123 Doc EDUCATION

FOUNDATION YEARS JOURNAL

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Volume 6, Issue 1: Geriatrics

FOUNDATION YEARS JOURNAL 2012

Volume 6, Issue 1

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.

Volume 6, Issue 1: Geriatrics

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 and 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. Each issue provides comprehensive clinical cases for trainees as well as practical teaching assessments for educators. Readers will benefit from:

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Editorial For Geriatrics Issue Of Foundation Years Journal 2012

Many persons like to hold a book or journal in the hand. The ability to browse by turning pages, for those to annotate, who are prepared to deface paper copies to read without needing to find electronic apparatus to enable viewing (whether by computer, by Kindle device or otherwise), all are powerful stimuli to keep to conventional hard copy, paper publications. The feel of a book, the smell of the paper (maybe the binding), the colourful printing, and the variations in font and style all contribute to this sensual experience. However, paper copies become dated and cannot easily be amended except in loose-leaf form where they lose much of their aesthetic appeal. They are more expensive to produce at the point of the user. They decay with use, whether aided by fingers, thumbs or by mice, and they are bulky for publishers and readers to transport.

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Hence, this trends towards electronic publishing. Electronic journals have many advantages and can be accessed from computers worldwide. This journal offers all of these advantages and on this occasion brings to readers aspects of important neurological topics relevant to Foundation Years practitioners.

The neurosciences, of which, everyday clinical neurology forms a part, have made amazing progress over the last couple of decades. The interactions between laboratory and clinical research, and with clinical medicine that deals with illness in patients at its most elementary level, have contributed to these advances. However, sometimes research and cutting edge thinking from the laboratory is difficult to apply to some of the immediate clinical problems exhibited by patients. Common sense (whatever that is) and thinking is needed with acute problems and so is rapid decision making. Some of the topics covered in this issue deal with acute medicine, and neurology is now very much part of this since nearly one fifth of those admitted acutely have neurological problems, and others with less acute matters still get admitted to hospital. Papers published here express some of the most important points that Foundation Years doctors experience during their everyday duties, lessons they wish to share with others in order to help prevent mishaps.

Indeed, such practitioners are encouraged to submit to this journal. There is so much to be learned from our everyday activities and our patients are in many ways our best teachers, using their symptoms and signs to make us think. It is in many ways a moral imperative to share this information with others and to publish for the widest circulation. Specific lessons that may be drawn from the papers in this issue of the Foundation Years Journal, include epilepsy and the causes of blackouts together with some useful tips on the use of the EEG in diagnosis, an important supportive test in some patients. Stroke is now an emergency in more ways than previously (since more can be done), a brain attack that needs handling acutely and which can result from venous sinus thrombosis, two more areas covered in this journal. The techniques of lumbar puncture are still important although much that was investigated previously by this technique now is revealed by the increasingly complicated imaging processes that have become available.

Acute neuromuscular weakness is a further presenting feature that has many causes and this condition may be quite puzzling in many patients. Increasingly complicated drugs and drug regimes may lead to toxicity, an important cause of disability that can easily be overlooked; baclofen is a useful drug for spasticity and intoxication is disabling. Trigeminal neuralgia can be treated in many ways; not all being effective and a paper on this topic should help guide those who deal with its early manifestations.

And what of that imperative to publish? Here we are guided in the values of clinic letters and of the role of the doctor as educator. All very important stuff, hopefully interesting, certainly enlightening, and without doubt we hope a stimulus for readers to provide further papers dealing with the many topics in neurology that may perplex all of us including those working in the Foundation Years.

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Notice

PARKINSON'S DISEASE - CASE BASED DISCUSSION

E Richfield

**Parkinson's Disease -
Case based discussion.
Good Clinical Care.**

Case History

A 72 year old man attends a general medical clinic with his son. The father has been referred by his General Practitioner with "General Decline". As the Foundation Year 2 doctor you are asked by your consultant to assess him. You notice that it takes him several minutes to enter the consultation room, and whilst going to assist him you observe a slight shake in his right hand. He has a soft voice, and frequently dabs at the corner of his mouth with a tissue. During the course of the consultation it is apparent that his most troubling symptoms are falls, difficulty being heard and hyper-salivation which he finds embarrassing.

You comment on his tremor and his son mentions that they have been concerned about the possibility of Parkinson's disease.

What other areas do you need to explore in the history?

Nature of the tremor

Tremor is a common complaint in older people. It is important to establish key points such as whether the tremor is unilateral or bilateral, when it was first noticed and how it has progressed over time. The tremor in Parkinson's disease (PD) is classically unilateral, and although it may progress to both sides continues to be more marked in the original arm. Tremor affecting both arms equally favours alternative diagnosis such as Essential Tremor.

A "step wise" progression or history of recurrent stroke may suggest a vascular cause, while a strong family history (more than one affected relative) makes PD less likely. Onset below 40 years of age is unusual in Parkinson's disease.

Non-motor features

Although we associate PD with movement problems, emphasis should also be placed on the non-motor features of the disease. These may become clinically apparent before the motor features and in future could represent a means for early diagnosis.[1] These are often the most debilitating aspects of the disease and should be actively sought.

Neuro-psychiatric	Dementia / Delirium / Psychosis
	Hallucinations
	Obsessional / Repetitive behaviours
Sleep Disorders	REM behaviour Disorder
	Daytime somnolence
	Insomnia
Autonomic	Urine – Urgency / Frequency / Nocturia
	Orthostatic Hypotension
	Sexual dysfunction
Gastro-intestinal	Hypersalivation
	Constipation
	Nausea / Vomiting / Dysphagia
	Weight loss
Sensory	Anosmia
	Pain – multiple modalities

Table 1 – Non motor features of Parkinson's Disease
(Adapted from Birmingham movement disorders course handbook – chapter 8)[2]

Concerns and anxieties

Patients with tremor are often concerned about the possibility of PD. They are likely to have fears about the progressive nature of the disease, associated cognitive decline and loss of autonomy. Identifying these issues early allows fears to be addressed and where appropriate support to be initiated. Parkinson's Disease Nurse Specialists (PDNS) and charitable groups such as Parkinson's UK often provide valuable support for patient and care giver.

You note he has several of the non-motor features of Parkinson's disease, including hyper-salivation. As he goes to the examination room you make a mental check list of the features you wish to examine for and the technique involved.

As you watch him walk in to the examination room, what features are you looking for?

Patients may have a short stepping or shuffling gait with stooped posture, leaning forward and narrow base (feet close together). This contrasts with the gait in vascular parkinsonism which classically has a wide base and upright posture. Arm swing is often reduced, unilaterally at first, and he may have festination.

PARKINSON'S DISEASE – CASE BASED DISCUSSION

E Richfield

Parkinson's Disease - Case based discussion. Good Clinical Care.

Examination
There are four key clinical features on examination.

Bradykinesia
A key diagnostic feature and it must be present to diagnose PD. Patients have reduced speed and amplitude of voluntary movement, and may struggle to initiate movements (ignition failure).

Practical Test
Ask the patient to perform finger taps (tap the tips of thumb and finger together rapidly) and watch for a reduction in amplitude and speed. Imagine the fingers are stuck together with chewing gum - movement becomes more difficult, and the fingers get closer together as the gum becomes stickier! Older patients may struggle with this test. Ask them to tap the back of the hand or tap the heel of their foot on the floor.

Tremor
It is often useful to describe tremor as present during rest, posture or action. Classically PD produces a rest tremor, more prominent in one side of the body, and in the upper limbs. It may be present as postural and action tremor, but remains more pronounced at rest.

Practical Test
Watch the patient with hands relaxed in their lap (Rest tremor), ask them to hold their arms outstretched (Postural tremor), and then reach to pick up a pen (Action tremor).

Note that in Parkinson's "Re-emergent" tremor may be present – When posture is assumed the tremor is initially absent, but becomes increasingly evident as the posture is held.

Rigidity
Increased tone is present throughout movement and may be "cog wheeling" in nature.

Practical test
Test tone at wrist and elbow, rigidity may be enhanced by the patient performing movement with the contra-lateral side (tap their knee with their left hand while examining the right)

Postural instability
Difficulty maintaining an upright, balanced posture, may contribute significantly to falls risk.

Practical Test
The patient stands with feet apart while the doctor pulls sharply on the shoulders from behind. Be prepared to support them if the test is positive! Postural instability may have multiple causes, especially in older patients. Remember – A diagnosis of PD requires Bradykinesia with at least one of the other 3 characteristics

Bradykinesia and 1 of:
Rigidity
Rest Tremor (4-6 Hz)
Postural instability

Table 2 Positive clinical features of Parkinson's disease
Adapted from UK Brain Bank Criteria[3]

What else do you need to examine?
It is important to carry out a full general and neurological examination to elicit other underlying disorders. Unilateral upper motor neurone signs for example, may raise the possibility of stroke disease suggesting vascular Parkinsonism.

Check for postural hypotension which may represent autonomic dysfunction and be contributing to falls risk. Loss of up-gaze raises the possibility of Progressive Supranuclear Palsy (PSP)

Which important differential diagnosis should you consider?
In older people common differentials include Essential Tremor and Vascular Parkinsonism, while it is important to consider the rare but reversible Normal Pressure Hydrocephalus, and remember the so called "Parkinson's Plus" syndromes.

Investigations
There is no single diagnostic test for Parkinson's disease. MRI can be useful to identify cerebrovascular disease, while DAT scanning, which identifies reduced pre-synaptic dopamine activity, helps distinguish Idiopathic PD from conditions which do not affect the Substantia Nigra, such as Vascular Parkinsonism and Essential Tremor.

PARKINSON'S DISEASE – CASE BASED DISCUSSION

E Richfield

At the end of the clinic you feel fairly certain that the diagnosis is Parkinson's disease. What do you do next?

PD is a clinical diagnosis and in the early stages there may be diagnostic inaccuracy, especially among non-specialists who deal with the condition less frequently. NICE guidelines are clear in recommending referral to specialist services before initiation of treatment to aid diagnostic accuracy and avoid "Therapeutic Trials" of dopaminergic medication.[4]

Explore patient concerns and consider referral to PDNS for further support and explanation.

Don't forget to address non-motor issues which may be present and consider falls reduction strategies.

You have established a good rapport with the patient and although he understands that you are not a specialist would like your opinion on possible treatments.

What types of treatment are available for Parkinson's disease?

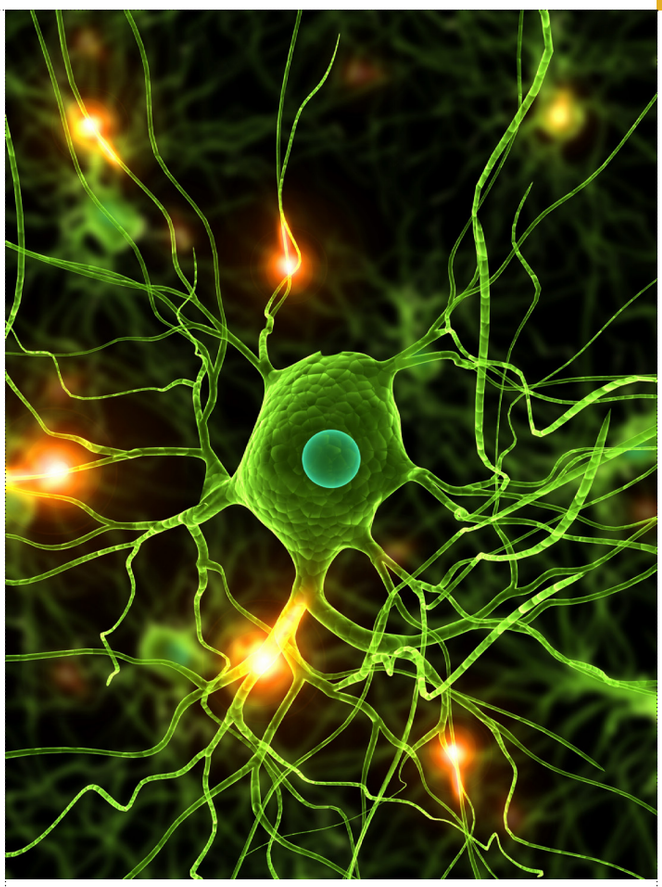
While as a Foundation Year doctor you should only initiate medications for Parkinson's disease in consultation with specialist colleagues, it is useful to be aware of treatment strategies.

Levodopa remains the mainstay of treatment, and comes in combination with a dopa-decarboxylase inhibitor to reduce peripheral breakdown. With time L-dopa may be associated with dyskinesias, reduced efficacy and development of "on – off" phenomena where patients experience rapid switch from clinically effective to ineffective drug levels. For this reason timing of L-Dopa initiation is important.

Pathways responsible for L-dopa metabolism can be targeted to maximise time spent in therapeutic range, reducing the peaks and troughs associated with intermittent dose administration. Examples include; Catechol-O-Methyl Transferase inhibitors (eg Entacapone) and Mono Amine Oxidase type B inhibitors (eg Rasagiline).

Dopamine agonists may be used to delay or supplement L-Dopa therapy and can reduce the associated dyskinesias. This serves to prolong the period of effective motor control.

Recent developments focus on potential neuro-protective effects for some drug therapies, and are likely to be an area of future advance. Select patients may be suitable for surgical treatments, including deep brain stimulation.



It is important to emphasise the non-medical management of PD, including physical and occupational therapy, speech and language services, and specialist palliative care applied throughout the disease course.

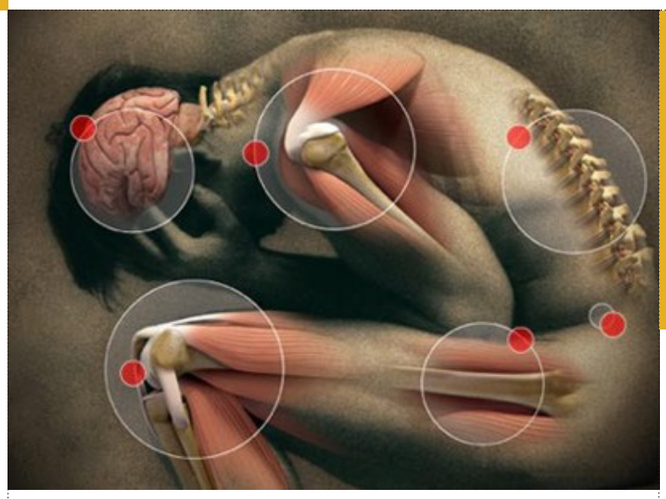
Over several years our patient has been established on L-dopa and a dopamine agonist with good response. He becomes unwell with an unrelated Community acquired pneumonia, causing severe sepsis. At admission he is too drowsy to take his medication.

What important steps must be taken with regards to his Parkinson's disease?

Missing Dopaminergic medications in PD can cause a syndrome similar to Neuroleptic Malignant Syndrome. Patients who are "off" may be more at risk of complications such as aspiration, and appear clinically worse than their underlying illness would suggest. Medication must not be omitted. Strategies for administration of medication in patients that are "nil by mouth" include Naso-Gastric tubes, transdermal Rotigotine patches and on occasion Sub-Cutaneous Apomorphine. These changes should be made in consultation with senior colleagues and ward pharmacist, and instigated as a matter of urgency.

PARKINSON'S DISEASE – CASE BASED DISCUSSION

E Richfield



Parkinson's Disease - Case based discussion. Good Clinical Care.

Self Assessment Questions

1. Which of the following is true of tremor in PD?

1 - Usually equal in both arms?
2 - Frequency 4-6 Hz?
3 - Increases with movement of the affected limb?
4 - Usually unilateral at presentation
5 - Commonly affects several family members?

2. The non-motor features of PD include:

1 - Excessive sleepiness
2 - Difficulty sleeping
3 - Autonomic features such as dry mouth
4 - Dyskinesias
5 - Urinary frequency

3. In the diagnosis of PD which of the following are true?

1 - L-dopa should be initiated as a therapeutic trial, to help distinguish patients with true PD?
2 - Neuro-imaging is a cornerstone of diagnosis?
3 - Tremor is essential to the diagnosis in accordance with Brain Bank criteria?
4 - Bradykinesia is essential to the diagnosis in accordance with Brain Bank criteria?
5 - Non-motor features usually emerge many years after the diagnosis?

Self Assessment Answers

1. Answer: 2 and 4 are correct.

The hand tremor in Parkinson's Disease is classically 4 – 6 Hz and described as "pill rolling". It is usually unilateral at onset, and although it may become bilateral with time, remains more marked in the original side. Tremor is one of the 4 cardinal features of PD but does not need to be present to make the diagnosis, unlike bradykinesia which is central to the UK Brain Bank diagnostic criteria.

2. Answer: 1, 2 and 5 are correct.

The non-motor features of PD are increasingly recognised and sleep disturbance, which may include both daytime somnolence and insomnia, is common. Autonomic disturbance including urinary disturbance and postural hypotension is a feature of the disease, but patients usually experience hyper-salivation rather than dry mouth. Dyskinesias are a motor feature, commonly as side effects from L-dopa therapy.

3. Answer: 4 is correct.

Clinical diagnosis of PD is difficult and best done in specialist clinics. Patients with suspected PD should be referred untreated to a physician with a special interest, and diagnostic therapeutic trials of L-dopa should be avoided. Neuro-imaging may assist in the diagnosis by helping to exclude specific differentials, but the diagnosis is based on clinical symptoms and signs. There remains no single diagnostic test for PD. As mentioned above Bradykinesia is central to the diagnosis of PD, while non-motor features are increasingly recognised pre-dating the diagnosis, sometimes by many years.

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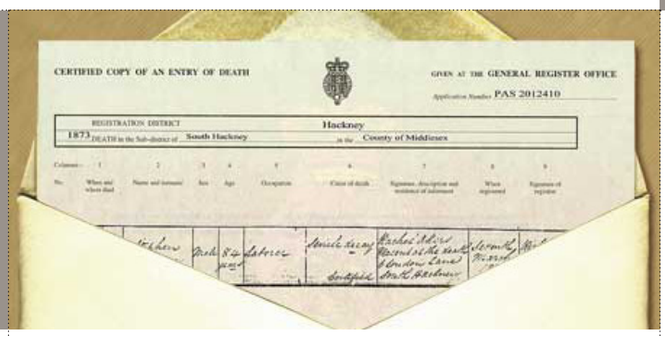
A GUIDE TO DEATH CERTIFICATION

A Illsley, A Cracknell, B Gill

A guide to Death Certification.
Professionalism In Practice.


CERTIFIED COPY OF AN ENTRY OF DEATH

GIVEN AT THE GENERAL REGISTER OFFICE

Application Number PAS 2012410

REGISTRATION DISTRICT Hackney

1873 DEATH in the Sub-district of South Hackney in the County of Middlesex

Abstract

Completion of the cause of death on the medical certificate is an important process frequently carried out by junior doctors and it is vital that correct processes are followed. This article looks at:

- **why accurate completion of the death certificate is important;**
- **a timeline of the history of death certification;**
- **the changes due to come into force in 2012;**
- **common errors made by doctors completing the certificate;**
- **how the information recorded on the death certificate is used.**

Why is death certification important?

Death certification serves a number of functions. A medical certificate of the cause of death enables the deceased's family to register the death. It provides a permanent legal record of the fact of death and enables the family to arrange disposal of the body and to settle the deceased's estate.

Information from death certificates is used to measure the relative contributions of different diseases to mortality which provides important statistical information regarding the health of the population. After registering the death, the family receives a certified copy of the register entry, which includes an exact copy of the cause of death. This provides them with an explanation of how and why their relative died. For all of these reasons it is extremely important that clear, accurate and complete information about the diseases or conditions causing a patient's death is recorded.

The General Medical Council has published guidance on end of life care which states that doctors "must be professional and compassionate when confirming and pronouncing death and must follow the law, and statutory codes of practice, governing completion of death and cremation certificates".[2]

If it is your responsibility to sign a death or cremation certificate, you should do so without unnecessary delay. If there is any information on the death certificate that those close to the patient may not know about, may not understand or may find distressing, you should explain it to them sensitively and answer their questions, taking account of the patient's wishes if they are known.

You must comply with the legal requirements where you work for reporting deaths to a coroner (England, Wales and Northern Ireland) or to a procurator fiscal (Scotland). You should be prepared to answer questions from those close to the patient about reporting procedures and post-mortems or to suggest other sources of information and advice. It is therefore vital that doctors completing the death certificate are familiar with the process requirements and seek senior support without hesitation if they are unsure.

Timeline of changes to death certification – 1953 to April 2012

1953

Requirements for recording the cause of death on the appropriate documentation to allow subsequent registration and release of the body for burial or cremation are officially outlined in the Births & Deaths Registration Act.

1998

The shocking discovery of the multiple murders committed by Dr Harold Shipman, a GP from Hyde, over a period of 24 years resulted in increased scrutiny of the British death certification process and coroners' system.

2003

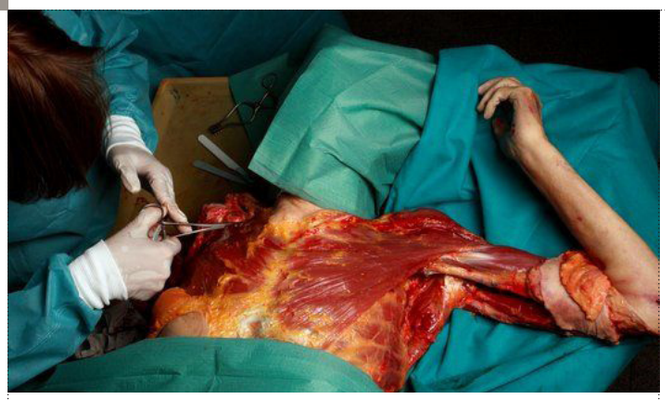
The Luce Review of Death Certification and Investigation in England, Wales and Northern Ireland: The Report of a Fundamental Review provided a damning report highlighting multiple failings of the traditional systems.

2005

The Shipman Inquiry, led by Dame Janet Smith, published its final report in a document entitled "Learning form tragedy, keeping patients safe". The Shipman Inquiry looked in detail at the death certification process and found that Harold Shipman had murdered at least 214 patients over 24 years without this being picked up through the current certification process. The inquiry recommended changes to protect patients, namely that there should be one system of certification for all deaths, whether burial or cremation is to take place, and that two forms should be completed, one by the doctor who confirmed death and the second by an independent doctor who scrutinises the case and the death certificate and who has been practising medicine for at least four years.

A GUIDE TO DEATH CERTIFICATION

A Illsley, A Cracknell, B Gill



2006
In response to the Luce Review and Shipman Inquiry, and prior to implementation of the suggested changes to the death certification process, the government announced plans to reform the Coroner Service in February 2006 and the Ministry of Justice published a draft Coroner Reform Bill.

2007
Two government consultation documents on related areas were published. The Ministry of Justice proposed creating a statutory duty on doctors and other public service workers to report particular types of deaths to the Coroner. The outcome of this consultation is reflected in legislation published by the Ministry of Justice – "Reform of the Coroners' System and Death Certification". The Department of Health also published a "Consultation on improving the process of death certification" as part of the government's response to the recommendations.

2009
As a result of these publications, primary legislation implementing the reform of the process of death certification in England and Wales is included in the Coroners and Justice Act.

2011 onwards
Pilots of the new death certification process are underway.

The government accepted the Shipman Inquiry's conclusion that the existing arrangements for death certification are confusing and inadequate. It recognised that improvements are required to make the process simpler for all concerned, to ensure effective medical scrutiny of all deaths certificates and to provide better information for local clinical governance purposes. It is important for doctors to stay up to date with the standards required in death certification as full implementation of the reforms is due to start in April 2012.

A step by step guide to completing the death certificate

The following information is a summary of the guidance from the Home Office for doctors completing the death certificate which every doctor should be familiar with. It is now the statutory duty of the doctor who attended in the patient's last illness to complete the death certificate. This statement is not strictly defined but is taken to mean a doctor who looked after the patient during the terminal illness and is familiar with the events leading to death and the patient's past medical history.

The certifying doctor should ensure they have access to the medical notes and investigation results. The consultant in charge of the patient's care takes overall responsibility for the information on the medical death certificate.[1]

The doctor completing the death certificate should provide legible personal details including time, date, name, grade, GMC registration number, and contact details in the patient's medical notes. This provides a permanent record of the doctor completing the death certificate in the notes for future reference. It is advisable for junior doctors to discuss cause of death with the responsible consultant. In fact many hospitals have specific local protocols for this to improve accuracy of recording information in the medical notes and on death certificates. In our Trust a proforma has been introduced that needs completing by the doctor issuing the death certificate and is subsequently filed in the medical notes (Figure 1 – published with the permission of Leeds Teaching Hospitals Trust).

When completing a death certificate the certifying doctor is required to state the cause of death to the best of their belief and knowledge. The death certificate is set out in two parts as per current World Health Organisation guidelines.6 Part I requires the certifying doctor to define the immediate cause of death followed by the sequence of events leading to death and, on the lowest line of part I, the underlying cause of death.[1]

Example:

Ia: Hepatic failure
Ib: Liver cirrhosis
Ic: Chronic hepatitis C infection

Therefore – chronic hepatitis C is the underlying cause of death, which lead to liver cirrhosis, which lead to the immediate cause (mechanism) of death which was hepatic failure.

The World Health Organisation defines the underlying cause of death as:

a. the disease or injury which initiated the train of morbid events leading directly to death or;

b. the circumstances of the accident or violence which produced the fatal injury.

A GUIDE TO DEATH CERTIFICATION

A Illsley, A Cracknell, B Gill

If more than one cause may have contributed to death you must include all causes in part I and then write "joint causes of death" in brackets. If you are waiting for results to confirm a suspected cause of death, the death certificate can still be completed, but the doctor must circle option 2 to indicate that information from a post mortem may be available later, as well as ticking box b on the back on the certificate, and initialling box b on the back to indicate that you may later be in a position to give additional information about the cause of death. If the cause of death is not known then you must refer the death to the Coroner.

Part II of the death certificate is to list all other diseases or conditions present at the time of death that contributed to, but did not directly cause, death. Mortality statistics are based on the underlying cause from Part I and the data collected from Part II is used for statistics on disease prevalence. You can include more conditions if needed by writing more than one cause on a line.[1]

Common pitfalls and specific causes of death

There are a number of common errors made by doctors completing death certificates, some of which are outlined here:

Avoid writing 'old age' alone as a cause of death.
Old age can only be used alone if a limited set of circumstances are completely satisfied:

- **You have personally cared for the deceased over a long period (years to months)**
- **You have observed a gradual decline in your patient's general health and functioning**
- **You are not aware of any identifiable disease or injury that contributed to the death**
- **You are certain that there is no reason that the death should be reported to the Coroner**

The lower age limit suggested in 1985 for death caused by old age alone was 70 years although current recommendations state that cases of a person dying aged under 80 with a cause of death stated as old age alone should be referred to the Coroner.

Avoid describing the mechanism of death not the cause of death.
Terms that do not identify a disease or pathological process are not acceptable as the only cause of death. This includes cardiac arrest, respiratory arrest, syncope or shock as they do not give information about the cause, merely the mechanism of dying. It is also important to note that abbreviations and symbols are not permitted on the death certificate and will result in it being rejected by the registrar of births and deaths, the only exceptions to this rule being HIV and AIDS.[1]

Never use natural causes alone.
The term "natural causes" alone, with no specification of any disease is not sufficient to allow the death to be registered without referral to the Coroner. If you do not know what disease caused death, it is up to the Coroner to decide what investigations may be needed.[1]

Avoid organ failure alone.
Do not certify death as due to the failure of any organ without specifying the disease or condition that led to the organ failure. The death will have to be referred to the Coroner if the disease responsible for organ failure is not specified.[1]

The Home Office guidelines for completion of the death certificate outline specific advice for the information required on the certificate when recording certain causes of death. A summary of this advice is outlined in table 1.[1]

Specific cause of death	Information to include on the death certificate
Stroke and cerebrovascular disorders	Nature and site of lesion Antecedent conditions (e.g. atrial fibrillation)
Neoplasms	Benign or malignant Histological type and site of lesion Specify whether metastases to or from primary lesion Clearly state if two primary cancers present If metastases with unknown primary state this clearly
Diabetes mellitus	State whether type I or type II If diabetes is direct cause of death state complication (e.g. ketoacidosis)
Notifiable diseases	Inform Health Protection Agency State manifestation or body site Antibiotic resistance patterns (e.g. MRSA) Route of infection (e.g. community acquired) Any conditions leading to reduced immunity
Healthcare acquired infections (HCAIs)	Reason for contracting HCAI and underlying condition being treated If HCAI present at death from another cause include in part II
Pneumonia	Organism Lobar or bronchopneumonia Hospital/community/ventilator acquired Conditions resulting in reduced immunity
Injury or poisoning	Must be reported to the Coroner Where injury occurred How injury occurred If fracture present state if pathological (e.g. osteoporotic)
Substance misuse	Deaths from diseases related to chronic alcohol or tobacco use need not be referred to the Coroner, provided the disease is clearly stated on the death certificate. All other deaths are reported to Coroner

Table 1 – Detailed information for specific causes of death[1]

A GUIDE TO DEATH CERTIFICATION

A Illsley, A Cracknell, B Gill

When to refer to the Coroner

In the current death certification process the doctor attending the patient in the last illness completes the death certificate and then the death can be registered by the Registrar of Births and Deaths, who will issue the death certificate. In the case of cremation, a cremation form is checked by a second doctor who has been qualified for at least five years.

It is however, worth noting that the new legislation will require all certificates, whether for burial or cremation, to be checked by a "medical examiner" attached to the relevant clinical governance team prior to issuing of the death certificate by the Registrar of Births and Deaths. In some circumstances a death is legally required to be reported to the coroner by the doctor completing the death certificate. A coroner must be a lawyer or a doctor, and in some cases are duly qualified. Each coroner has a deputy and assistant deputies, and one of these must be available at all times. Circumstances of death which must be reported to the Coroner are:

- **No doctor attended the deceased during his or her last illness**
- **Although a doctor attended during the last illness the deceased was not seen either within 14 days before death nor after death**
- **The cause of death appears to be unknown**
- **The death occurred during an operation or before recovery from the effects of an anaesthetic**
- **The death was due to an industrial accident, disease or poisoning**
- **The death was sudden or unexpected**
- **The death was unnatural**
- **The death was due to violence or neglect**
- **The death was in other suspicious circumstances**
- **The death occurred in prison or the person was detained in state custody**[9]

After a death has been referred to the Coroner there are three potential outcomes.

1. The Coroner may decide that a post-mortem examination and inquest are unnecessary because the cause of death is clear and there is a doctor who can sign the death certificate to that effect.[9]

2. The Coroner may ask a pathologist to examine the body and carry out a post-mortem examination. The Coroner is not required to obtain the consent of the relatives for a post-mortem examination to be made, but is required to inform certain persons of when and where the examination will take place. These include the deceased's relatives and others with an interest in the death. A coroner may not order an inquest after a post-mortem examination if there is no reason to suspect that the person died a violent or unnatural death, and they did not die in prison. The Coroner then releases the body for the funeral and sends a form to the Registrar of Births and Deaths stating the cause of death as disclosed by the post-mortem examination report, so that the death can then be registered.[9]

3. The Coroner may request a post-mortem, and then hold an inquest. Legally this must happen if the cause of death remains unknown, if the Coroner suspects that the deceased died a violent or unnatural death, or if the deceased died in prison. After the post-mortem examination is completed the Coroner may permit burial or cremation even if the inquest is not yet concluded. The death cannot be registered but an interim certificate of fact of death can be issued by the Coroner. This certificate should be acceptable to banks and financial institutions, unless it is important for them to know the outcome of the inquest (for example, for an insurance settlement). An inquest is a fact-finding inquiry to establish who has died, and how, when and where the death occurred. It does not have statements and examination of witnesses by prosecution and defence teams, like a criminal trial, the Coroner simply aims to find the answers to the above questions.[9] Some inquests are held in front of a jury, usually when the inquests is regarding a death in police custody, death due to industrial accident or poisoning, death after injury inflicted by a police officer, or death as the result of an event that may endanger public safety if repeated (e.g. a train crash).

A GUIDE TO DEATH CERTIFICATION

A Illsley, A Cracknell, B Gill

A guide to Death Certification.
Professionalism In Practice.

How is information from the death certificate used?

As well as the legal importance of accurately completing a death certificate it is important doctors are aware of how the information they provide on the death certificate is used for creating mortality and health statistics.

The Office of National Statistics collects data from death certificates and uses this to produce a variety of statistical information used to inform research, tailor and assess public health interventions and to monitor disease prevalence and patterns. For example, they produce weekly and yearly mortality statistics, yearly injury and poisoning mortality statistics, as well as cause of death trend data over the 20th century. The conditions listed on the medical certificate of death are coded using the World Health Organisation International Classification of Diseases and then this data is analysed.

The importance of accurate recording of disease is highlighted by the case of spinal and paraspinal abscesses. Under the International Classification of Diseases coding system used to collate data, spinal and paraspinal abscesses are recorded as tuberculosis unless the causative organism is recorded on the death certificate – this can lead to falsely high tuberculosis rates in the yearly mortality and disease prevalence statistics.[11]

The importance of getting it right

This article gives an overview of the current standards required for doctors completing the medical death certificate. The changes put in place after the recommendations of the Shipman Inquiry in 2005 have resulted in increased scrutiny of the information recorded on the death certificate and these changes are to be enforced by legislation in 2012. Therefore it is vital all doctors are aware of their legal duty when completing death certificates to record accurate information.

The information provided on the death certificate is not only important in meeting legal requirements but also provides vital information for the family of the deceased and for national statistics used to inform a wide variety of areas of healthcare research and public health planning.

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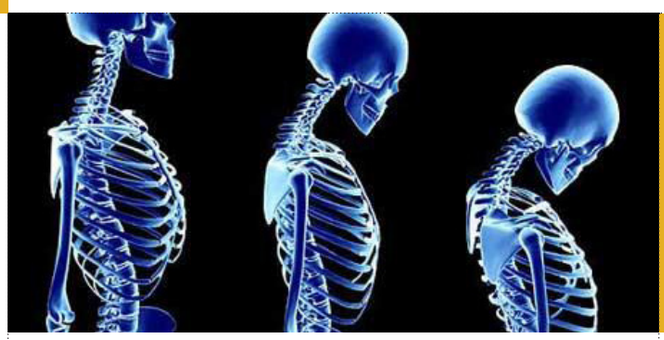
CASE BASED DISCUSSION: OSTEOPOROSIS, FRACTURES & BISPHOSPHONATES

I Gunawardena



Case Based Discussion: Osteoporosis, Fractures & Bisphosphonates. Good Clinical Care.

Abstract

Osteoporotic fractures are important because they are associated with an increased rate of death, substantial morbidity, enormous costs, and devastating effects on independence and quality of life. Therefore effective assessment of clinical risk factors and initiating appropriate treatment is essential. Bisphosphonates have proven efficacy in reducing the incidence of osteoporotic fractures and are used as the first line treatment in majority of the cases. However, several case series and multiple individual case reports suggest that some subtrochanteric hip fractures might occur in patients who have been on long-term bisphosphonate therapy.

Ms MA presented with bilateral, sequential bisphosphonate-related subtrochanteric fractures. Unique clinical and radiographic features surrounding these atypical fractures that have been illustrated in the literature are identified in this case.

Case Report

A 67 year old lady Ms MA, presented to hospital unable to weight bear with right sided hip pain, having sustained a spontaneous right sided subtrocanteric fracture of the femur. There was no history of preceding hip trauma, however she complained of right sided hip pain on walking for a period of four months prior to her presentation. She was a known patient with pemphigus vulgaris and was on long-term glucocorticoids, which she had been on for five years. She was started on a bisphosphonate (alendronate) and calcium with vitamin D two years prior to her presentation for osteoporosis related fracture prevention.

The fracture was treated with an intra-medullary gamma nail and she recovered from surgery successfully. A pathological fracture was suspected and she had a number of investigations. Blood tests including bone profile, vitamin D levels, thyroid function tests and a myeloma screen were normal. A CT scan of her hips showed a healing fracture of the right hip and nothing sinister was noted in the right or the left hip. A whole body bone scan showed increased uptake involving the right proximal femur and less intense uptake was noted involving a similar area on the proximal left femur. Histopathological examination of bone specimens was normal. She had a normal mammogram two years prior to presentation. Established, pathological causes for atypical femoral fractures including malignance, metabolic and endocrine causes were hence excluded.

The patient continued on the bisphosphonate and calcium with vitamin D, along with the long-term glucocorticoids. She unfortunately presented two years after the initial fracture, at age 69 years, with left sided hip pain. There was no preceding hip pain or a history of trauma. She had sustained a left sided subtrocanteric fracture of the femur, for which she successfully under went an intra-medullary gamma nail insertion. She was taken off her bisphosphonate and was discharged on calcium with vitamin D. A bone mineral density measurement with dual energy X-ray absorptiometry (DXA) six months before her second fracture showed that she was osteopaenic (with a T score of -2 at the hip) but not osteoporotic. Out-patient arrangements were made to repeat her DXA scan and to consider alternative treatment for osteoporosis, if appropriate.

X-rays of her right and left hips are shown in Figs 1 and 2 respectively. The transverse fracture pattern on the lateral half of the femoral cortex raises the suspicion of a possible pre-existing linear crack, which gave way and progressed into an oblique fracture on the medial cortex. Also it can be appreciated that there is significant cortical hypertrophy of the femoral shaft below the fractures. These atypical features are distinct from the typical spiral hip fractures seen in osteoporosis and are consistent with bisphosphonate related fractures. (1)

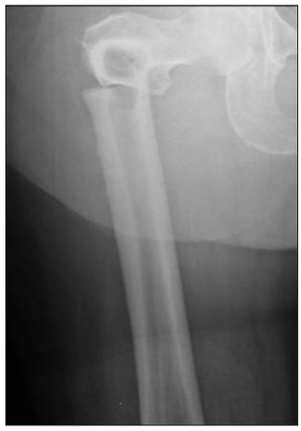


Fig 1

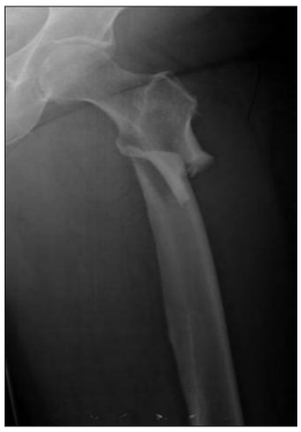
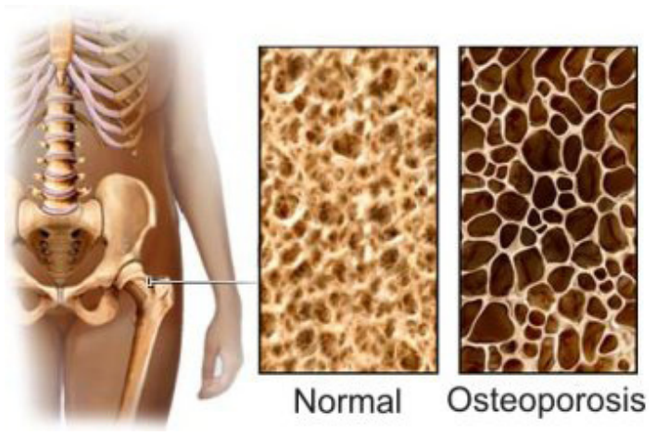


Fig 2

The transverse fracture pattern on the lateral half of the femoral cortex both in the right and left hips raises the suspicion of pre-existing linear crack involving the lateral half of the femoral cortex, which gave way and progressed into an oblique fracture on the medial cortex.

CASE BASED DISCUSSION: OSTEOPOROSIS, FRACTURES & BISPHOSPHONATES

I Gunawardena



Discussion

Osteoporosis & Risk Assessment

Osteoporosis results from reduced bone mass and disruption of the micro-architecture of bone, giving decreased bone strength and increased risk of fractures, particularly of the spine, hip, wrist, humerus, and pelvis. Fractures caused by osteoporosis affect one in two women and one in five men over the age of 50, resulting in an estimated annual cost to the health services of around £1.8bn in the United Kingdom.(2) The World Health Organization's definition of osteoporosis is based on bone mineral density in the spine and proximal femur measured with dual energy x-ray absorptiometry (DXA). Osteoporosis is classified as a bone mineral density 2.5 or more standard deviations below normal peak bone mass—that is, a T-score less than or equal to –2.5 SD.(3) Severe osteoporosis (established osteoporosis) describes osteoporosis in the presence of 1 or more fragility fractures.(4)

Apart from bone mineral density other factors that determine intervention thresholds include the presence of clinical risk factors and the cost and benefits of treatment. The ten year probability of a major osteoporotic fracture (clinical spine, hip, forearm or humerus) can be determined using the FRAX® tool (www.shef.ac.uk/FRAX).(5) Men and women with probabilities below the lower assessment threshold on the tool can be reassured. Those with probabilities between the lower and upper assessment threshold can be considered for testing of BMD using DXA (if not done already) and their fracture probability reassessed. Men and women with probabilities above the intervention threshold should be considered for treatment.

The risk of fractures increases steeply with age and most of those affected are over 75.(2) Age related bone loss starts in the fourth or fifth decade of life. It occurs as a result of increased bone breakdown by osteoclasts and decreased bone formation by osteoblasts.(6) Oral glucocorticoids, which are taken by about 1% of the population and 2.5% of those aged over 75, are a common cause of osteoporosis

Management of Osteoporosis

Treatment of osteoporosis includes assessment of the risk of falls and their prevention. Maintenance of mobility and correction of nutritional deficiencies, particularly of calcium, vitamin D and protein, should be advised. Intakes of at least 1,000 mg/day of calcium, 800 IU of vitamin D and of 1 g/kg body weight of protein can be recommended. The major pharmacological interventions are the bisphosphonates, strontium ranelate, denosumab, raloxifene and parathyroid hormone peptides (Teriparatide). All these interventions have been shown to reduce the risk of vertebral fracture when given with calcium and vitamin D supplements. Some have been shown to also reduce the risk of nonvertebral fractures. The low cost of generic alendronate, which has a broad spectrum of anti-fracture efficacy, makes this the first line treatment in the majority of cases. Alendronate is approved for the prevention and treatment of glucocorticoid-induced osteoporosis. Teriparatide and zoledronate (intravenous bisphosphonates) are approved for treatment of glucocorticoid-induced osteoporosis where there is an increased risk of fractures. Other approved pharmacological interventions for postmenopausal women include calcitonin, calcitriol, etidronate and hormone replacement therapy.(7)

Bisphosphonates & Subtrochanteric Femoral Fractures

Bisphosphonates, the major class of drugs used to treat osteoporosis decrease osteoclast-mediated bone resorption and bone turnover markers and increase bone mineral density. They have been shown to reduce the risk of osteoporotic fracture in numerous large clinical trials.(8, 9) Bisphosphonates have an excellent benefit-to-risk ratio for women with osteoporosis over 3 to 5 years or perhaps longer, but both efficacy and safety data beyond 5 years are limited. Several case series(10-14) and multiple individual case reports(11-14) suggest that some subtrochanteric fractures and those of the femoral shaft (usually not the distal shaft) might occur in patients who have been treated with long-term bisphosphonates.

Several unique clinical and radiographic features surrounding these atypical fractures have been identified in the literature. Major features suggestive of these atypical fractures include the lack of trauma or minimal trauma, fractures being located distal to the lesser trochanter and proximal to the supracondylar flare, transverse or short oblique configuration and noncomminuted, complete fractures extend through both cortices and may be associated with a medial spike and incomplete fractures involve only the lateral cortex.

Some of the minor features include prodromal symptoms such as dull or aching pain in the groin or thigh, bilateral symptoms and fractures (either simultaneous or sequential), delayed fracture healing, use of pharmaceutical agents such as bisphosphonates and glucocorticoids, localized periosteal reaction of the lateral cortex, generalized increase in cortical thickness of the diaphysis (15) and normal or low bone mass but no osteoporosis at the hip. (14)

CASE BASED DISCUSSION: OSTEOPOROSIS, FRACTURES & BISPHOSPHONATES

I Gunawardena

It is possible that these atypical fractures begin with a stress reaction and/or stress fracture. At this stage, prodromal pain might be present. The stress fracture cannot be repaired adequately and the fracture progresses spontaneously to a full complete femur fracture, under the influence of no more trauma than bearing the weight of the body on the bone. (16)

The putative mechanism of these fractures is largely unknown. Bisphosphonates have prolonged residence in the skeleton and are not metabolized. Osteoclasts are rendered functionally deficient and osteoclast apoptosis is increased. Increased secondary mineralization leads to changes in crystal size and loss of heterogeneity in tissue mineral density and porosity, increasing stiffness. (16) The long-term impact of potent reduction in bone remodelling on femoral bone, which is subject to the greatest forces in the body, is unknown. Whether there could be an influence of specific focal deposition of bisphosphonate and an inability to remodel at that specific site is speculated. It is also speculated that some patients have intrinsically vulnerable osteoclasts. (14)

It is possible that a heterogeneous form of osteoporosis with intermediate osteoclast dysfunction could effect a particularly susceptible population. There might also be a subset of patients who have reduced bone formation ability prior to antiresorptive therapy, which could potentially be a risk factor with these fractures. Finally, it is possible that medications that improve strength in the proximal femur could result in excess mechanical weakness of bone below this site and predispose to fractures occurring distal to the hip region. (16)

Conclusion

Osteoporotic fractures are associated with an increased rate of death, substantial morbidity, enormous costs, and devastating effects on independence and quality of life. Bisphosphonates have proven efficacy in reducing the incidence of osteoporotic fractures. The literature however suggests the existence of atypical fractures associated with long-term bisphosphonate therapy. The true incidence of these fractures is largely unknown. The literature suggests the increase in absolute risk with bisphosphonate use is around 5 cases per 10,000 patient years. (17)

There is no rationale to withhold bisphosphonate therapy from patients with osteoporosis, although continued use of bisphosphonate therapy beyond a treatment period of 3 to 5 years should be re-evaluated annually. Consideration should be given to stopping (at least temporarily) bisphosphonate therapy in those patients who are reassessed to be at low or low-moderate risk (no incident fractures, T-score > -2.0, and no other major risk factors. (16)

In patients who have been on long-term bisphosphonates, physicians should specifically solicit information about thigh and groin pain. Radiographic imaging, including bone scan and/or MRI, might be warranted in such patients to detect these atypical fractures at an early stage, when perhaps major surgical procedures might be avoided or at least performed on a less emergent basis. In patients who do have atypical fractures it is recommended to discontinue the bisphosphonate. Furthermore, in those patients who appear to have impaired fracture healing, there is some evidence that treatment with teriparatide might help accelerate the healing of the fracture. (14,15)

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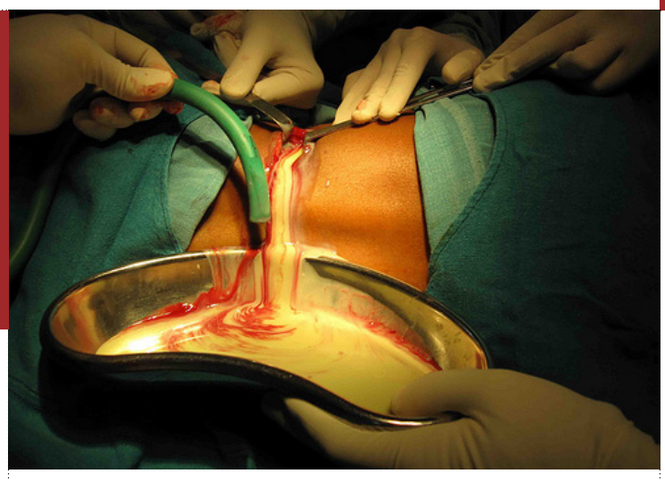
CARE BASED DISCUSSION: PSOAS ABSCESS SECONDARY TO OSTEOPOROTIC VERTEBRAL FRACTURE

B Abdulrahman, H Emery, M Vassallo

Cae Based Discussion: Psoas Abscess secondary to osteoporotic vertebral fracture. Patient Management.



Summary

Psoas abscess is a rare medical condition. It is most prevalent in young people and is rare in the elderly population.

An 84 year old lady was admitted with reduced conscious level from a nursing home. Her past medical history included dementia, schizophrenia, CREST syndrome and inflammatory erosive arthritis. On admission she was found to be pyrexial with crepitations on chest examination and left basal shadowing on chest x-ray. She was started on intravenous benzyl-penicillin and oral doxycycline for a left lower lobe community acquired pneumonia. Blood culture results showed a growth of Staphylococcus aureus.

Staph aureus pneumonia is rare and another source of infection was sought. A computerised tomography scan (CT) of the abdomen and pelvis showed a right psoas abscess secondary to osteoporotic vertebral fracture and discitis. We were unable to drain the abscess due to the patient's comorbidities. She was treated with a prolonged course of flucloxacillin which reduced the size of abscess. However the patient deteriorated and died.

Psoas abscess formation following vertebral collapse is very rare and highlights the need to consider multiple sources of infection in patients with atypical presentation.

Abstract

Psoas abscess is rare in elderly people and often presents with vague clinical findings. A high index of clinical suspicion is required for the diagnosis. This case emphasizes the importance of bacteriological confirmation of micro-organism in order to raise suspicion of disease and establishing the appropriate investigation and treatment early in the disease process. Staphylococcus aureus remains the commonest causative pathogen in abscess formation.(2) Successful treatment necessitates prolonged drainage and administration of a long course of the appropriate antibiotic(s).

Case report

An 84 year old lady was admitted with a one day history of reduced oral intake, being drowsy and withdrawn in her nursing home. Usually she was conversational although confused because of her background dementia. She had a fall a year prior to admission and sustained a fractured neck of left femur and had Dynamic Hip Screw fixation. Her other past medical history included CREST, seronegative arthritis and schizophrenia.

On admission she was uncommunicative with a GCS of 10/15 and a temperature of 38.1°C. She was clinically dehydrated and had inspiratory crackles in the left lung base. Her abdomen was soft with no tenderness in any quadrant. She had 5 x 5 cm sloughy surface ulcer on her left heel and 10 x 10 cm necrotic surface ulcer on her right lower leg. Full blood count showed a raised white cell count (16.3x109/L), ESR 79. Urine dipstick was negative. Chest x-ray showed left basal consolidation. Blood cultures and MRSA swabs were sent. On the diagnosis of community acquired pneumonia intravenous benzyl-penicillin and oral doxycycline treatment was commenced.

On day two of her admission, the blood culture grew gram positive Staphylococcus aureus in both bottles and benzyl-penicillin was changed to intravenous flucloxacillin. As the presentation did not fit staphylococcus pneumonia a secondary survey for the source of infection was carried out. Leg swabs showed normal skin flora. A transthoracic echocardiogram showed no valvular lesions and good left ventricular function. The abdominal ultra-sound showed a fluid collection, 10.5 x 4.6 x 3.3 cm in size, within the right psoas muscle consistent with abscess. A CT abdomen and pelvis scan was arranged.

This showed a multi-loculated ileopsoas abscess secondary to acute L3 fractured vertebrae, complete L1 collapse and extensive osteopenia. (Fig 1) On the basis of her poor pre-morbid condition drainage was not possible and intravenous antibiotic continued with regular monitoring of inflammatory markers.

After 14 days of intravenous antibiotics the white cell count normalized, ESR decreased from 79 to 58 and CRP from 60 to 23 and she was started on oral flucloxacillin on microbiologist's advice. She developed bilateral leg oedema up to mid-thigh and later her abdomen became distended with tenderness in right lower quadrant. The patient remained more confused than was normal for her.

After a further seven days of oral antibiotics the white cell count started to climb again to 14. The CT abdomen scan was repeated to examine the response to treatment. It showed a decrease in abscess size to 10 x 2.8 Apx. x 1.8 cm (Fig 2). Flucloxacillin was changed to long term intravenous and oral doxycycline regime as per microbiologist advice as there was poor control of the abscess with oral route. However the patient deteriorated, became less responsive and died on day 38 of admission.

CARE BASED DISCUSSION: PSOAS ABSCESS SECONDARY TO OSTEOPOROTIC VERTEBRAL FRACTURE

B Abdulrahman, H Emery, M Vassallo

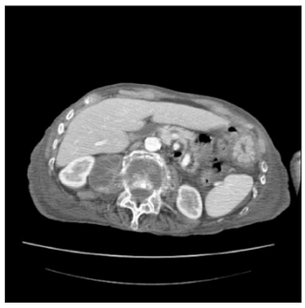


Figure 1: Initial CT scan showing the size of abscess and collapsed L3 vertebrae.

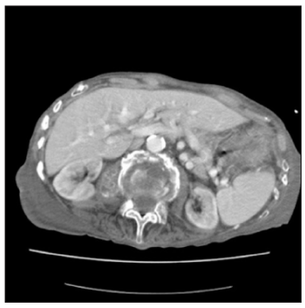


Figure 2: Second CT scan of abdomen showing the reduction in abscess size.

Discussion

Psoas abscess is a rare disorder especially in elderly population and is often difficult to diagnose on admission.(1) The psoas muscle extends from 12th thoracic rib and fifth lumbar vertebrae to lesser trochanter of the femur. It is anatomically related to colon, aorta, iliac lymph nodes, ureter, pancreas and spine. Contagious spreads of infection from these organs to psoas muscles can occur. Also its rich vasculature make the muscle prone to haematogenous spread of infections from remote occult infection.(2) This type of abscess is usually seen in patients with infective spondylitis, infections of sacroiliac joints and renal infections.(3) In our case it thought to be secondary to osteoporotic fracture of vertebrae caused some bleeding in to the muscle and haematoma formation and transient bacteremia from an unknown source led to the formation of an abscess in the ileopsoas muscle.

Common pathogens of primary psoas abscess include staphylococcus aureus in 80% of cases, as in our case, other pathogens include pseudomonas aerogenosa, proteus mirabilis and haemophilus aphrophilus, the usual cause of secondary psoas abscess, include Salmonella enteritidis, Enterobacter species, Streptococcus species and Escherichia coli. The most prevenelent cause in developing world is Mycobactrium tuberculosis.(1)

The abscess usually has insidious onset and may be presented as pyrexia of unknown origion,vague back pain, groin or abdominal pain.(4) Typically patients have lumbar lordosis and pain on hip flexion. An inguinal region mass should raise the suspicion of distal extension of the abscess. Proximity to the hip capsule can precipitate symptoms that mimic a septic hip. Co-existing septic hip occurs in 15% of population due to iliopectineal bursa spread of infection to joint.(4) In our case the only presentation was pyrexia, with swelling of the abdomen and tenderness of the right lower quadrant in the later stages of disease.

Early diagnosis often depends on clinical suspicion and timely acquisition of appropriate investigations most importantly imaging studies.(4) In this case, the high inflammatory markers and positive staphylococcus blood cultures with no clear source of infection that led to ultrasound examination which localized the psoas abscess. CT is the investigation of choice for diagnosis with 80-100% diagnostic value.

MRI has no greater role than CT in diagnosis of psoas abscess with the disadvantages of higher cost and more discomfort for the patient.(2) This case also suggests that repeated imaging studies should be considered when there is no improvement of clinical condition despite appropriate management.

The mainstay of treatment of abscess is prolonged drainage. The current gold standard of psoas abscess treatment is percutaneous CT-Guided drainage of abscess.(5) Our patient's poor co-morbid state made this intervention technically difficult with lots of potential complications if it were performed. It highlights the dilemmas physicians face when dealing with complicated cases in the elderly population.

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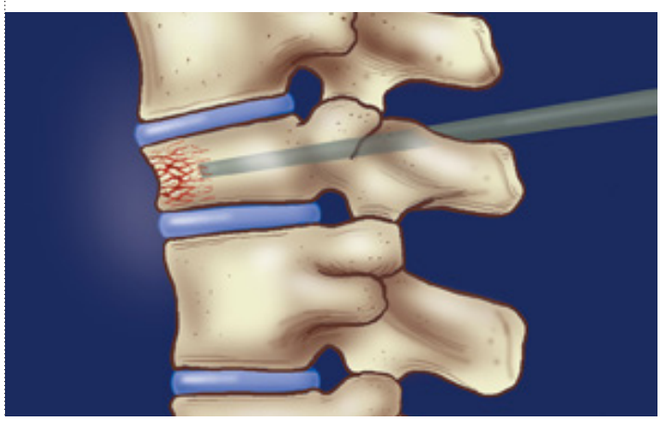
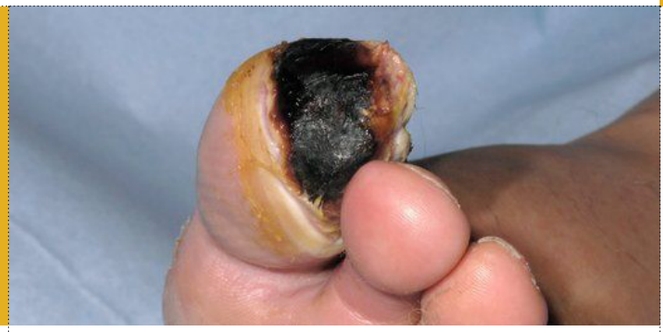


CASE BASED DISCUSSION: A MEDIASTINAL ABSCESS DEVELOPING SECONDARY TO A GANGRENOUS DIABETIC TOE

RJ Graham & M Vassallo

Case Based Discussion: A mediastinal abscess developing secondary to a gangrenous diabetic toe. Good Clinical Care.



Summary

Acute mediastinitis and abscess formation in the anterior mediastinum is rare and carries a high mortality if diagnosed late. A 79 year old gentleman presented with left sided shoulder pain. His past medical history included ischaemic heart disease, type 2 diabetes, and a recent admission for an infected gangrenous diabetic toe. He was found to have a 10cm mass in his left anterior chest wall on computed tomography (CT) imaging. Ultrasound confirmed this to be an abscess.

The patient deteriorated and died before the abscess could be drained. A post-mortem confirmed a Staphylococcus aureus anterior mediastinal abscess, and signs of sepsis likely to have developed secondary to haematogenous spread from the infected gangrenous toe.

The case highlights the high mortality of this condition and therefore the need for a high index of suspicion, as early diagnosis and treatment are essential.

Introduction

Acute mediastinitis is the result of bacterial infection of the mediastinum, most commonly caused by oesophageal perforation.(1) Abscess formation of the anterior mediastinum is unusual and generally occurs in the setting of acute mediastinitis.(2) Other less common causes include postoperative infection following medial sternotomy,(3) leakage from the oesophagus into the mediastinum through a necrotic neoplasm,(3) or extension of infection from adjacent anatomical regions.(4) A case of vertebral osteomyelitis leading to mediastinal abscess has been cited as an example of local spread.(5) We report a case of presumed haematogenous spread from an infected toe in a patient with non-insulin dependent diabetes.

The mortality rate for acute mediastinitis is high (50%).(4) Chest radiography may show mediastinal widening(6) and findings of mediastinal abscess including gas bubbles or an air/fluid level. CT is more sensitive than chest radiography for detecting the presence and extent of mediastinal fluid collections and the presence of extraluminal gas.(7) CT is helpful in assessing the relationships of fluid collections to adjacent thoracic or extrathoracic structures. It may also play a role in guiding procedures to drain the mediastinal fluid collections.

Case Report

A 79 year old Caucasian male with a history of ischaemic heart disease (requiring two bypass grafts) and diabetes presented with left-sided chest and shoulder pain, sweats, rigors and vomiting. It was noted that two weeks prior to admission he had an infected gangrenous toe, which had been treated with erythromycin for ten days. No cultures were available from this admission.

The differential diagnosis on admission included acute coronary syndrome, pulmonary embolus (PE), pneumonia and musculoskeletal pain. Troponin T was negative, D Dimer was markedly elevated and chest X-ray was normal. A PE was suspected and he was started on enoxaparin. However, a subsequent ventilation/perfusion (VQ) scan was negative. He subsequently developed a low-grade fever (37.5°) together with a raised white cell count (14 x10^9/L) and so was started on amoxicillin for a suspected pneumonia.

A chest X-ray at this time (10 days after this admission) showed hazy shadowing bibasally, but no sign of a widened mediastinum. His condition deteriorated and he developed renal failure, oedema and confusion. He was also noted to have hiccups. At this stage (14 days after admission) a chest X-ray showed a widened mediastinum and a CT chest (Fig. 1) showed a 10cm mass in his left anterior chest wall, extending anteriorly into the superficial tissues and posteriorly into the chest where it was noted to be in contact with the superior aspect of the anterior mediastinum.

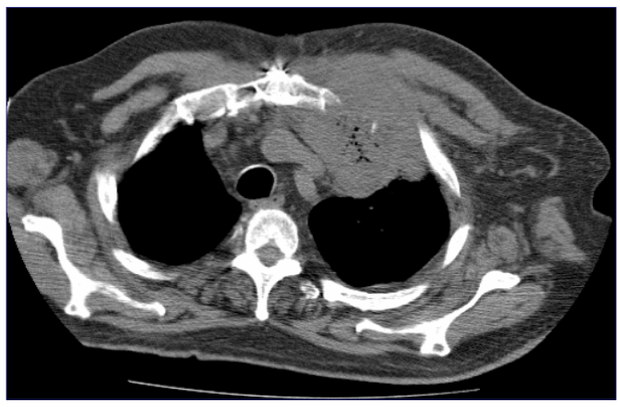


Figure 1

Computed tomography scan showing a 10cm mass in the superior mediastinum extending anteriorly into the subcutaneous tissues.

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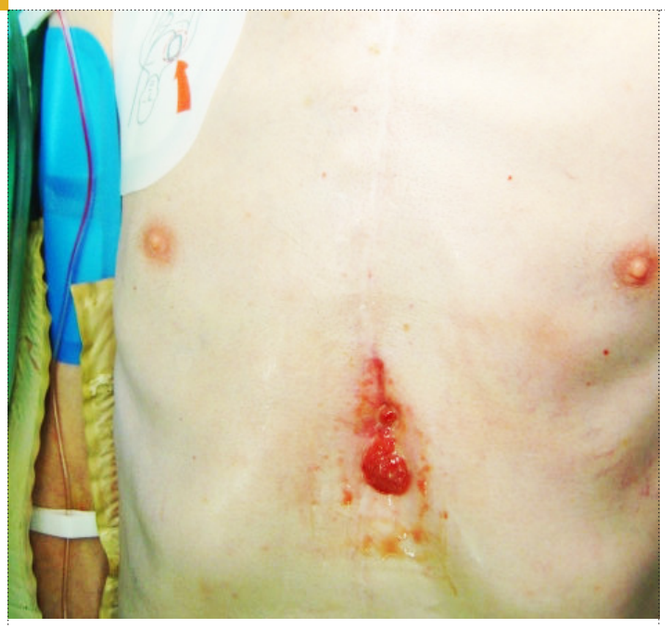

Due to his renal failure (creatinine 160 µmol/L), a contrast CT could not be performed. An ultrasound confirmed the mass to be a large abscess, but his condition deteriorated before this could be drained. Blood and sputum cultures prior to his death grew Staphylococcus aureus. Post mortem revealed a pus-filled abscess in the left deep soft tissue and subcutaneous fat in the subclavicular region and anterior mediastinum, extending up to the medial pleural surface of the left lung. Swabs from the abscess grew Staph. Aureus. No obvious bronchopneumonic consolidation was identified. Splenomegaly was consistent with sepsis, confirmed by swabs collected from the spleen, which also grew Staph. Aureus.

Discussion

Mediastinal abscesses commonly present with chest pain, fever, rigors and tachycardia. Hiccups are also not uncommon.(7) Diagnosis came only after CT and ultrasound imaging.

The management of the abscess is incision and drainage, which was planned for this patient.(8) Unfortunately the patient's condition worsened and he had a cardiac arrest. The source of sepsis was likely to be from the infected toe leading to septicaemia. Unfortunately no swabs were taken from the toe on his previous admission but would be likely to be caused by Staph aureus.

Diabetes is a known predisposing factor for sepsis and we suspect that the infection spread via the haematogenous route from his gangrenous toe and settled at the site of his previous sternotomy scar.

Haematogenous spread has been reported previously in a case of septic arthritis,(9) but to our knowledge has not been reported secondary to infected gangrene. This case also highlights the high mortality of this condition and therefore the need for a high index of suspicion, as early diagnosis and treatment are essential.

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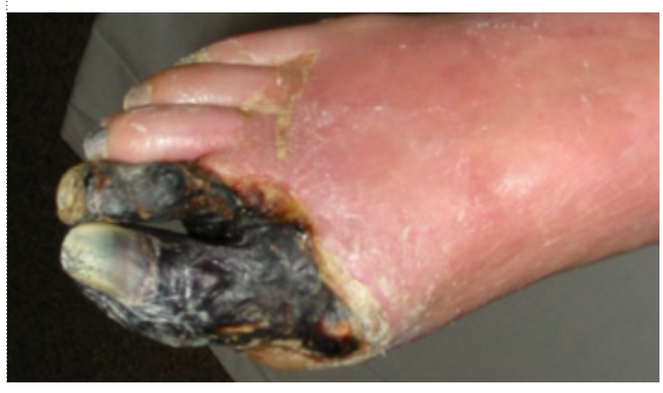
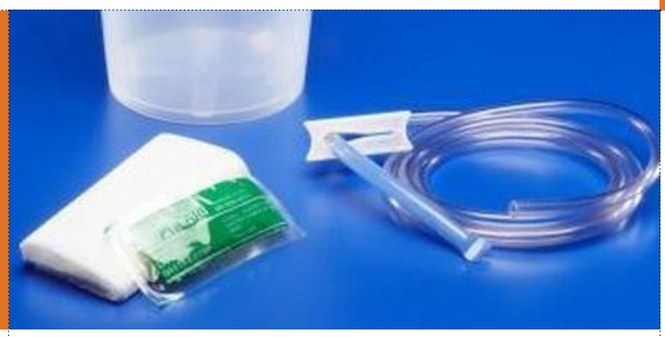


RIGID SIGMOIDOSCOPY AND FLATUS TUBE INSERTION FOR SIGMOID VOLVULUS

S Harrison, H Benziger, D Brown

Rigid Sigmoidoscopy and Flatus Tube Insertion for Sigmoid Volvulus
Sigmoid Volvulus.



Introduction

Sigmoid volvulus is a common cause of large bowel obstruction in the elderly and in many cases it can be recurrent. If promptly diagnosed, it can be safely treated by performing a rigid sigmoidoscopy and then inserting a flatus tube. Very often, this would provide almost instant relief for the patient.

After reading this article, the reader would have a good understanding of how to perform a rigid sigmoidoscopy and insert a flatus tube. They would also have an understanding of the various indications and contra-indications for this procedure.

The Case

Mr DW is a 76 year old gentleman who was referred to the general surgical department of his local hospital as he had developed marked abdominal distension with mild discomfort. Since his symptoms developed 5 hours ago, he has not passed any flatus and he also feels slightly nauseated. His past medical history of note includes ischaemic heart disease, chronic constipation and a stroke five years ago which has left him with very poor mobility. He also reports having multiple episodes similar to the present one in the past. Currently, he is on regular Aspirin, Perindopril, Frusemide, Atorvastatin and Movicol.

On examination he is found to be haemodynamically stable and apyrexial. Cardiorespiratory examination was unremarkable but examination of his abdomen revealed a visibly distended abdomen which was tympanitic on percussion with mild diffuse tenderness. Bowel sounds were minimal, there was no palpable organomegaly and a digital rectal examination revealed an empty rectum.

Given the findings of the physical examination and his previous history a tentative diagnosis of recurrent sigmoid volvulus was made which was then subsequently confirmed on x-ray (figure 1). Blood tests revealed no significant abnormalities. Arrangements were made for a rigid sigmoidoscopy and flatus tube insertion to be performed.

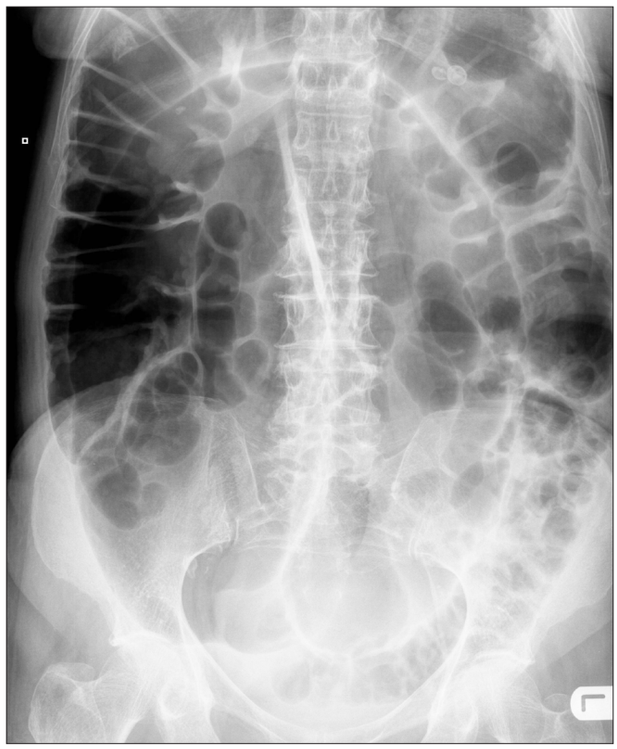


Figure 1: An abdominal radiograph demonstrating the classical "closed loop obstruction" appearance of sigmoid volvulus. The large gas-filled structure arising from the pelvis corresponds to a grossly distended loop of sigmoid colon. Compression of the medial walls of the loop against each other gives rise to the dense (white) linear vertical stripe centrally, known as the "Coffee Bean" sign. Also note the absence of gas in the rectum and the gaseously distended colon above (proximal to) the obstruction. This example is the classical appearance of sigmoid volvulus, but note the condition will often present with less typical features on a plain x-ray.

RIGID SIGMOIDOSCOPY AND FLATUS TUBE INSERTION FOR SIGMOID VOLVULUS

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Preparation

Adequate preparation for performing a rigid sigmoidoscopy and subsequent flatus tube insertion requires familiarity with the necessary equipment and proper positioning of the patient. This is in addition to the protective measures that the person doing the procedure should undertake. These factors ensure that the procedure is carried out in the most efficient and safest way possible.

It is important that the procedure is explained to the patient beforehand and informed consent is obtained. The patient's dignity must be ensured at all times.

Personal Protection

The person performing the procedure should ensure that they take adequate precautions to avoid contact with faecal material from the patient. Gloves, an apron and a mask preferably with a protective visor should be worn at all times (figure 2).

Figure 2: Adequate protective apparels should be used as the resolution of a sigmoid volvulus is usually accompanied with the passage of large amounts of loose stool and flatus.

The Equipment

A functional rigid sigmoidoscope consists of a scope, stylet, obturator, an eye piece, an air filter, bellows and a light source (figure 3). The bellows and eye piece attach to the back of the obturator along with the light source. These in turn attach onto the scope once the stylet has been removed. Prior to actually performing the procedure it is important to ensure that all the necessary equipment is available and that they all fit together properly.

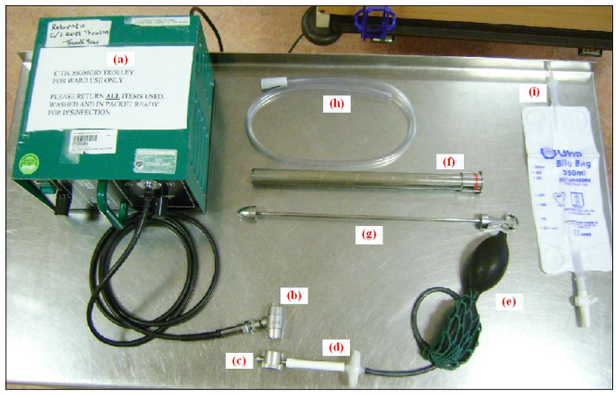


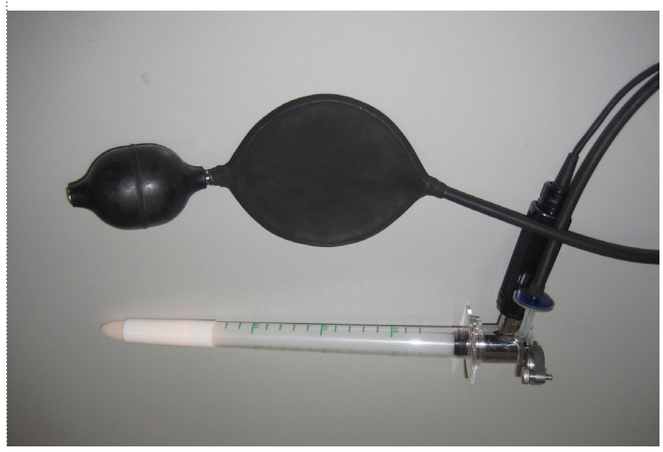
Figure 3: light source (a), obturator (b), eye piece (c), air filter (d), bellows (e), scope (f), sylet (g), flatus tube (h), collection bag (i).

Patient Positioning

The patient should be lying on their side (the lateral decubitus position), preferably the left side dependent and an attempt should be made to draw the knees up as close as possible to the chest. (1) The buttocks of the patient should be as close to the edge of the bed as possible as this would aid visualisation of the lumen and manoeuvrability of the scope. Ideal positioning is often difficult to achieve due to patient discomfort or safety and the operator should be prepared to find a position satisfactory to both himself and the patient.

The Procedure

- The procedure is best performed in the treatment room on a ward as it ensures adequate privacy for the patient. An assistant may be required to help the patient maintain their position.
- It is important to perform a digital rectal examination prior to the procedure to ensure the rectum is not faecally loaded. The peri anal area should also be inspected. If the rectum is found to be faecally loaded it would be necessary to administer a suppository or enema and perform the procedure at a later time.
- Once the patient has been adequately positioned and it is ensured that the proper equipment is available, the stylet is inserted into the scope and the end of the scope is lubricated with a water based lubricant (figure 4).



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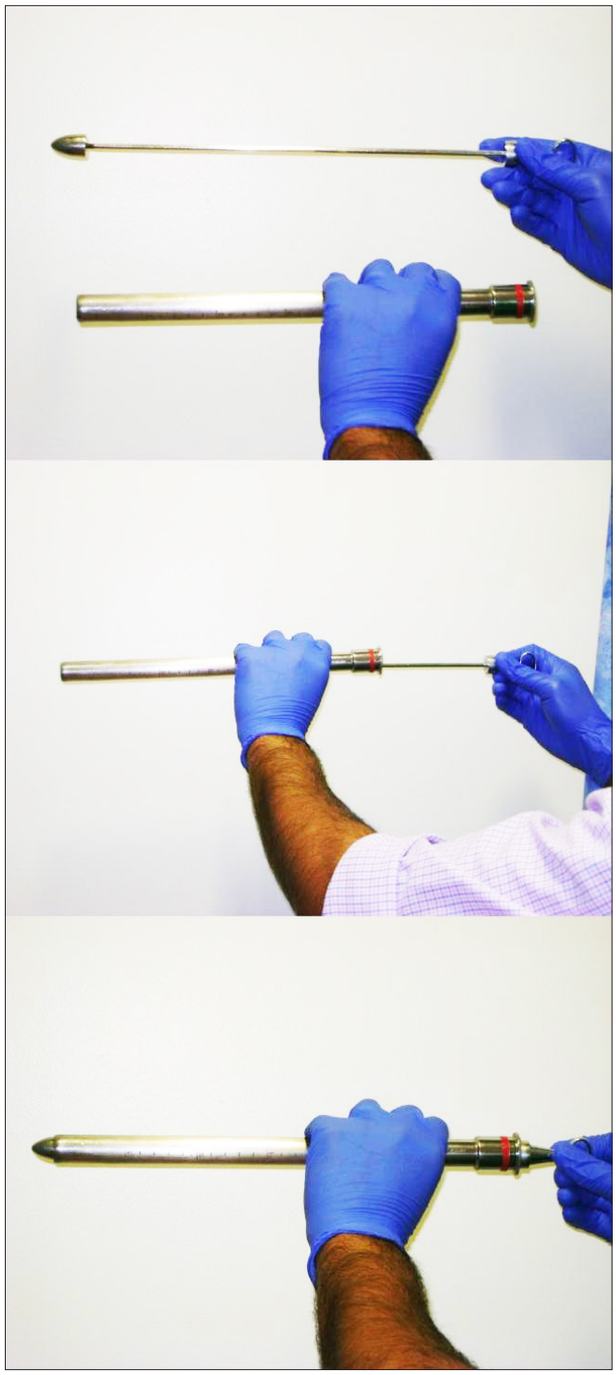

Figure 4: The stylet is inserted into the scope as illustrated in the above photographs. Once fully inserted, the end of the scope and stylet is lubricated with a water based lubricant.

- The scope along with the sylet is inserted through the anal opening with the scope being angled to point in the direction of the umbilicus (figure 5).

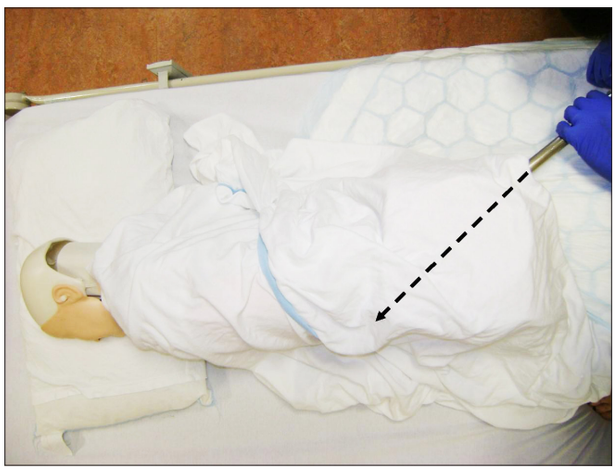

Figure 5: The scope with the stylet is inserted into the anal opening in such a way that the tip of the scope is directed towards the umbilicus.

- 'The stylet is then removed with the scope held in position (Figure 6). The obturator with the eye piece, light source and bellows is then attached to the back of the scope (figure 7).

Figure 6: Once the scope with the stylet is passed the anal opening, the scope is held in place and the stylet removed.

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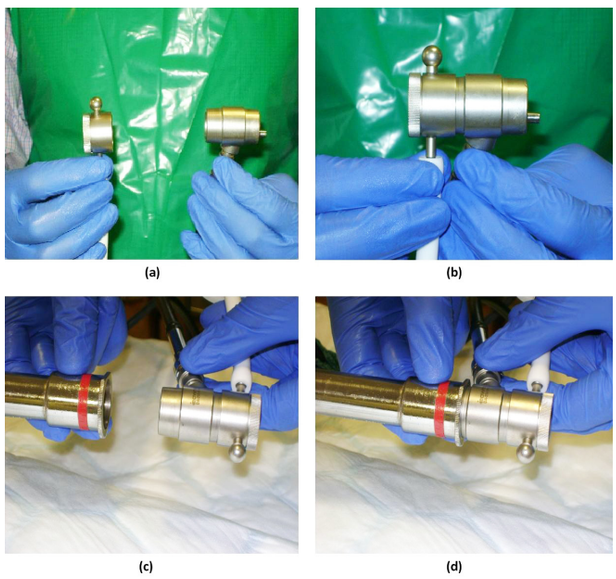

Figure 7: The eye piece is attached to the obturator along with the bellows and air filter. This is connected to the back of the scope as indicated in the above photographs.

- The bellows is used to insufflate the rectum with air which would make visualising the lumen much easier. The angulation of the rectum would mean that the inclination of the scope would need to changed by bringing it closer to the patient's legs. The scope is advanced only as far as the lumen is visualised (figure 8). This is usually between 15cm and 20cm.

Figure 8: Advancement of the scope is only done once the lumen of the bowel has been clearly visualised. For this to be possible it is necessary to change the direction of the scope to take into account the angulation of the rectum. This involves bringing the end of the scope closer to the patient's legs.

- Advancement of the scope causes the sigmoid colon to "de-volve". This is easily recognised by the sudden gush of bowel gas through the scope when the eye piece is removed. This can also be accompanied by copious amounts of loose stool. The patient experiences immediate relief and there is resolution of the abdominal distension.

- The flatus tube is inserted through the lumen of the scope (figure 9).

Figure 9: Once the scope has been advanced as far as visualisation of the lumen and patient comfort allow, the obturator with the attached eye-piece is removed from the back of the scope and the flatus tube inserted through the scope.

- The scope is then removed holding the flatus tube in position. A collection bag should be attached to the end of the flatus tube to prevent any faecal soiling of the patient's clothes or bed (figure 10).

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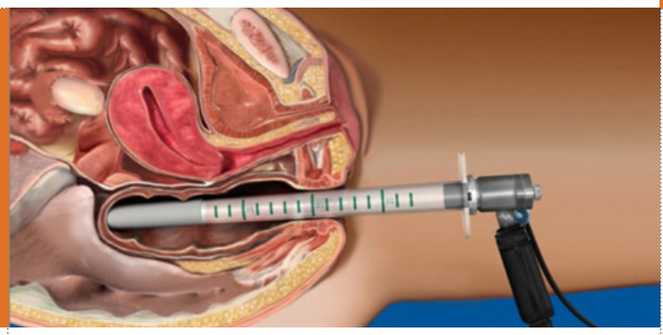

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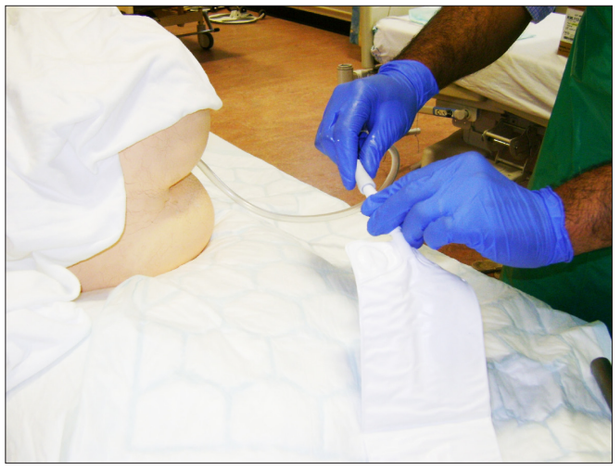

Figure 10: The flatus tube is held in position and the scope removed over it. A collection bag is attached to the end of the flatus tube.

· Once the patient has been cleaned the procedure should be clearly documented in the patient's notes. The flatus tube is usually left in situ for 24 hours.

· Soiled equipment should be washed and then sent off for sterilisation unless disposable instruments were used. Non re-usable equipment should be disposed of safely.

In the context of a sigmoid volvulus, a rigid sigmoidoscopy is contra indicated if a bowel perforation is suspected or if the patient is peritonitic.(2) Definitive treatment for recurrent sigmoid volvulus would involve surgical intervention, however this may not be appropriate if the patient's general health is poor. (3) Also, termination of the procedure is indicated if at any time during the procedure the patient experiences pain.

Rigid sigmoidoscopy can also be used to take biopsies of ano-rectal lesions which are within the reach of the scope. Complications such as perforation are rare when used for the purpose of treating a sigmoid volvulus, but can occur when biopsies are taken.(4)

Test Yourself

1. In the case history presented above, which of the following are risk factors for developing a sigmoid volvulus?

a. Ischaemic heart disease
b. Chronic constipation
c. Age
d. Use of Atorvastatin
e. Poor mobility

2. With regards to rigid sigmoidoscopy, which of the following are correct?

a. The scope should be angled posteriorly first and after advancing a few centimetres, the angulation should be directed anteriorly.
b. The scope should be angled anteriorly first and after advancing a few centimetres, the angulation should be directed posteriorly.
c. A patient with known recurrent sigmoid volvulus and presenting with gross abdominal distension and peritonism requires urgent decompression with a rigid sigmoidoscope.
d. Rigid sigmoidoscopy can be used for the conservative management of a caecal volvulus.
e. A flatus tube should be left in for 5-7 days.

Answers

Answer 1: Chronic constipation is known to predispose to the occurrence of a sigmoid volvulus. The presence of a redundant sigmoid colon is particularly susceptible to volving especially if it is faecally loaded. Although sigmoid volvulus is known to occur in elderly patients, rather than the age per se, it is the predisposing factors such as chronic constipation or neuro-psychiatric diseases that cause the volvulus.

Patients with poor mobility are at risk of developing a sigmoid volvulus only if the cause of the poor mobility (eg neurological disorders) has an effect on bowel motility. Ischaemic heart disease and the use of statins are not associated with sigmoid volvulus.

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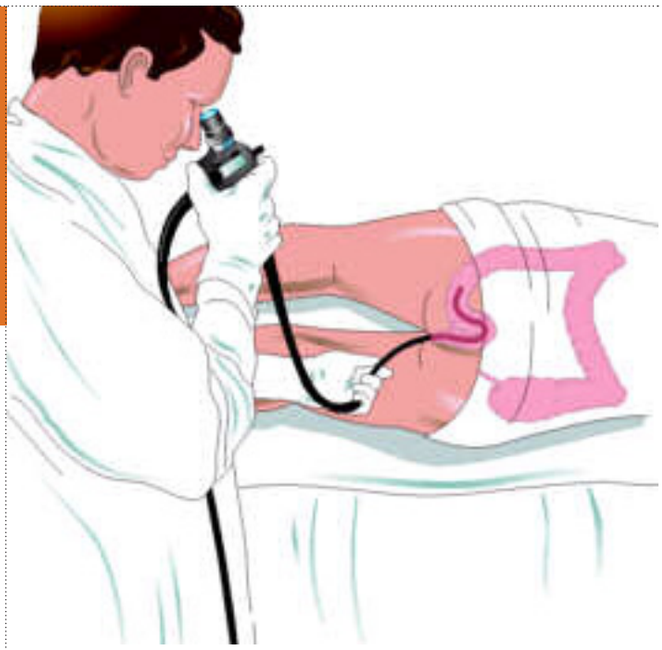

Answer 2: A patient diagnosed to have a sigmoid volvulus can be treated conservatively in most cases with a rigid sigmoidoscopy and flatus tube insertion. This is only contra-indicated if the patient is peritonitic in which case, depending on the fitness of the patient, operative intervention would be appropriate.

Due to the anatomy of the rectum, the scope is inserted with the tip pointing towards the umbilicus and after inserting a few centimetres, it is angled posteriorly before advancing further. A flatus tube should be inserted at the same time and is usually left in situ for 24 hours before being removed.

A rigid sigmoidoscope does not reach beyond 20-25 cm and therefore has no role in the treatment of caecal volvulus.

Acknowledgements

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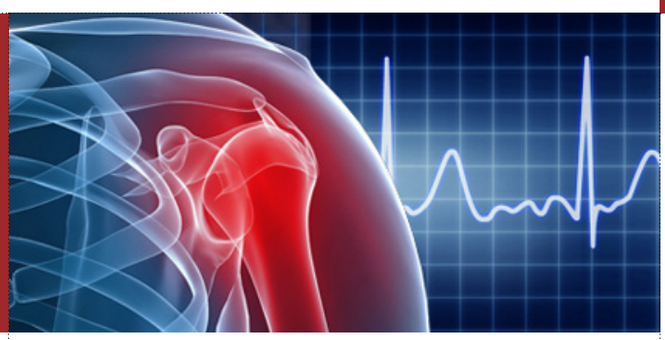
CASE BASED DISCUSSION: POLYMYALGIA RHEUMATICA

A Wheldon



Case Based Discussion: Polymyalgia Rheumatica. Patient Management.

Abstract

This article looks at the presentation, investigation and appropriate management of Polymyalgia Rheumatica (PMR). This is the most common inflammatory rheumatic condition in the elderly. There is a wide differential diagnosis. Investigations to help make the diagnosis and regular assessment of the clinical course must be made to ensure successful treatment.

The aim of this article is to illustrate, through the use of a case, the important aspects of the history, examination, investigation and treatments for a patient presenting with PMR.

Case Study

In the emergency outpatient clinic, you are asked to see an 82 year old lady who presents with a 3 week history of bilateral shoulder and hip pain and a feeling of stiffness, worse in the mornings for the past 6 weeks. This usually lasts for at least 2 hours every morning. She had been feeling tired and thought that she may have lost some weight over the preceding few months. She has felt intermittently feverish. She denies any visual disturbance. She has not had any joint swelling. Her past medical history includes a previous myocardial infarction and hypercholesterolaemia. Her medications are: Aspirin 75mg od, bisoprolol 2.5mg od, and simvastatin 40mg od. Her medications have not changed for 6 years.

On examination she had no scalp tenderness on palpation. She was unable to raise her arms above her head due to pain and stiffness in the shoulders. There was no photophobia or neck stiffness. The visual fields were normal. The cardiovascular, respiratory and abdominal examinations were all normal.

What is the differential diagnosis?

The differential diagnosis is broad. Given the symmetrical nature of the symptoms and constitutional symptoms, a new diagnosis of rheumatoid arthritis or other inflammatory rheumatic disease is a strong possibility. Polymyositis should be considered, although this would normally be associated with proximal muscle weakness. There may be an associated heliotrope rash on the eyelids and cheeks and Gottron's papules (red papules on extensor aspect of the fingers).

Bilateral subacromial impingement would certainly account for the shoulder pain and stiffness but should not cause the fevers and weight loss described. Hypothyroidism can present in a similar fashion with lethargy, myalgia, depression and an intolerance to cold. This would normally be associated with weight gain.

Given the constitutional symptoms of weight loss and fatigue, an underlying malignancy must be included in the differential diagnoses, and careful assessment through history, examination and investigation must be undertaken. Underlying infection is also a possibility given the fevers and fatigue for example a viral illness or tuberculosis.

The symptoms might be caused by drugs. Statins are well documented to cause both a myositis and a myalgia. However, the medications have not changed in 6 years and so it seems unlikely that they are now causing such problems.

Polymyalgia rheumatica (PMR) presents in patients over 50 years old and the symptoms often gradually worsen over several weeks. Classically, there is pelvic girdle or bilateral shoulder aching and pain. Both may be present. This is associated with morning stiffness for more than 45 minutes each day. There are usually raised inflammatory markers.

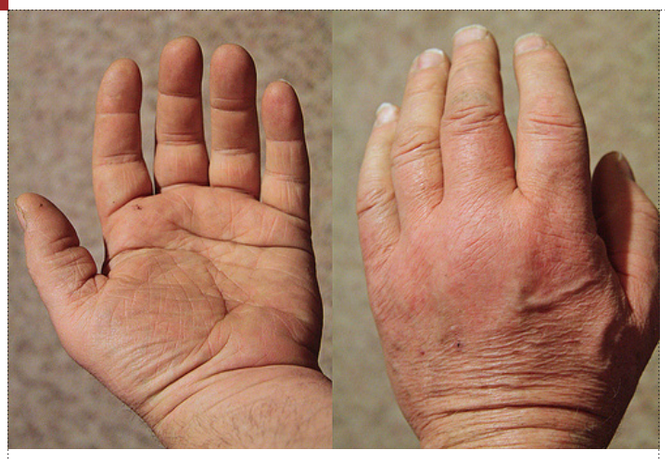
PMR may co-exist with Giant cell arteritis, (GCA) a vasculitis affecting the medium and large vessels, predominantly affecting the cranial branches from the arch of the aorta.(2) It usually affects patients in their seventies and is rare before aged 50. It is three times more common in females than in males.

GCA (also known as temporal arteritis) commonly has an abrupt onset of headache which is usually temporal and unilateral but may be more diffuse. There may be an ache in the jaw (jaw claudication) and tongue. The scalp is often painful to the touch and patients often describe discomfort while brushing their hair. Due to the diffuse inflammatory nature of the vasculitis, patients may describe feelings of fatigue, fever, weight loss and low mood.

People may present with a sudden onset of visual symptoms including blurring of vision and amaurosis fugax.(1) There can be sudden onset of blindness which is irreversible due to involvement of branches of the ophthalmic artery causing ischemia to the optic nerve. This is a devastating consequence of GCA and makes the diagnosis and management of the condition a medical emergency.

CASE BASED DISCUSSION: POLYMYALGIA RHEUMATICA

A Wheldon



Investigations

In this case, the history and clinical findings are suggestive of Polymyalgia Rheumatica. Initial investigations to help with diagnosis must include:

- Full blood count
- Urea and electrolytes
- Liver function tests
- Bone profile and Calcium
- CRP and ESR/plasma viscosity
- Protein electrophoresis and urinary Bence Jones Protein
- Thyroid Function Tests
- Creatine Kinase
- Rheumatoid Factor (and anti CCP) and ANA
- Urinalysis
- Chest x-ray

If there are any concerns regarding an underlying malignancy other useful tests may include abdominal ultrasound or CT depending on the initial presentation.

In this case the patient was found to have:
Hb 10.1, MCV 94, platelets 756, ALP 402, ALT 56, ESR 106 and CRP 88. All other blood tests were normal. The chest radiograph was reported as normal and the urine dipstick was negative.

Diagnosis

Careful assessment of the symptoms and signs and all investigations need to be taken into account before a diagnosis is made. The British Society for Rheumatology(1) state that the core inclusion criteria for the diagnosis of PMR are:

- **Age over 50 years, duration over 2 weeks**
- **Bilateral shoulder or pelvic girdle aching, or both**
- **Morning stiffness duration of over 45 minutes**
- **Evidence of an acute-phase response**

PMR can be seen with normal markers of inflammation. Anaemia due to chronic inflammation and a thrombocytosis is a common finding due to the systemic inflammatory response. The liver function tests will often have a raised ALP due to the acute phase response. On protein electrophoresis Ð1 and Ð2 globulins are sometimes raised.(2) The presence of infection or malignancy make the diagnosis less likely but it is important that the patient is reassessed once these have been treated. All patients must be assessed for the possibility of GCA as this is a medical emergency requiring urgent, high dose steroid administration.

Treatment

Treatment was commenced with a dose of 15mg of prednisolone daily. The patient was also given a proton pump inhibitor and bone protection (once weekly bisphosphonate and calcium supplementation).(3)

Discussion and Potential Pitfalls

Once a diagnosis of PMR is made it is recommended that a standard dose of Prednisolone 15mg is given and follow up arranged. Patients should be counselled regarding some of the possible side effects including weight gain, increases in blood sugars in people with concomitant diabetes, gastritis and peptic ulceration.

In the elderly, the risk of confusion, agitation and psychosis must be considered. Patients must be commenced on a proton pump inhibitor or H2 antagonist to help reduce the risk of peptic ulceration and as they are likely to require steroid for up to 2 years, bone protection with a bisphosphonate must be considered to avoid osteoporosis.(3)

The patient was followed up at her GP surgery 3 weeks after commencing treatment. Her shoulder pain and stiffness had almost completely resolved. The ESR and CRP had significantly reduced. A further follow up was arranged at 6 weeks post initiation of treatment.

If the diagnosis is correct, patients should have a dramatic improvement in their symptoms and have near normalisation of the ESR and CRP by 4 weeks. (1) It is advised that patients are followed up regularly to ensure continued response and to allow a gradual weaning of the steroids.

CASE BASED DISCUSSION: POLYMYALGIA RHEUMATICA

A Wheldon

Usually, 1 to 2 years of prednisolone is required before it can be stopped completely. Patients with a classical PMR picture are often managed entirely in the primary care setting. However, patients with unusual features, for example, onset in the young, prominent weight loss, normal inflammatory markers or other features suggestive of a possible underlying rheumatic condition should be referred for a rheumatology or elderly medicine opinion for further investigation.

Questions

1. Which is true? Polymyalgia Rheumatica:

a. Is a disease prevalent from aged 30 upwards
b. Often presents with a severe temporal headache
c. Presents with morning stiffness of >45 minutes duration
d. Has usually resolved within 2 weeks
e. Is effectively excluded with a normal ESR

Which is true? Polymyalgia Rheumatica:

a. Is a rare condition
b. Requires a dose of Prednisolone equivalent to 1mg/kg for optimal treatment
c. Treatment with a bisphosphonate is recommended
d. The markers of inflammation will remain elevated for several months even with treatment
e. 6 months of steroid will usually result in remission

Answers

Question 1:

a. False: PMR is unusual before the age of 50 years. Presentation before this should prompt the search for another cause for the symptoms.
b. False: PMR may well co-exist with Giant cell arteritis. Severe temporal headache may represent Giant Cell Arteritis, a medical emergency requiring prompt, high dose steroid.
c. True: Patients with PMR often complain of limb girdle stiffness present in the morning, lasting at least 45 minutes but often several hours.
d. False: Features of PMR are present for at least 2 weeks. A duration of longer than 2 weeks is one of the core inclusion criteria for the diagnosis of PMR.
e. False: The ESR and CRP are usually elevated due to the systemic inflammatory component of the disease. However, a normal ESR does not exclude the possibility of PMR as a diagnosis. It is suggested that such patients are referred to a rheumatologist for further investigation.

Question 2:

a. False: PMR is the most common inflammatory rheumatic condition seen in the elderly population
b. False: GCA requires high dose prednisolone to avoid severe consequences such as loss of vision and limb claudication. PMR should be treated with a standardized dose of 15mg Prednisolone once daily and the response to this assessed.
c. True: Prednisolone 15 mg once daily for a prolonged period is sufficient to induce osteoporosis. Bisphosphonates and consideration of a proton pump inbibitor to avoid gastric ulceration are recommended.
d. False: The inflammatory markers should improve significantly/normalise within a month of treatment. If the inflammatory markers remain high, other possible reasons for elevated inflammatory markers should be investigated.
e. False: The response to steroids should be assessed and a weaning process started if the patient is responding to the prednisolone. However, usually between 1 to 2 years of treatment is necessary before the steroid can be completely withdrawn.

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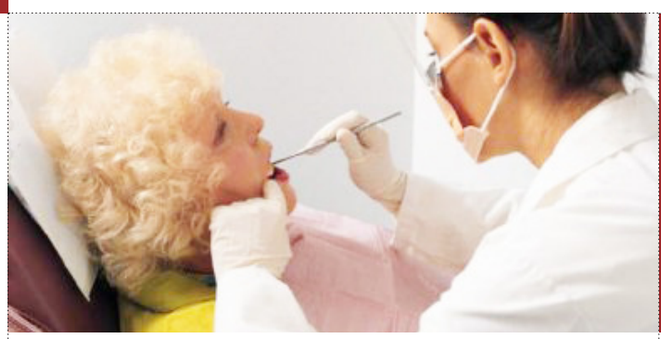
CASE-BASED DISCUSSION: INFECTIVE ENDOCARDITIS IN AN OLDER PERSON

S Boot, E Thomas, M Charlesworth, OJ Corrado



Case-Based Discussion: Infective Endocarditis in an Older Person
Patient Management.

Abstract

Infective endocarditis in the elderly population has significantly increased over the past few decades.(1,2) In older people it is often associated with another disease or problem, such as diabetes, immunosuppression, cancer, calcific aortic stenosis and alcoholism. Infective endocarditis is a disease that can be easily missed or misdiagnosed due to its initial presentation.

Symptoms are frequently subtle and nonspecific such as fatigue, fever and a flu-like illness. In this article, we describe a case of infective endocarditis in a male octogenarian with unusual presenting features. Our aim is to illustrate the difficulty in establishing a diagnosis of infective endocarditis and to describe the investigation and management of the condition as it is becoming more prevalent in the elderly population.

Case Study

An 82-year-old man was admitted to hospital having fallen at home. His son found him on the floor and the fall was not witnessed. He was pyrexial, acutely confused and unable to give a history. It was unclear how long he had BEEN lying on the floor and what had lead to his collapse.

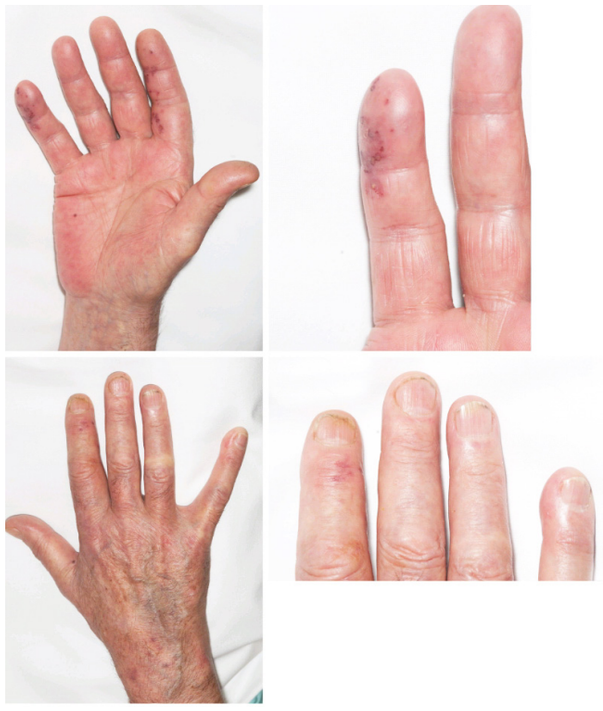
On examination he had a temperature of 38.4ºC, a respiratory rate of 22 per minute, an oxygen saturation of 96% breathing room air, blood pressure of 124/74 mmHg, a heart rate 78 per minute and an Abbreviated Mental Test Score of 0 out of 10. Heart sounds were normal with no murmurs detected. He had crackles at the left base. His abdomen was soft and non-tender with no masses or organomegaly and examination of the central and peripheral nervous system was normal.

Investigations showed a white cell count of 3.0 x 10^9, a neutrophil count of 2.72 x 10^9, a lymphocyte count of 0.19 x 10^9, a low platelet count of 87 x 10^9 and normal haemoglobin of 12.6 g/dL. Liver function tests were within normal parameters and his urea and electrolytes were sodium 129 mmol/L, potassium 3.9 mmol/L, urea 11.7 mmol/L, creatinine 122 mmol/dL and eGFR 53. C-reactive protein was also raised at 225 mg/L. Magnesium and phosphate were within normal limits. Creatnine kinase levels were raised at 403 iu/L. A plain chest radiograph showed consolidation at the left base. Blood cultures were taken and he was commenced on intravenous fluids and antibiotics for a community acquired pneumonia.

The patient remained pyrexial and clinically septic despite treatment. Blood cultures were positive for a Staphylococcus species, but this was not in keeping with the clinical picture and initial diagnosis of pneumonia. Further blood cultures were taken and his case was discussed with the Microbiology Department. We searched for other causes of sepsis and on advice changed the antimicrobial agents to intravenous vancomycin and oral rifampicin.

The patient had a prosthetic hip replacement 8 years earlier. He was tender to palpation over this area and also over his left elbow. A plain x-ray and a subsequent CT scan of the pelvis were organised to rule out osteomyelitis or a septic collection and he had an MRI scan of his elbow to look for any source of infection. He had no evidence of cellulitis.

The patient subsequently developed cutaneous lesions of his hands and toes.



CASE-BASED DISCUSSION: INFECTIVE ENDOCARDITIS IN AN OLDER PERSON

S Boot, E Thomas, M Charlesworth, OJ Corrado

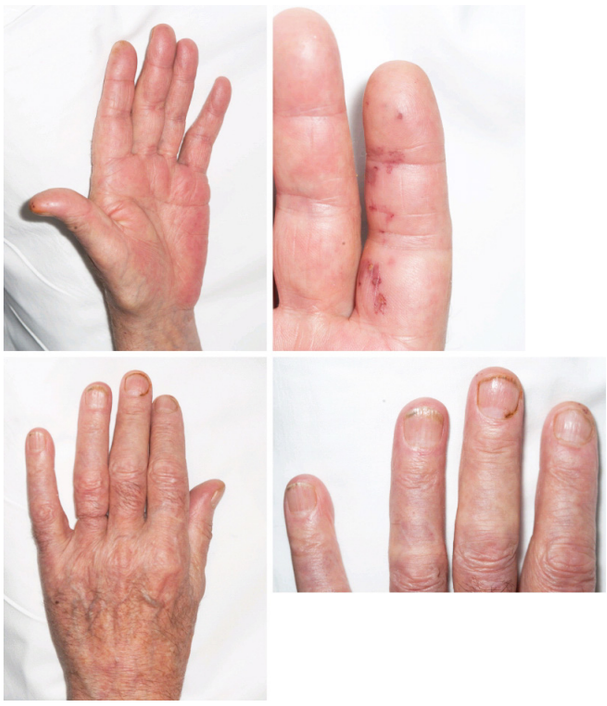


Figure 1. Cutaneous lesions on the patient's hands.

He developed a new soft systolic murmur. These findings raised the possibility of infective endocarditis as the cause of his infection. A trans-thoracic echocardiogram was arranged. He had bronchial breathing at the left base and bi-basal crackles. A repeat chest radiograph was requested to ensure the pneumonia was not progressing. The chest x-ray showed some resolution of the pneumonia and no other pathology.

The CT scan of the pelvis was unremarkable, with no evidence of prosthetic infection. The MRI scan of the left elbow demonstrated a large elbow joint effusion but no evidence of osteomyelitis. Senior orthopaedic surgeons reviewed both of these scans. Osteomyelitis was subsequently ruled out as a cause of the staphylococcal bacteraemia.

The transthoracic echocardiogram demonstrated a small mobile echogenic mass attached to the aortic valve of uncertain aetiology, moderate aortic regurgitation, mild to moderately impaired left ventricular systolic function, mild tricuspid regurgitation and mildly elevated pulmonary artery pressure.

A trans-oesophageal echocardiogram was requested. This demonstrated aortic valve endocarditis with a small aortic root abscess and mild to moderate aortic regurgitation. The patient was given a six week course of intravenous antimicrobials.

ECGs and Echos were repeated at regular intervals to exclude conduction abnormalities and left ventricular compromise respectively. His case was discussed with a cardiologist with an interest in endocarditis who felt it best to manage his endocarditis medically. The patient's inflammatory markers improved, blood culture results were subsequently confirmed as a methicillin resistant Staphylococcus aureus species.

Discussion

This case describes an elderly man presenting acutely unwell with non-specific symptoms of infection who developed signs of endocarditis. We will discuss the differential diagnosis, investigations and management of this condition.

Infective endocarditis is an infection of the endocardial surface of the heart. The condition itself can present acutely or sub-acutely. It is important to have a high index of clinical suspicion, as diagnosis is often difficult to diagnose. Prevalence in the elderly population is on the rise in developed countries(1,2) and mortality rates are high.(3)

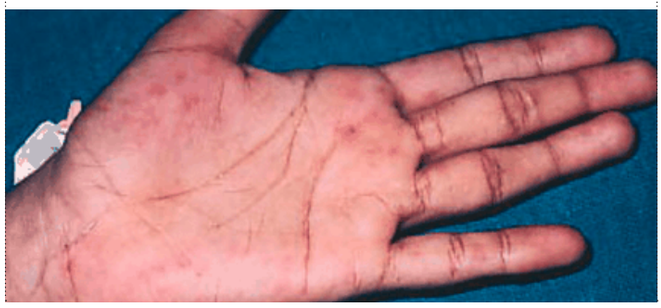
Several studies have demonstrated that males are more commonly affected. Known risk factors in the elderly population are diabetes mellitus, intestinal neoplasia, urinary tract lesions, invasive procedures of any nature, valve prosthesis, haemodialysis and immunosupression. Clinical features which occur in younger patients are seldom present in the elderly.

The diagnosis of infective endocarditis is made with the commonly accepted modified Duke Criteria(4) summarised in Table 1.

Major Criteria	Minor Criteria
1. Positive Blood Culture 2. Vegetation 3. Abscess 4. Dehiscence of prosthesis or new prosthesis regurgitation 5. Serologic test results	1. Predisposing native cardiac condition 2. Drug abuse 3. Fever 4. Embolic events 5. Osler nodes, Roth spots or Janeway lesions

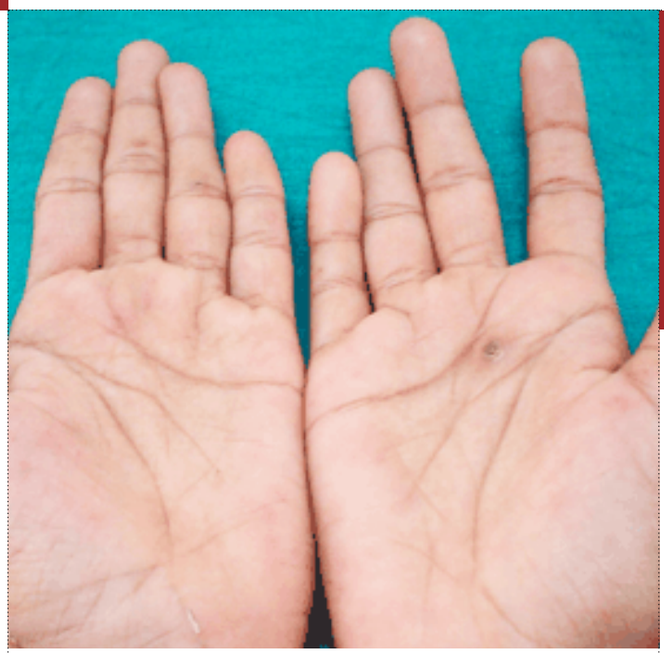
Table 1.

Modified Duke Criteria for the diagnosis of Infective Endocarditis. A diagnosis can be suggested in any of three ways: two major criteria, one major and three minor criteria or five minor criteria.



CASE-BASED DISCUSSION: INFECTIVE ENDOCARDITIS IN AN OLDER PERSON

S Boot, E Thomas, M Charlesworth, OJ Corrado



Staphylococcus aureus (More common in patients with prosthetic valves, intra venous drug abuse and acute infective endocarditis)	31%
Streptococcus viridans (more common in subactue infective endocarditis)	17%
Enterococci	11%
Coagulase negative staphylococcus	11%
Streptococcus bovis	7%

Table 2.
Common causative organisms and their approximate incidence

Echocardiography plays an important role but inter and intra-operator variability leads to varied sensitivity and specificity. Common aetiological organisms are shown in Table 2 with approximate incidence.

Despite the non-specific presentation in older people, a detailed history must be taken and specific information sought. Systemic symptoms include fever, malaise, myalgia, headache, weight loss and anorexia. Cardiovascular symptoms include chest pain, breathlessness and leg swelling. Weakness or "stroke mimic" may be the only feature in some cases.

There are many classic clinical stigmata of infective endocarditis. These include evidence of large and small emboli, presenting with physical signs affecting the hands and feet, conjunctiva, fundi and skin.

Case-Based Discussion: Infective Endocarditis in an Older Person
Patient Management.

The peripheral cutaneous or mucocutaneous lesions are Osler nodes, Janeway lesions, Roth spots, splinter haemorrhages and petechiae. Splinter haemorrhages are seen in the nail beds as linear reddish-brown lesions. Janeway lesions are found on the palms and soles, they are macular, non-blanching nodules. Roth spots are retinal haemorrhages. On cardiovascular examination a murmur may be heard. The most common murmur is aortic regurgitation. There may also be features of congestive cardiac failure.

Basic initial investigations include:

- blood tests: full blood count, urea and electrolytes, liver function tests, CRP and ESR;
- three sets of blood cultures;
- urine dip testing (for haematuria);
- baseline ECG – monitor regularly for signs of ischaemia, conduction delay and heart block;
- chest radiograph for signs of septic emboli or heart failure.

Further investigation is cardiac ultrasound. Trans-thoracic echocardiography will detect vegetations in 60% of cases but is less sensitive in patients with prosthetic valves. Trans-oesophageal echocardiography is more sensitive and specific and detects over 90% of vegetations.

The principles of management include liaison with microbiologists for appropriate antibiotic therapy and referral to cardiology as the patient may require surgery. Duration of antibiotic treatment varies but is usually around 6 weeks.

CASE-BASED DISCUSSION: INFECTIVE ENDOCARDITIS IN AN OLDER PERSON

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Elderly patients have a lower complication rate. They have fewer strokes, less embolic disease and less surgical intervention.

Cardiac risk factors include heart failure, abscess formation, pericarditis and aortic valve dissection. Cardiac complications are the ones which are most commonly encountered.

Embolic complications include stroke, myocardial infarction, gangrene, paralysis, and visual disturbance.

Neurological complications include encephalopathy, meningitis, cerebral haemorrhage and brain abscess formation.

The kidneys may be affected with glomerulonephritis. Bones may be affected with osteomyelitis. Importantly, iatrogenic complications can arise due to antibiotic use such as the development of Clostridium difficile. or local solid organ toxic effects.

Complications can also arise from surgical intervention. Even when the patient has a diagnosis and treatment plan with subsequent clinical improvement, the patient should be carefully monitored for complications.

Prognosis varies widely depending on whether the valve is native or prosthetic and on the organism involved. Despite the lower rates of complications among the elderly population, the rate of in-hospital death is twice that of younger patients.

Conclusion

This case describes the diagnosis, investigation and immediate/long term management of an elderly patient presenting with infective endocarditis.

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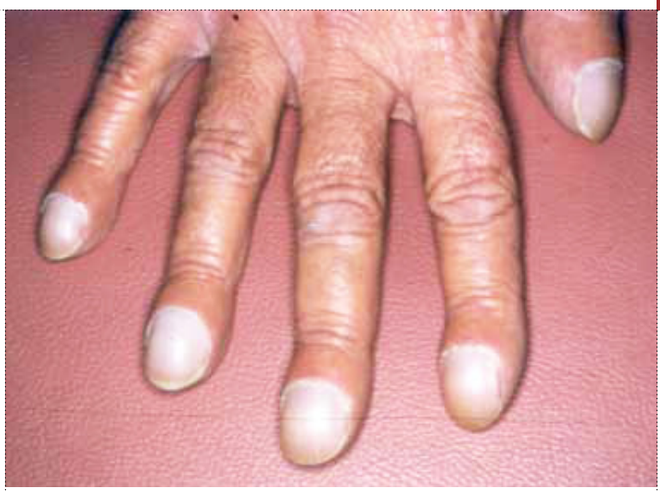

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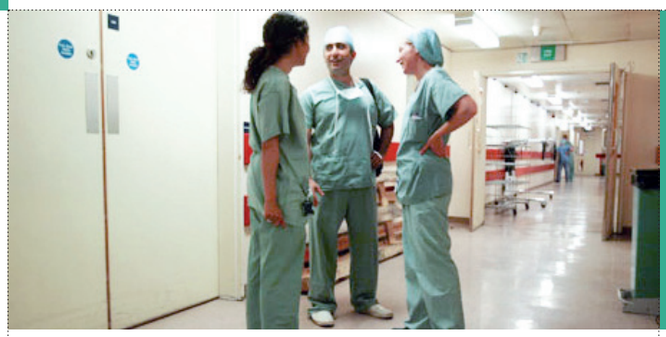
REFLECTIVE PIECE – PART 2

K Payne



Reflective Piece – Part 2 .
Teaching & Training.

In April last year I published a reflective article in this journal, describing my first day as a Foundation Year 1 doctor, my trouble with a surgical patient, difficulties with senior cover and obtaining a psychiatric review. Two years later I am two weeks away from completing my foundation training, with good days, bad days, and lots of horrible oncall night shifts behind me.

Perhaps it is difficult not to develop an air of cynicism working as a doctor in the current climate of the NHS. Understaffing plays havoc with junior rotas, cutbacks are everywhere, pay has been frozen, and now politicians tell us we will be working longer and receiving a smaller pension. How do we survive? Not wanting to sound like a cliché, but I think we must remember that we still do good, and that is a privilege that on occasion can have the highest of rewards. I describe one such scenario.

A 79-year-old lady presented with acute onset shortness of breath, on a history of previous bilateral pulmonary embolisms, and a stroke 1 month ago. She was hypoxic, tachycardic, with a pulmonary embolism being the most likely diagnosis. As she was being weighed to calculate her treatment dose low molecular weight heparin, she collapsed, going into a pulseless electrical activity cardiac arrest.

Immediate CPR was started and the Advanced Life Support algorithm followed. Her pulmonary embolism was the most likely cause, but we had a dilemma surrounding treatment. Can we ignore the absolute contraindication of a recent stroke, and thrombolyse her to treat the PE? Intense discussion between the medical registrar and ITU registrar resulted in the opinion that we had nothing to lose, so we thrombolysed her, only remembering afterwards that CPR needed to continue for 45 minutes!

After 2 two minute cycles of CPR she regained a pulse, only to re-arrest 5 minutes later. The situation looked dire, and the patient was mottled and grey, all hope was gone. However after 3 more cycles of CPR the patient again regained a pulse, and displayed signs of spontaneous breathing. One hour later the patient remained extubated, unconscious and looking very sick. She was deemed not to be a candidate for ITU, and was intubated, given high flow oxygen and observed. She was not expected to make it through the night.

Miraculously her condition didn't deteriorate. She slowly began to rally, and 10 hours later was making incomprehensible sounds, and regaining consciousness. At this point I went home to sleep, still not knowing if the patient would be alive when I returned in 12 hours.

On arriving back to work the patient was sitting up in bed with a smile on her face. I sat next to her and explained how pleased I was that she was awake. She complained about an aching pain in her chest and I sheepishly answered that my CPR efforts had been very rigorous, and the broken ribs were probably my fault, and she smiled even more.

If I reflect on my attitude to work and healthcare, then I can't help but think that I have turned from an eager young doctor, to a cynical old man. From conversations spent around a pub table with colleagues, I know that other junior doctors also share my views.

The horrible hours, the pressure of passing even more exams, and even more interviews just to get another job. But for many years to come I will remember the 79 year old lady who lived. The chances of surviving a PEA arrest are around 1%.

However in one moment working as part of a team, we beat the odds and gave life back to someone who was definitely going to die. That is something very special, and something that makes everything else worthwhile. I am sure every junior doctor at some stage will have the same experience, and hopefully be inspired that regardless of politics; to do good is still the first and most important thing we achieve.

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