer level (119ng/ml) remained below the cut-off value prior to her discharge.

Review of the case notes from her previous admissions showed that the patient presented with breathlessness and pleuritic chest pain on both occasions. In addition, the patient was found to be hypoxic on air with an unremarkable chest radiograph. The patient had mildly raised inflammatory markers but D-dimer tests had been negative for thromboembolism. Although the possibility of PE was raised, she was diagnosed with chest infections on the basis of the negative D-dimer tests.

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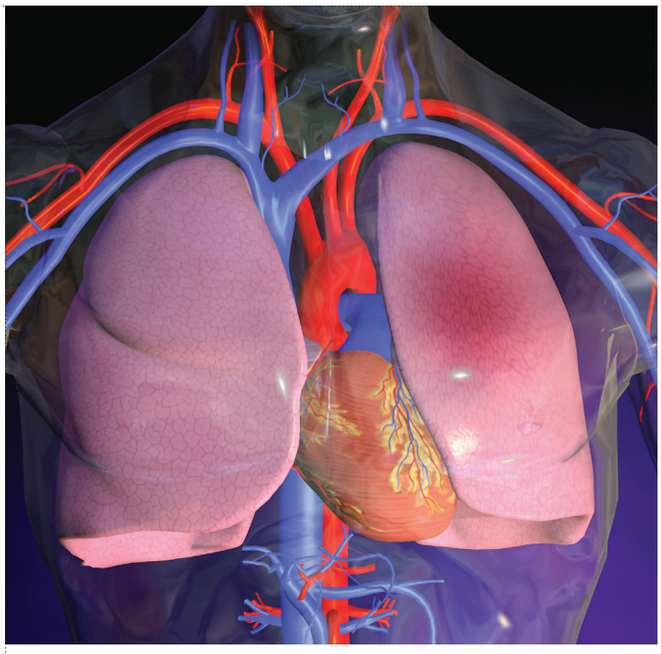
A CASE OF D-DIMER NEGATIVE PULMONARY EMBOLISM

Lawrence Li and Brodie Paterson

Discussion

The diagnosis of PE is often challenging because the clinical features can mimic other conditions when patients present with non-specific symptoms. Undiagnosed PE and subsequent delay in appropriate anticoagulation carry significant mortality and morbidity[1]. D-dimer assays are often used in the acute sector, such as the Emergency Department, to exclude the diagnosis. The careful selection of patients for D-dimer testing reduces the need for further investigations. However, injudicious use may be misleading as the test is neither 100% sensitive or specific. Previous studies have shown D-dimer assays to have a high sensitivity and negative predictive value for excluding the diagnosis of DVT and PE[2, 3]. The British Thoracic Society (BTS) has published a set of guidelines on the diagnosis of PE. D-dimer is recommended if the patient is deemed to be at low risk after clinical assessment. Although the use of D-dimer is also recognised in patients at intermediate risk, this is dependent on the sensitivity of the individual D-dimer assay[2]. Various clinical scoring systems have been devised to help with establishing the clinical probability, of which the Wells Score has been extensively validated[4, 5].

This patient presented with pleuritic chest pain and was found to be hypoxic on high flow oxygen. She had a mild pyrexia along with a raised white cell count and CRP. These features suggested a alternative diagnosis such as pneumonia. Her relatively high $PaCO_2$ was unexpected given her increased respiratory rate and lack of known respiratory pathology, such as chronic obstructive lung disease. Her chest X-ray was unremarkable and the use of hormone replacement therapy is considered a minor risk factor[2] and therefore the diagnosis of PE is as likely as a chest infection. On reviewing her previous admission records when she had been discharged with the diagnosis of chest infection, the patient was found to be hypoxic on arterial blood gas but the chest X-ray was reported as showing clear lung fields. Although the clinical probability of PE on those occasions was not commented on, the suspicion of PE was clearly raised and included in the list of differential diagnoses. However, PE was discounted on both counts because of negative D-dimer results.



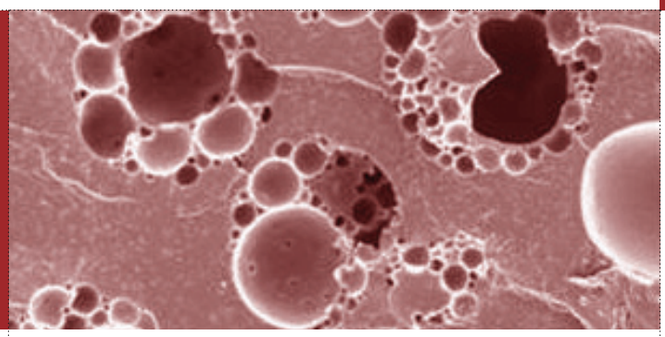
Breen et al. recently published a similar case report of a patient with D-dimer "negative" PE[6]. The report described a patient whose D-dimer level was 495.1ng/mL (cut-off being 500ng/mL) using an ELISA assay but was shown to have radiographic evidence of PE. Our case differs in that our patient's D-dimer level was well below the cut-off value despite being at intermediate risk after clinical assessment.

Our hospital uses the Biopool Auto-Dimer test. For the exclusion of DVT at least, this assay has been shown to have a stand-alone sensitivity of 95.2% at a cut-off value of 189ng/mL[3]. This value is also used for excluding PE in our hospital. This report highlights a case of D-dimer negative PE where the diagnosis has been discounted on previous occasions based on negative D-dimer tests. The use of D-dimer assays in selected patients can be useful. However, it should only be relied upon in patients who are at low clinical risk. In a case where other diagnoses is unlikely, more definitive investigations in the form of V: Q scan or computed tomography of pulmonary angiography are necessary. One could argue that the use of D-dimer test may have even been dangerous in this case as it gave false reassurance for excluding a potentially fatal condition.

A CASE OF D-DIMER NEGATIVE PULMONARY EMBOLISM

Lawrence Li and Brodie Paterson

D-dimer testing.
Patient Management.



Test Yourself

Q1. D-dimer testing may be useful in which of the following 5 patients?

a. A 75-year-old patient with a history of lung cancer presented with chest pain and increased shortness of breath.
b. A 21-year-old woman presenting with chest pain, saturating at 98% on air and the following blood gas results: pH 7.55, PaO_2 14.5kPa, $PaCO_2$ 2.9 on room air.
c. A patient presenting with shortness of breath and Wells Score of 4.5 (moderate probability).
d. A 68-year-old man with recent right total knee replacement presenting with swelling of the right calf.
e. A 30-year-old pregnant woman presenting with increased shortness of breath and tachycardia with the following blood gases results: pH 7.45, PaO_2 10.2kPa, $PaCO_2$ 3.9 on room air. Examination was normal.

Q2. Which of the following statements is TRUE?

a. Cigarette smoking is a recognised major risk factor for PE.
b. D-dimer should be routinely used as a "screening test" when PE is suspected.
c. Only a negative D-dimer test is useful if another diagnosis (other than PE) is unlikely and there is absence of major risk factors.
d. The SimpliRED (agglutination) test should be used in patients with an intermediate probability of having a PE.
e. Patients with DVT/PE should always be treated as an inpatient

Answers

Q.1. b. Patients c, d and e are high risk of having a DVT or PE. Additionally, D-dimer levels may be raised in the patient a due to a history of malignancy. Patient e has a risk factor for PE and her ABG results showed an increased arterial-alveolar gradient despite a near normal PaO_2.

Q.2. c. Negative D-dimer results are useful only in cases where the patient is deemed to be at low risk after clinical assessment. Cigarette smoking is not a recognised major risk factor for PE. The SimpliRED assay should only be used in patients at low risk of PE. Patients with confirmed venous thromboembolism, but whose condition is stable, can be treated as outpatients. Please refer to the BTS Guidelines[2].

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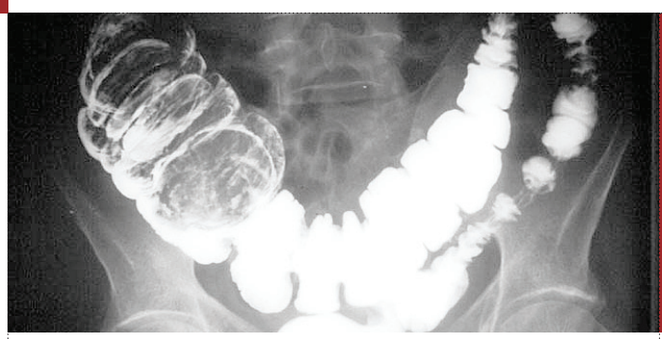
RARE MANIFESTATIONS OF RIGHT ILIAC FOSSA PAIN

Mouhamed El-Sayad and Norzeihan Jan Bappu



Right iliac fossa.
Patient Management.

Diagnosis and management of right iliac fossa pain can be very challenging. It presents in approximately 50% of all acute abdomen cases, and although appendicitis raises the first suspicion during initial assessment, the preoperative diagnosis is seldom accurate. Cross-referrals may occur between specialties and unexpected pathology may sometimes only be encountered as operative or histo-pathological finding. We present two rare entities of right iliac fossa pain, which signify the importance of clinical assessment and differential diagnoses in order to ensure correct diagnosis and management are achieved.

Case 1: Appendiceal stump appendicitis

We present a case where the relatively common entity of appendicitis was in no way suspected prior to cross-sectional imaging, which incidentally revealed rare appendiceal stump appendicitis, significantly changing clinical management.

A healthy 28-year-old Nigerian man presented with 4-day history of central abdominal pain which migrated to the right iliac fossa and associated with episodes of vomiting. He denied chest pain, dyspnoea or change in bowel function. His past medical history was significant for open appendicectomy in Nigeria 10 years ago. On observation, he had low-grade pyrexia of 37.8^{0}C, his oxygen saturation was 98% on room air, blood pressure of 100/70mmHg and respiratory rate of 25 breath/min. Physical examination revealed very tender right iliac fossa on superficial palpation with voluntary guarding but no organomegaly or peritonism. Rovsing's sign was positive. Previous Lanz excisional keloid scar was noted.

Laboratory investigation showed marked leucocytosis of 18.1 × 10^{9} cells/L (absolute neutrophil count: 13.6; 75.1%), blood urea 9 and serum creatinine 68. Serum electrolytes were as follows: sodium 133, potassium 4.2, chloride 95 and CO_2 23. Urinalysis was unremarkable with only a trace of ketones. Plain abdominal X-rays were obtained which showed mild faecal loading and no signs of bowel obstruction. Given the pathognomonic features of appendicitis despite previous appendicectomy, an abdominal CT scan with contrast was performed which showed a perforated retrocaecal blind-ended viscous with faecolith near its origin (see Figure 1). The patient was commenced on intravenous ceftriaxone and metronidazole and we proceeded with laparoscopic appendicectomy, which revealed acute gangrenous 8cm appendiceal stump and adjacent thickening of the ileocaecal region. A localised resection with preservation of ileocaecal valve was performed. Histopathological inspection of the excised appendix verified the diagnosis of acute gangrenous appendicitis with associated inflammatory infiltrates.

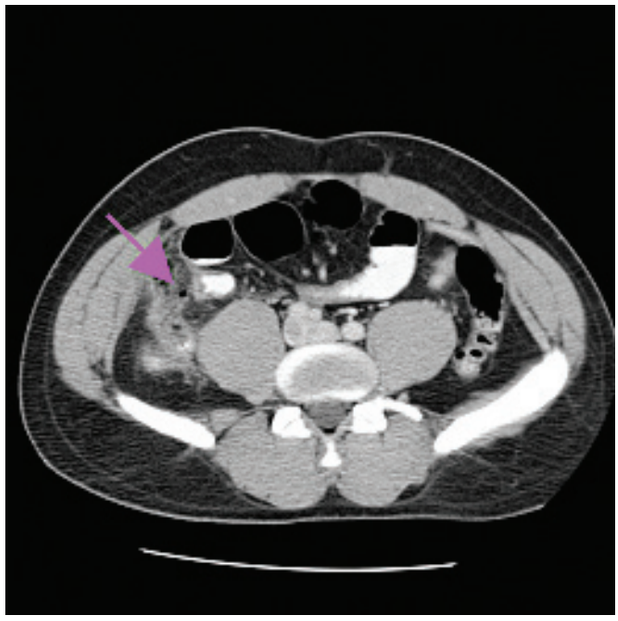


Figure 1: Appendiceal stump appendicitis as seen on CT.

The patient was given three further doses of antibiotics prior to discharge. His post-operative recovery was complicated with a readmission 10 days later with further low-grade pyrexia and mild abdominal tenderness. An abdominal ultrasound was carried out which showed a 4cm x 4cm pelvic collection which was too small for drainage. His symptoms settled with conservative management and he was discharged 4 days later, with follow-up arranged in clinic.

Case 2: Ovarian dermoid cyst

A 39-year-old lady presented to the A&E department with sudden onset of severe generalised abdominal pain, which was markedly worse at the right iliac fossa. This was associated with one episode of vomiting at the onset of the pain. Her past medical history only included paraumbilical hernia. On examination she was apyrexial, blood pressure was 92/44mmHg, pulse 100bpm and oxygen saturation was 99% on room air. On abdominal examination, there was no organomegaly but she was tender in the right iliac fossa with guarding and rigidity. Rovsing's sign was positive.

RARE MANIFESTATIONS OF RIGHT ILIAC FOSSA PAIN

Mouhamed El-Sayad and Norzeihan Jan Bappu

Laboratory results showed a raised white cell count of 16.4 × 10^9 cells/L (neutrophis of 14.9); her urea and electrolytes were all within normal range. Her Ð-HCG level was negative. Plain abdominal X-rays showed no abnormalities. The patient was kept nil by mouth and resuscitated with intravenous fluids and opioid analgesia which only mildly relieved the pain. Due to the unknown cause of her pain and to rule out appendicitis, she had an abdominal ultrasound which showed a 5.5cms x 5.2cms cystic structure on her right ovary and normal gall bladder, kidneys and ureters. She was rushed to theatres given the assumptive diagnosis of right ovarian cyst with torsion of the right ovary. Laparoscopic right salpingo-oopherectomy was performed and on retrieval of the ovary from the main port, there were some hair-like structures seen surrounding the cyst (see Figure 2). Histopathology verified the diagnosis of dermoid cyst.

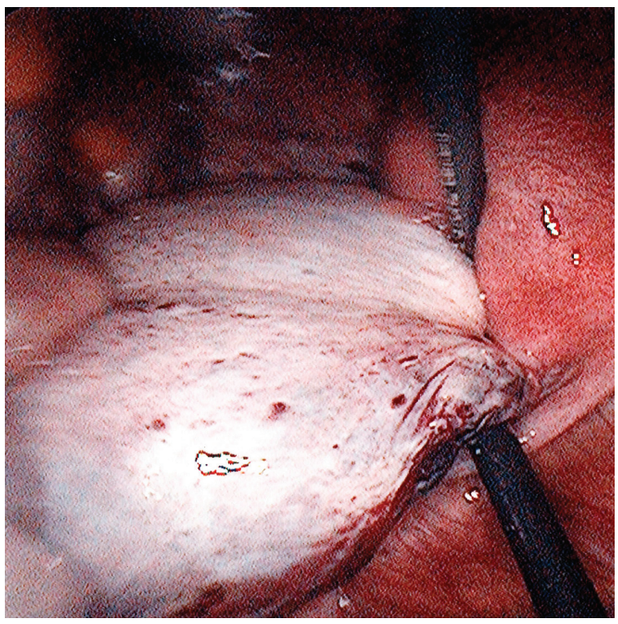


Figure 2: Ovarian cyst found during operation necessitating right salpingo-oopherectomy.

The patient had an uneventful post-operative recovery and was sent home the next day.

Discussion

These two cases demonstrate variable presentations of right iliac fossa pain across the specialties, particularly in the A&E department, surgical and gynaecological units.

Although common things are common, it is pertinent for Foundation doctors to take into account all the differential diagnoses and symptoms assessments as rare cases, such as post-appendiceal stump appendicitis or dermoid cyst may still be a possibility. These two cases demonstrate that relevant history and examination are pivotal to making a safe working diagnosis and can establish the need for further appropriate investigations, such as CT scan with contrast and ultrasound scan, as seen in these two cases respectively.

Stump appendicitis is associated with recurrent inflammation of remnant appendix tissue. Presenting symptoms are similar to initial appendicitis, with focal right iliac fossa pain in 90% of cases and increased leucocytosis in 85% of reported cases[1, 2]. The interval between first operation and subsequent presentation may vary from 2 months to 50 years[1]. Stump appendicitis should be included in the differential diagnoses of any patient presenting with right iliac fossa pain, even with history of previous appendicectomy.

Case 2 demonstrates a rare manifestation of dermoid cyst presenting as right iliac fossa pain. A dermoid cyst develops from a totipotential germ cell which is retained within the ovary. Being totipotential, the cell can give rise to all orders of cells necessary to form mature tissues and often recognisable structures, such as hair, bone and sebaceous material, neural tissue and teeth. Ovarian cysts may mimic presentation of appendicitis as seen in Case 2 where the Rovsing's sign was positive. Uncertainty in clinical grounds should be allayed by further imaging investigation, such as pelvic and abdominal ultrasound scans. Historical evidence dated back to more than 100 years ago whereby patient initially thought of having appendicitis was discovered to have ovarian cyst at the operation[3]. Specific urinary and laboratory testing, such as Ð-HCG level, is important to exclude ectopic pregnancy even in suspected cases of ovarian cysts and appendicitis. Inflammatory markers, such as white cell count and C-reactive protein (CRP), can also be a helpful diagnostic tool for diagnosis of appendicitis versus other surgical causes in acute right iliac fossa pain[4].

Radiological imaging is useful in management of right iliac fossa pain to minimise misdiagnosis based on clinical grounds only. These two cases showed the use of ultrasound and contrast-enhanced CT scans in different case setting. The sensitivity and specificity of ultrasound in detecting an ovarian cyst is 94% and 99% respectively. CT scan is the preferred imaging for suspected appendicitis with diagnostic accuracy of 95% to 99% and also useful for characterising periappendiceal inflammatory mass which might not be visible on ultrasound.

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RARE MANIFESTATIONS OF RIGHT ILIAC FOSSA PAIN

Mouhamed El-Sayad and Norzeihan Jan Bappu

The role of diagnostic laparoscopy in the management of the patient with right iliac fossa pain is in conjunction with imaging techniques (CT and ultrasound scanning). Patient Management.

The role of diagnostic laparoscopy in the management of the patient with right iliac fossa pain is in conjunction with imaging techniques (CT and ultrasound scanning). Consensus European Association of Endoscopic Surgeons (EAES) guidelines have emphasised the value of routine laparoscopy as a diagnostic tool in young women as symptoms may evoke various differential diagnoses including gynaecological causes and unnecessary incision could be avoided for cosmetic reason[5]. Laparoscopy is also a useful in male patients where preoperative diagnosis cannot be made clinically.

It is imperative that these patients are followed-up carefully after the operation to avoid complications, such as pelvic collection, seen in the first case. Histopathology report must be obtained post-operatively to exclude other unexpected causes, such as malignancy. Carcinoid has a prevalence of 0.5% of all appendicectomies6, whereby malignant ovarian cysts may occur in up to 3% of benign-looking cyst in pre-menopausal women[7].

Conclusion

These two cases feature similar presenting complaints of right iliac fossa pain albeit with completely different pathologies, hence different course of clinical management.

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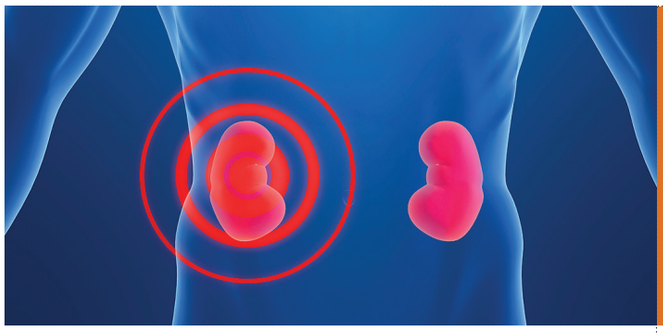
UROLOGICAL EMERGENCIES: QUESTIONS



A great time to test your knowledge.
Test Yourself.

Q1. Differential diagnoses in urological emergencies.

For patients who present to the A&E department with loin pain, choose the most likely diagnosis given the clinical information.

1. Appendicitis.
2. Leaking abdominal aortic aneurysm.
3. Bowel perforation.
4. Diverticulitis.
5. Pyelonephritis.
6. Urinary tract injury.
7. Urosepsis.
8. Renal colic.

Q1.1. A 78-year-old diabetic man with an indwelling catheter who is both hypotensive and hypothermic with generalised abdominal pain as well as bilateral loin pain.

Q1.2. A 26-year-old woman with dysuria, rigors, right-sided abdominal pain through the loin into the back, a pyrexia of 38.9 and a urine dip positive for nitrites and leucocytes.

Q1.3. A 24-year-old rugby player referred from GP with microscopic haematuria and right-sided loin pain. His non-contrast CT is negative for obstruction or stone.

Q1.4. A 53-year-old lady with stable observations with severe pain in the right loin, who is nauseous and rocking on the side of her bed.

Q1.5. A 69-year-old male smoker with hypotension and tachycardia complaining of pain in the left loin radiating through to his back.

Q2. Initial investigations in urological emergencies.

For a GP admission to the A&E department with query renal colic choose the most appropriate initial investigations given the clinical information. (MC&S = microscopy, cultures and sensitivity).

1. FBC, U&E.
2. FBC, U&E, Urine dip +/- MC&S.
3. FBC, U&E, Urine dip +/- MC&S, beta-HCG.
4. FBC, U&E, Urine dip +/- MC&S, digital rectal exam.
5. FBC, U&E, Urine dip +/- MC&S, clotting screen.
6. FBC, U&E, Urine dip +/- MC&S, renal tract ultrasound.
7. FBC, U&E, Urine dip +/- MC&S, clotting screen, renal tract ultrasound.
8. FBC, U&E, Urine dip +/- MC&S, bladder scan.

Q2.1. A 64-year-old woman with painless macroscopic haematuria.

Q2.2. A 27-year-old woman who is acutely unwell with symptoms of renal colic.

Q2.3. A 57-year-old woman referred from the GP with symptoms of renal colic, the referral letter informs you urine dip is clear.

Q2.4. A 73-year-old man with dementia who is unable to give any history but presents with severe suprapubic pain and a lower abdominal mass which is dull to percussion.

Q2.5. A 64-year-old man with history of recent weight loss and urinary incontinence has become “off-legs”.

Q3. Choices of imaging in emergency presentations of query renal colic given the clinical information:

1. FAST ultrasound scan.
2. Renal tract ultrasound scan.
3. CT with contrast.
4. CT without contrast.
5. IVU.
6. Abdominal X-ray.
7. Abdominal X-ray and erect chest X-ray.
8. KUB.

UROLOGICAL EMERGENCIES: QUESTIONS

For a patient who presents with the symptoms of acute onset renal colic, choose the most appropriate first line imaging modality.

Q3.1. If the patient is found to have rebound tenderness of the abdomen.
Q3.2. If creatinine is 132, KUB is negative.
Q3.3. If creatinine is 237, KUB is negative.
Q3.4. If creatinine is 187, KUB is negative and patient is claustrophobic.
Q3.5. If there is a known radio-opaque stone previously treated conservatively.

Q4. Appropriate medical and surgical management in renal colic:

1. Expectant management on the ward with antibiotics and tamsulosin.
2. Conservative management as outpatient, repeat KUB in 2–3 weeks.
3. Conservative management as outpatient, repeat IVU in 2–3 weeks.
4. Conservative management as outpatient, repeat CT in 2–3 weeks.
5. Decompression with stent or nephrostomy.
6. Extracorporeal shock wave lithotripsy.
7. Ureteroscopic lasertripsy.
8. Laparoscopic or open removal.

For a patient who presents with symptoms of acute onset renal colic choose the most appropriate management in each of the following scenarios.

Q4.1. Initial IVU reveals a 13mm obstructing stone in distal ureter, pain is not controlled with analgesia and patient is not septic.

Q4.2. Initial IVU reveals a 12mm obstructing stone in distal ureter, pain is controlled with analgesia and the patient has become septic.

Q4.3. Initial IVU reveals a 5mm non-obstructing stone in the proximal ureter. Patient has been well and pain-free on the ward for the last 48 hours.

Q4.4. Initial IVU reveals a 9mm stone in the collecting system with consequent dilatation of the collecting system.

Q4.5. Initial IVU reveals a 4mm filling defect in the distal ureter, there is no evidence of obstruction, pain is controlled with analgesia and patient is pyrexial but not septic.

Answers

A1.1. = 7. Long-term catheters and diabetes are both risk factors for urinary tract infections. His hypotension and hypothermia are likely to be due to sepsis.
A1.2. = 5. These are all classical symptoms suggestive of pyelonephritis.
A1.3. = 6. Blunt sporting injury to the lower ribs may lead to kidney trauma.
A1.4. = 8. Classical symptoms of renal colic are severe loin to groin pain causing writhing, associated with nausea and vomiting.
A1.5. = 2. Although this could be a presentation of renal colic, the diagnosis of a leaking or dissecting aortic aneurysm must be urgently excluded with left and right brachial blood pressures and CT if indicated.

A2.1. = 7. Common causes of painless macroscopic haematuria include urinary tract malignancy or clotting disorder and so both need to be eliminated.
A2.2, = 3. Given this woman is of childbearing age and has become acutely unwell, a urine or serum beta-HCG is crucial to exclude rupture of ectopic pregnancy.
A2.3. = 2. A repeat urine dip on admission to the A&E department is mandatory in call cases of query renal colic. If there are any abnormalities on urine dip the sample should be sent to the laboratory for microscopy, culture and sensitivity (MC&S).
A2.4. = 8. The most likely diagnosis here is urinary retention and if the bladder scan confirms this he will need to be catheterised.
A2.5. = 4. These symptoms should alert you to the possibility of metastatic disease to the spine with spinal cord compression. In particular prostate cancer can metastasize to bone. A digital rectal exam is mandatory in this presentation. Anal tone and perianal sensation should be assessed to eliminate cauda equina syndrome. If the prostate is hard and irregular it suggests prostatic cancer. If there is suspicion of spinal cord compression urgent MRI is indicated, as decompression may be required.

A3.1. = 7. Suggests peritonitis, perforation is a strong differential and so urgent erect CXR and AXR are indicated to look for free air.
A3.2. = 4. Non-contrast CT is gold standard imaging for acute presentation of renal colic.
A3.3. = 4. As for Q3.2. the non-contrast CT is indicated. Impaired renal function is only a contraindication in contrast imaging (e.g. IVU or contrast CT).
A3.4. = 5. IVU is an acceptable alternative first line imaging modality when CT not available or appropriate provided creatinine is below 200. Indeed IVU is still used in preference to CT in some specialist urology centres.
A3.5. = 8. KUB is always the first line choice of imaging in renal colic as 60% of stones are radio-opaque. In this case we already know the stone is radio-opaque so a repeat KUB is essential to track the progress of the stone to plan subsequent management.

A4.1. = 7. This stone is too large for expectant management and as the patient is symptomatic removal via ureteroscopic lasertripsy is indicated.
A4.2. = 5. Urgent decompression with nephrostomy or stent is indicated to preserve the kidney function. Once decompressed and the infection has been treated with antibiotics, elective surgery to remove the stone can be arranged.
A4.3. = 2. This patient does not need to stay in hospital as there is a good chance the stone will pass spontaneously with conservative management. As the stone is radio-opaque and non-obstructing there is no need to expose the patient to higher doses of radiation on CT or IVU contrast when an outpatient KUB will suffice.
A4.4. = 6. Ureteroscopy to the collecting system may prove difficult. As this stone is radio-opaque it cannot be a cysteine stone and therefore this stone is suitable for fragmentation by ESWL.
A4.5. = 1. Clearly this patient cannot be sent home while pyrexial. However, expectant management with antibiotics and tamsulosin are likely to be effective given that the stone is distal and small.

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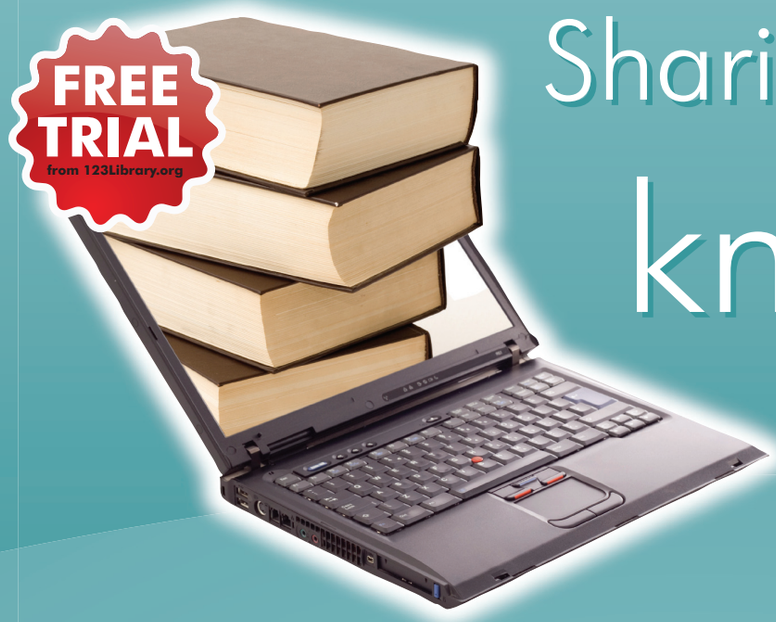

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