Appendicitis: staying out of hospital

Antibiotics are an effective and safe alternative to appendicectomy in cases of CT confirmed acute appendicitis, but the best regimen isn’t yet clear. This Finnish study found that either one week of oral antibiotic (moxifloxacin) or two days of intravenous antibiotic (ertapenem) followed by five days of levofloxacin and metronidazole met the prespecified threshold for treatment success—discharge from hospital without needing surgery and no recurrence within one year.

The oral treatment is more practical to deliver, and moxifloxacin is broad spectrum, but runs the risk of antibiotic resistance. Unfortunately, the oral option failed to demonstrate non-inferiority in this trial. The World Society of Emergency 2020 guideline suggests antibiotics as a safe alternative to surgery in uncomplicated acute appendicitis without appendicolith, and this has, for obvious reasons, been more widely adopted during the pandemic.

Machine learning for predicting adverse events

Devising an individualised management strategy after an acute coronary syndrome relies on accurate prediction of ischaemic and bleeding events. But current prediction tools are often not accurate enough at an individual level. This study found that a risk stratification model based on machine learning (the PRAISE score) performed well in predicting all cause death, recurrent acute myocardial infarction, and major bleeding after acute coronary syndrome. The days of one treatment size fits all are receding.

The quest for heart failure drugs that work

There’s a gap in the market for treatments that boost systolic function and improve outcomes in patients with heart failure and reduced ejection fraction. Omecamtiv mercabil, a novel selective cardiac myosin activator, is already known to improve cardiac function.

This study claimed a lower incidence in a composite of heart failure event or death from a cardiovascular cause among the patients receiving omecamtiv mercabil than among those taking placebo. But the results were actually quite disappointing; there was no significant difference in the death rate from cardiovascular causes (19.6% v 19.4%) or the frequency of ischaemic or ventricular arrhythmia events. There was only a modest fall in primary outcome events—a composite of first heart failure event requiring hospitalisation or an urgent visit or death from cardiovascular cause (37.0% v 39.1%). The study was limited to patients under 85 years old in a stable clinical condition; so not fully representative of the patient population.

Saliva as good as a swab up the nose?

Nasopharyngeal swabbing, done properly, is unpleasant; spitting into a tube is much easier and more acceptable to patients. So this study comparing saliva with nasopharyngeal nucleic acid amplification testing (NAAT)—a generic term covering techniques such as PCR—in testing for covid-19 is timely, and the results are welcome. The systematic review and latent class meta-analysis found that saliva NAAT had a similar sensitivity (83.2% v 84.8%) and specificity (99.2% v 98.9%) to that of nasopharyngeal NAAT.

The findings seem robust despite significant heterogeneity among the included studies. However, an important caveat is that there were few data about patients’ symptoms or how ill they were, so it remains unclear whether the two sampling methods are equally good in all circumstances.

If the shoe fits

Walking is the only activity left for many of us in these pandemic days. So pity those whose enjoyment of walking is hampered by osteoarthritis of the knee. This study asked whether wearing flat, flexible shoes is preferable to stable, supportive shoes as some previous evidence has suggested.

This was clearly a highly motivated group of participants; an impressive 98% completed the six month intervention of wearing flat flexible or stable supportive shoes for at least six hours a day. No evidence was found that one type of shoe is better than the other. Stable, supportive ones were associated with some modest reduction in pain, knee related quality of life, and hip pain on the same side, but there was no difference in physical function. There was no control group who wore “usual shoes,” and the results aren’t necessarily generalisable to a wider population. The type of shoes probably matters less than finding a comfy pair.

Ann Robinson is an NHS GP and health writer and broadcaster.
EASILY MISSED?

Cauda equina syndrome

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This is one of a series of occasional articles highlighting conditions that may be more common than many doctors realise or may be missed at first presentation. The series advisers are Anthony Harnden, professor of primary care, Department of Primary Care Health Sciences, University of Oxford, and Dr Kevin Barraclough, School of Social and Community Medicine, University of Bristol.

A 42 year old woman presented to an out-of-hours general practitioner with a five day history of low back pain with burning pain radiating into her right foot. She had a body mass index of 39 and a 12 year history of chronic low back pain with intermittent left sided “sciatica” pain in her thigh. She had noted “tingling” in her genital area. There was no incontinence. The general practitioner considered, on examination, that anal tone and perianal sensation were normal, as were power, tone, reflexes, and sensation in the legs. The patient was treated with analgesia and given advice to seek review if she developed bilateral sciatica, became incontinent, or developed leg weakness. Three days later, she was admitted with cauda equina syndrome and underwent surgical decompression. She was left with lower limb weakness, numbness of the genitalia, loss of sexual function, and urinary and faecal incontinence.

What is cauda equina syndrome?

Cauda equina syndrome (CES) is a rare condition in which the lumbosacral nerve roots that extend below the spinal cord itself are compressed within the lumbosacral spinal canal (figure). Usually the cause is a central disc prolapse at the L4/5 or L5/S1 level. More rarely, compression can occur due to pathology in a higher disc or to infection or a tumour.

The nerve fibres in the cauda equina supply saddle, bladder, and rectal sensation; sensation and motor control of the external urethral and anal sphincters; and the fine calibre parasymathetic fibres of the pelvic viscera. There are also lower motor neurones, so any weakness in CES will be flaccid, not spastic. The autonomic fibres, in particular, are fine in calibre and are rapidly and irreversibly damaged by pressure.

The clinical features of CES are those of loss of bladder and urethral sensation, and alteration of saddle sensation in someone who usually also has unilateral or bilateral sciatica. The clinical features of CES can present in one of three ways: as one of the first features of lumbar disc herniation (type I), as an endpoint in someone with chronic low back pain (type II), or develop slowly over days (or even weeks) with gradually progressive saddle numbness and urinary symptoms (type III).

CES is diagnosed when there are both clinical features of CES and evidence of compression of the cauda equina on magnetic resonance imaging (MRI). This poses a challenge since: (a) some of the features of CES occur frequently in patients without radiological evidence of CES, and (b) radiological evidence of central disc prolapse causing compression of the cauda equina occurs in patients without clinical features of CES.

WHAT YOU NEED TO KNOW

- Although bilateral sciatica is the classic “red flag” symptom for cauda equina syndrome (CES), it is present in only about 50% of cases
- It is critical to diagnose CES before the patient becomes incontinent. Advice to return if the patient becomes incontinent is too little too late
- Pain inhibition may cause difficulty passing urine, but patients with pain inhibition alone do not have loss or reduction in bladder or urethral sensation or perianal sensory disturbances
- Assessment of anal tone is a poor predictor of cauda equina function, while subjective disturbance of saddle sensation is an unusual symptom that needs to be considered carefully. The accuracy of perianal sensory testing is unknown, and normal results should not be over-interpreted
"red flag" features but do not give adequate lead time for intervention and do not advise clinicians how to weight the features. Urinary incontinence is a well recognised “red flag” for CES, for example, but, by the time a patient with CES has urinary incontinence, it is too late: the fine parasympathetic nerves and the sacral sensory fibres in the cauda equina that maintain continence and sexual function will not recover, and the patient is likely to remain incontinent for life. As is often the case in clinical medicine, it is unclear which features might reasonably “rule out” CES (for example, normal examination findings) or “rule in” the condition (for example, any perianal symptoms in someone with back pain). This leaves the assessing generalist clinician, who is unlikely to have ever encountered the condition before, torn between not wanting to miss the diagnosis or to needlessly alarm patients with unnecessary testing.

Why does it matter?

If a patient who is developing CES but is not yet incontinent undergoes surgery, there is a reasonable chance of avoiding the potentially catastrophic consequences of urinary incontinence, faecal incontinence, loss of sexual function, saddle anaesthesia, neuropathic pain, and sometimes paralysis of the legs. This early stage is often referred to as cauda equina syndrome incomplete (CESI). The patient may have reduced bladder or urethral sensation and/or saddle sensory disturbance, but they retain bladder control. Once the patient has developed urinary retention and overfl ow incontinence (known as CES with retention or CESR), the outcome of surgery is much worse. 8 9 CES is one of the leading causes of medical litigation in the UK, and the average compensation, even by 2004, was £336 000. 10

How is it diagnosed?

The initial crucial step is that the clinician must take a careful history with the possibility of CES and its multiple presentations in mind. The clinician also needs to determine how much weight to put on the presence or absence of clinical symptoms and (importantly) clinical signs.

The clinical features of CES are usually considered to be a combination of the following occurring in someone with acute or chronic low back pain: disturbance of urinary function, disturbance of saddle sensation, reduced anal tone, and possibly bilateral sciatica. Many patients with severe low back pain have some diffi culties with passing urine. They may fi nd it diffi cult to maintain the necessary position (sitting or standing) over the lavatory, for example, and relaxing the muscles of the pelvic floor can exacerbate already severe low back pain. Pain may also cause an involuntary inhibition of micturition. Drugs such as gabapentin, anticholinergics, and opiates also affect bladder function. Crucially, in patients with pain inhibition alone, there is no loss of bladder sensation and no disturbance of saddle sensation.
“Red flags” for cauda equina syndrome in patients with sciatica

- Onset of bilateral numbness or weakness in the legs
- Onset of any sense of numbness or pins and needles around the anus (a “numb bum”) or genitals
- Any alteration in the sensation of a full bladder, desire to pass urine, or awareness of passing urine

A patient with neurological bladder dysfunction due to CES will have some or all of the following symptoms:
- Reduced awareness of bladder filling
- Loss of the urge to void
- Reduced awareness that micturition is occurring
- Inability to voluntarily interrupt the stream of urine in mid-flow
- Recent onset or progressively worsening weak urinary stream with terminal dribbling (this is obviously also common in men with bladder outflow obstruction and can occur with anticholinergic drugs)
- Loss of urethral sensation or altered sensation over the genitalia
- Some alteration of perianal sensation (a “numb bum” on wiping). There may also be perianal paraesthesia or pain.

Of note, patients with loss of bladder muscle tone can sometimes “compensate” by contracting abdominal muscles or pressing on the lower abdomen to force micturition.

Bilateral sciatica

Around half of patients with CES have bilateral sciatica.5,7,11 Sciatica is pain or altered sensation in a nerve root distribution that generally goes below the knee to the foot. Many people with severe low back pain get pain referred into the posterior thighs. That is typically not radicular nerve pain (sciatica). In CES, the affected dermatomes are almost always L4, L5, or S1,2 and these cause pain or altered sensation that extends below the knee into the foot. However, half of patients have only unilateral sciatica, and, somewhat paradoxically, as a disc fragment migrates centrally to cause CES, the sciatica may improve.

Three useful UK studies retrospectively examined the clinical features of sequential patients referred to tertiary units with features of suspected CES who underwent MRI scans.3,4,7 Of the total of 413 patients in the three studies, 106 (26%) had CES. Of the 106 patients with CES, 43 (41%) had bilateral sciatica. Of the 307 patients without CES, 78 (25%) had bilateral sciatica. Thus, the absence of bilateral sciatica does not exclude CES, nor does its presence rule it in; the appropriate safety-netting advice for someone with sciatica but no other features to suggest CES is also unclear.

Perianal sensation, saddle anaesthesia, and reduced anal tone

If a patient has acute loss or reduction of perianal sensation, they will usually be quite aware of it, just as a patient who has had dental anaesthesia will be aware that the skin over the jaw is numb. No studies have examined the internal agreement of clinicians when assessing perianal sensation. It is not an examination that general practitioners do often, so the reliability of findings (normal or abnormal) should not be over-estimated.

Reduced anal tone was recorded in 23 (22%) of 106 patients with CES,3,4,7 and normal anal tone was recorded in 35 (33%) of those with CES. Other studies of anal tone assessments in clinical circumstances and using model simulations have shown poor levels of sensitivity, specificity, and internal agreement.12,13

Objective saddle numbness was recorded in 63 (59%) of those with CES, and normal saddle sensation was recorded in 31 (29%).3,4,7 Thus, around 40% of those with CES were not recorded as having objective signs of perianal sensory loss. However, one potential limitation of the data is that the experience level of the examining clinician is not recorded. It could be argued that, if a patient has symptoms of disturbed saddle sensation, then specialist opinion should be sought irrespective of signs, while if there are no sensory symptoms it is most unlikely there will be signs. It also seems likely that symptoms which are volunteered (such as, “When I wipe myself it seems numb”) are likely to have a higher positive predictive value than elicited symptoms (such as “Have you noticed any numbness when wiping your bottom?” “Well, yes. A bit.”). However, this has not been studied, and it is not what current guidance states.

Thus, the perceived absence of clinical signs (particularly with non-specialist assessment) does not necessarily rule out CES in patients in whom the clinical suspicion is high.

Guidance on suspicion and referral for assessment of possible CES

Ideally, prescriptive advice would describe early features of CES, not late or irreversible features such as urinary and faecal incontinence. A general practitioner could consider advising a patient with sciatica to return or seek urgent help if they develop the clinical features listed as “red flags” (see box).

In the case described above, the general practitioner followed current guidance on the assessment and management of possible CES. However, safety netting advice to seek help if incontinence develops was probably ill advised and illogical, since bladder function will be lost by that stage. Perianal sensory disturbance is an unusual symptom and should possibly be given more weight than current guidance suggests, even in the absence of clear signs of perianal sensory loss. It is also necessary to be clear that a significant proportion of patients with CES never develop bilateral sciatica or limb weakness.

How is cauda equina syndrome managed?

Cauda equina syndrome incomplete (CESI) or CES with retention is diagnosed if the patient has clinical features of CES and radiological evidence of compression of the cauda equina on MRI. CES is a neurosurgical emergency and patients undergo surgical decompression.

Competing interests: None declared.

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Acute coronary syndromes: summary of updated NICE guidance

Simon J Corbett, Saoussen Ftouh, Sedina Lewis, Kate Lovibond, on behalf of the Guideline Committee

Acute coronary syndromes (ACS), comprising ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina, are an important cause of morbidity and mortality in the UK and worldwide. The National Institute for Health and Care Excellence (NICE) previously published four guidelines to improve care for people in the UK who have had an ACS. In 2018, NICE identified eight key areas of clinical practice across all aspects of existing ACS guidelines that should be reviewed for update on the basis of new evidence and stakeholder feedback. These guidelines have been incorporated into one updated guideline covering all aspects of ACS management (NG185).

Recommendations

NICE recommendations are based on systematic reviews of best available evidence and explicit consideration of cost effectiveness. When minimal evidence is available, recommendations are based on the Guideline Committee (GC)’s experience and opinion of what constitutes good practice. Evidence levels for the recommendations are in the full version of this article on bmj.com.

STEMI—early management

The infographic summarises the early management of people with STEMI from diagnosis to hospital discharge. Primary PCI from the radial route remains the preferred reperfusion treatment for STEMI, but evidence from randomised trials and health economic modelling has led to new recommendations on the choice of anti-platelet and anti-thrombin therapy in primary PCI, the use of drug-eluting stents, and complete revascularisation in STEMI patients with multivessel coronary artery disease.

- For people with acute STEMI who are having primary PCI, offer
  - prasugrel as part of dual anti-platelet therapy with aspirin if they are not already taking an oral anticoagulant (use the maintenance dose in the summary of product characteristics. For people aged 75 and over, think about whether the person’s risk of bleeding with prasugrel outweighs its effectiveness, in which case offer ticagrelor or clopidogrel as alternatives).
  - clopidogrel, as part of dual antiplatelet therapy with aspirin, if they are already taking an oral anticoagulant.

- If stenting is indicated, offer a drug-eluting stent to people with acute STEMI undergoing revascularisation by primary PCI.

- Offer complete revascularisation with PCI for people with acute STEMI and multivessel coronary artery disease without cardiogenic shock. Consider doing this during the index hospital admission.

- Consider culprit vessel only revascularisation during the index procedure for people with acute STEMI and multivessel coronary artery disease with cardiogenic shock.

NSTEMI and unstable angina—early management

The infographic summarises the early management of people with NSTEMI or unstable angina from diagnosis to hospital discharge. Table 1 (see bmj.com) summarises the relative benefits and risks of early invasive versus initial conservative management, which can be tailored for discussion with people with unstable angina or NSTEMI according to their individualised 6-month risk of mortality or repeat cardiovascular event. As for STEMI, recommendations on anti-platelet therapy and drug-eluting stents have been updated in light of new evidence from randomised trials and original health economic modelling.

- As soon as the diagnosis of unstable angina or NSTEMI is made, and aspirin and anti-thrombin therapy have been offered, formally assess individual risk of future adverse cardiovascular events using an established risk scoring system that predicts 6 month mortality (for example, Global Registry of Acute Cardiac Events).

- Offer immediate coronary angiography to people with unstable angina or NSTEMI if their clinical condition is unstable.

WHAT YOU NEED TO KNOW

- Prasugrel is recommended as dual anti-platelet therapy in combination with aspirin for people with ST-elevation myocardial infarction being treated with primary percutaneous coronary intervention (PCI).
- Prasugrel or ticagrelor is recommended as dual anti-platelet therapy in combination with aspirin for people with non-ST-elevation myocardial infarction or unstable angina being treated with PCI.
- In people with acute coronary syndromes treated with PCI, who have a separate indication for oral anticoagulation (eg, atrial fibrillation), use clopidogrel and oral anticoagulant for up to one year. Do not use prasugrel or ticagrelor, and avoid long term addition of aspirin.
• Consider coronary angiography (with follow-on PCI if indicated) within 72 hours of first admission for people with unstable angina or NSTEMI who have an intermediate or higher risk of adverse cardiovascular events (predicted 6 month mortality above 3.0%) and no contraindications to angiography (such as active bleeding or comorbidity).

• Consider coronary angiography (with follow-on PCI if indicated) for people with unstable angina or NSTEMI who are initially assessed to be at low risk of adverse cardiovascular events (predicted 6 month mortality 3.0% or less) if ischaemia is subsequently experienced or is demonstrated by ischaemia testing.

• Be aware that some younger people with low risk scores for mortality at 6 months may still be at high risk of adverse cardiovascular events and may benefit from early angiography.

• Do not offer dual anti-platelet therapy to people with chest pain before a diagnosis of unstable angina or NSTEMI is made.

• For people with unstable angina or NSTEMI who are having coronary angiography, offer – prasugrel or ticagrelor, as part of dual anti-platelet therapy with aspirin, if they have no separate indication for ongoing oral anticoagulation (if using prasugrel, only give it once coronary anatomy has been defined and PCI is intended, and use the maintenance dose in the summary of product characteristics. For people aged 75 and over, think about whether the person’s risk of bleeding with prasugrel outweighs its effectiveness) – clopidogrel, as part of dual anti-platelet therapy with aspirin, if they have a separate indication for ongoing oral anticoagulation.

• If stenting is indicated, offer a drug-eluting stent to people with unstable angina or NSTEMI undergoing revascularisation by PCI.

Cardiac rehabilitation and secondary prevention
The infographic summarises the recommendations for cardiac rehabilitation and secondary prevention following ACS. Most of these recommendations are unchanged from those in guideline CG172, but new evidence from randomised trials has allowed guidance to be written regarding the treatment options for people with ACS who have a separate indication for oral anticoagulation, such as atrial fibrillation or venous thromboembolism. No relevant clinical studies were identified for the GC to answer the question of, “What is the optimal duration of β-blocker therapy to improve outcomes for adults without left ventricular dysfunction after myocardial infarction?” as set out in the scope of the guideline update.

• For people who have a separate indication for anticoagulation, take into account all of the following when thinking about the duration and type (dual or single) of anti-platelet therapy in the 12 months after an acute coronary syndrome:
  – bleeding risk
  – thromboembolic risk
  – cardiovascular risk
  – person’s wishes.

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE
Committee members involved in this guideline update included lay members (EG and LG) who contributed to the formulation of the recommendations summarised here.

GUIDELINES INTO PRACTICE
• How do treatment pathways in my hospital need to change so that I can offer patients with ACS optimal dual anti-platelet therapy?
• How should I ensure in primary care that people with ACS treated with PCI who have a separate indication for oral anticoagulation (e.g., atrial fibrillation) are on a recommended combination of anticoagulant and anti-platelet therapy?

• Be aware that the optimal duration of aspirin therapy has not been established, and that long term continuation of aspirin, clopidogrel, and oral anticoagulation (triple therapy) significantly increases bleeding risk.

• For people already on anticoagulation who have had PCI, continue anticoagulation and clopidogrel for up to 12 months. If the person is taking a direct oral anticoagulant, adjust the dose according to bleeding risk, thromboembolic risk, and cardiovascular risk.

• For people with a new indication for anticoagulation who have had PCI, offer clopidogrel (to replace prasugrel or ticagrelor) for up to 12 months and an oral anticoagulant licensed for the indication which best matches the person’s – bleeding risk
  – thromboembolic risk
  – cardiovascular risk
  – wishes.

• Do not routinely offer prasugrel or ticagrelor in combination with an anticoagulant that is needed for an ongoing separate indication for anticoagulation.

• For people with an ongoing indication for anticoagulation 12 months after a myocardial infarction, take into consideration all of the following when thinking about the need for continuing antiplatelet therapy:
  – the indication for anticoagulation
  – bleeding risk
  – thromboembolic risk
  – cardiovascular risk
  – the person’s wishes.

• Consider continuing a β-blocker for 12 months after a myocardial infarction for people without reduced left ventricular ejection fraction.

• Discuss the potential benefits and risks of stopping or continuing β-blockers beyond 12 months after a myocardial infarction for people without reduced left ventricular ejection fraction. Include in the discussion – the lack of evidence on the relative benefits and harms of continuing beyond 12 months
  – the person’s experience of adverse effects.

Competing interests: See bmj.com.
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Acute coronary syndrome: Early management
Highlighting the changes in the updated NICE guidance

The updated NICE guidance [https://www.nice.org.uk/guidance/ng185] on acute coronary syndrome replaces four separate guidelines published between 2010 and 2013. This graphic shows a summary of the early management part of the new guidance, highlighting the new additions.

**STEMI**
ST elevation myocardial infarction

- **Offer aspirin**
  - 300 mg loading dose
  - As soon as possible

- **Initial antithrombin therapy**
  - **Low Bleed risk:** Offer fondaparinux
  - **High Bleed risk:** Think about choice and dose of antithrombin

- **Estimate 6 month mortality**
  - **Low risk** Predicted 6 month mortality ≤ 3%
  - **Intermediate or higher risk** Predicted 6 month mortality > 3%

- **Continuing myocardial ischaemia or cardiogenic shock?**
  - **YES**
  - **NO**

- **Can PCI* be delivered within 120 minutes?**
  - **YES**
  - **NO**

- **Consider**
  - **Offer**

- **Fibrinolysis** Give an antithrombin at the same time

- **New section on drug therapy**

- **Offer**
  - **Already taking oral anticoagulant?**
    - **No**
      - **Offer prasugrel with aspirin**
      - **Offer clopidogrel with aspirin**
    - **Yes**

- **Low Bleed risk:**
  - **Offer ticagrelor with aspirin**
  - **Consider clopidogrel with aspirin, or aspirin alone**

- **High Bleed risk:**
  - **Consider clopidogrel with aspirin, or aspirin alone**

- **STenting**
If stenting is indicated, offer a drug eluting stent

- **Assess left ventricular function**

- **Radial**
  - **Offer**
    - **Unfractionated heparin with bailout GPI§**

- **Femoral**
  - **Consider**
    - **Bivalirudin with bailout GPI§**

- **Offer immediate angiography with follow-on PCI if indicated by ECG**

- **Seek specialist advice for recurrent myocardial ischaemia and offer angiography with follow-on PCI if appropriate**

- **Consider angiography during same admission if stable after successful fibrinolysis**

- **Consider ischaemia testing before discharge**

- **Consider angiography if ischaemia develops or is shown on testing**

- **Management strategy**
If follow-on PCI not done, consider angiography findings, comorbidities and risks and benefits when discussing management strategy with:

  - The interventional cardiologist
  - The cardiac surgeon
  - The patient

- **Cardiac rehabilitation and secondary prevention**

**NSTEMI or unstable angina**
Non-ST elevation myocardial infarction

- **Unless immediate angiography will be performed**

- **If creatinine > 265 (μmol/L)**
  - **Consider unfractionated heparin**

- **Adjust dose to clotting function**

- **Offer immediate angiography with follow-on PCI if indicated by ECG**

- **Seek specialist advice for recurrent myocardial ischaemia and offer angiography with follow-on PCI if appropriate**

- **Consider angiography during same admission if stable after successful fibrinolysis**

- **Consider ischaemia testing before discharge**

- **Consider angiography if ischaemia develops or is shown on testing**

- **Management strategy**
If follow-on PCI not done, consider angiography findings, comorbidities and risks and benefits when discussing management strategy with:

  - The interventional cardiologist
  - The cardiac surgeon
  - The patient

- **Cardiac rehabilitation and secondary prevention**

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Acute coronary syndrome: rehabilitation

Highlighting the changes in the updated NICE guidance

The updated NICE guidance on acute coronary syndrome (https://www.nice.org.uk/guidance/ng185) replaces four separate guidelines published between 2010 and 2013. This graphic summarises the cardiac rehabilitation and secondary prevention part of the new guidance, highlighting new additions.

Orange boxes show changes between updated (2020) and previous (2010-13) NICE guidance.

Cardiac rehabilitation

Start cardiac rehabilitation before hospital discharge

Assessment appointment to take place within 10 days of discharge

Drug therapy for secondary prevention

ACE* inhibitor

Titrage upwards (with monitoring) every 12 to 24 hours

Complete titration in 4 to 6 weeks of hospital discharge

Continue indefinitely

Diagnostic testing before starting and after 1 to 2 weeks

Renal function

Serum electrolytes

Blood pressure

Beta-blocker

If contraindicated, consider diltiazem or verapamil if no pulmonary congestion or reduced left ventricular ejection fraction

Titrage to the maximum tolerated or target dose

Continue indefinitely if reduced left ventricular ejection fraction.

Otherwise consider continuing for at least 12 months

Statin

Continue indefinitely

New advice

Antiplatelets with anticoagulants

Do not routinely offer prasugrel or ticagrelor with an anticoagulant needed for a separate indication

Already on anticoagulation

Continue

Offer oral anticoagulant

New indication for anticoagulation

Person has PCI‡

Offer clopidogrel (to replace prasugrel or ticagrelor)

For up to 12 months

Person has no PCI‡

Consider continuing aspirin if not at high bleeding risk

Clopidogrel if aspirin is contraindicated

For up to 12 months

Lifestyle changes

Healthy eating

Mediterranean diet, including more:

Bread

Fruit

Fish

Vegetables

Plant oils

Alcohol

Low-risk drinking

No more than 14 units a week

Regular physical activity

20 to 30 minutes a day to slight breathlessness

Stopping smoking

Reaching and maintaining a healthy weight

Heart failure

with reduced left ventricular ejection fraction

Offer aldosterone antagonist

Start 3 to 14 days after myocardial infarction, preferably after ACE* inhibitor

Monitor renal function and serum potassium before and during treatment. If hyperkalaemia is a problem, halve dose or stop drug

* ACE = Angiotensin-converting enzyme † ARB = Angiotensin II receptor blockers ‡ PCI = Percutaneous coronary intervention

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**SPOT DIAGNOSIS**

**A lichenoid rash with hepatic origins**

A woman in her 70s presented with a widespread rash of three months’ duration. The rash had started on her arms and spread to the trunk and legs. She described severe itching and a burning sensation. She also had non-specific symptoms of heartburn, weight loss, and loss of appetite for a similar period.

On examination, her limbs and trunk had an extensive grey-brown rash with a hint of purple, composed of small papules and excoriated, lichenified (thickened and leathery) plaques (figure). She had no mucosal involvement.

Investigations showed a marginally raised alkaline phosphatase level of 144 U/L (normal range 30-130 U/L). After referral to the gastroenterology department, active hepatitis C infection was diagnosed after a complete liver screen (table).

What is the most likely diagnosis?

Submitted by Leila Nemazee and Janice Ferguson
Patient consent obtained.

Cite this as: BMJ 2021;372:m4976

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### RESULTS OF LIVER SCREENING TESTS

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<td>30-130 U/L</td>
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<td>Alanine aminotransferase</td>
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<td>&lt;55 U/L</td>
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<td>Transferrin</td>
<td>3.7</td>
<td>1.7-3.8 g/L</td>
</tr>
<tr>
<td>Aspartate aminotransferase</td>
<td>66</td>
<td>5-34 U/L</td>
</tr>
<tr>
<td>Lactate dehydrogenase</td>
<td>231</td>
<td>125-264 U/L</td>
</tr>
<tr>
<td>Corrected calcium</td>
<td>2.48</td>
<td>2.2-2.60 mmol/L</td>
</tr>
<tr>
<td>Phosphate</td>
<td>0.85</td>
<td>0.8-1.50 mmol/L</td>
</tr>
<tr>
<td>Creatine kinase</td>
<td>92</td>
<td>25-200 U/L</td>
</tr>
<tr>
<td>Hepatitis C virus genotype</td>
<td>3</td>
<td>Viral load 809 000 IU/mL</td>
</tr>
</tbody>
</table>

Endgame:

Results of liver screening tests

You can record CPD points for reading any article. We suggest half an hour to read and reflect on each.

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**LEARNING POINTS**

• Consider screening for hepatitis C in people with newly diagnosed lichen planus if they have gastrointestinal or systemic symptoms, or any risk factors for hepatitis C virus.

• Consider lichen planus in people with a widespread, itchy rash and a history of hepatitis C.

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**SPOT DIAGNOSIS**

**A lichenoid rash with hepatic origins**

Grey-purple excoriated papules and plaques on upper back, and post-inflammatory pigmentation
**MINERVA**

**Serpiginous erythema on the trunk**

This is erythema gyratum repens in a man in his 70s with myelodysplastic syndrome. He presented with persistent, pruritic, generalised erythroderma on the trunk and extremities of two months’ duration. He had no medical history and was otherwise well. The concentric, raised, serpiginous bands with skin peeling are typical of erythema gyratum repens, which has only been described in around 100 case reports. Differential diagnoses investigated were of drug eruption, tinea corporis, skin lymphoma, collagen disease, bullous pemphigoid, erythema annulare centrifugum, and erythema chronicum migrans. Full blood count showed pancytopenia, and myelodysplastic syndrome was diagnosed after subsequent bone marrow biopsy. About 70% of previously reported cases of erythema gyratum repens are related to mainly solid cancers, but the condition can also occur in haematopoietic malignancy.

**Pain after trauma**

Paracetamol is as good as a non-steroidal anti-inflammatory drug for post-traumatic pain and no advantage is gained in combining both treatments, according to a trial from an emergency department in Tunisia. More than 1500 people presenting with traumatic injury to an extremity were randomised to a week’s treatment with either paracetamol or piroxicam or both when they were discharged (Acad Emerg Med doi:10.1111/acem.14169). The need for additional analgesia was highest in the group allocated to piroxicam. Return visits and adverse effects were more frequent in this group too.

**Shoulder surgery**

Assessed by outcomes that included pain, function, and health related quality of life, a BMJ clinical recommendation concluded that surgical decompression in people with subacromial pain syndrome was no better than placebo (BMJ doi:10.1136/bmj.1294). Long term follow-up of one of the trials included in that review reinforces the finding. Evaluated five years after the intervention, subacromial decompression provided no benefit over diagnostic arthroscopy or exercise (Br J Sports Med doi:10.1136/bjsports-2020-102216).

**Shift work and asthma**

People who work shifts have an increased risk of metabolic disorders and cancer. The reason isn’t known but it’s assumed to be something to do with misalignment of internal circadian rhythms and the external light/dark cycle. A cross sectional study using data from 280 000 UK Biobank participants finds that shift workers also have a higher risk of asthma, even after adjusting for likely confounding factors such as smoking history, socioeconomic status, and body mass index (Thorax doi:10.1136/thoraxjnl-2020-215218).

**Wasted resources in healthcare**

Minerva is no expert in the strengths and weaknesses of crosswalk analysis. But her eye was caught by one which attempted to classify wasted resources in the healthcare systems of the US into six categories: clinical inefficiencies, missed prevention opportunities, overuse, administrative waste, excessive prices, and fraud and abuse (Am J Public Health doi:10.2105/AJPH.2020.305865). Aggregate estimates of wasteful medical care spending ranged from $600bn to a jaw dropping $1.9tn annually. That’s the equivalent of between $1800 and $5700 per person per year.

**Fatigue in people with multiple sclerosis**

Fatigue, a subjective feeling of lack of mental and physical energy, is a common and disabling symptom in people with multiple sclerosis. The results of a randomised trial of three commonly prescribed medications show why it is so hard to treat (Lancet Neurol doi:10.1016/S1474-4422(20)30354-9). Compared with placebo, participants were more likely to report adverse events while taking amantadine, modafinil, or methylphenidate. But judged by the primary outcome measure, a self-rated scale of fatigue, none of these drugs led to any improvement.

**Optic neuritis**

Although the commonest disease associated with optic neuritis is multiple sclerosis, analysis of a primary care database in the UK finds that it accounts for only about a third of cases. Other causes include syphilis, Behçet disease, vasculitis, sarcoidosis, and infection with Epstein-Barr virus and mycoplasma. Individual risk factors are female sex, obesity, being of reproductive age, and smoking. Compared with white people, people of South Asian origin or of mixed race or ethnicity are at lower risk (JAMA Neurol doi:10.1001/jamaneurol.2020.3502).