education

FROM THE JOURNALS Edited highlights of Richard Lehman's blog on http://bmj.co/Lehman

Single dose analgesics for acute limb pain

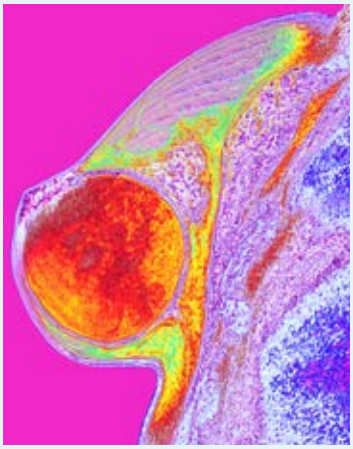
People came to two emergency departments run by the same institution in New York City with acute pain in an "extremity"—defined as anywhere distal to the shoulder or hip. This was classified by severity (moderate to severe) rather than by cause: about one in five had a fracture. On average, their pain scores diminished by over a third in two hours, after they had been given one of four different analgesic combinations, all containing paracetamol. Paracetamol 1000 mg with ibuprofen 400 mg was as effective as paracetamol 300-325 mg combined with any of three opioids: oxycodone, hydrocodone, or codeine. The bottom line message appears to be that an oral paracetamol/ibuprofen combination will relieve acute limb pain by 40% in most patients, and there seems little point in using opioids as first line treatment, though about 18% across groups needed "rescue treatment," ie, extra pain relief in the first two hours. That's fine as far as it goes. Unfortunately, it went for only two hours, there was no measurement of adverse effects, and nothing to indicate whether it had an effect on long term analgesic use. There is plenty more work to be done in this simple but important area.

JAMA doi:10.1001/jama.2017.16190

Inflammatory bowel disease drugs and lymphoma risk

Treatment choices in inflammatory bowel disease are rarely easy, but at some stage most patients will receive a thiopurine (usually azathioprine) and/or an anti-tumour necrosis factor agent. Here's a nice illustration of the importance of framing in risk communication: you're a young patient with inflammatory bowel disease and somebody tells you about a survey of nearly 200 000 patients with the condition in France, which measured the risk of lymphoma in people taking these drugs. "Taking azathioprine increases your lymphoma risk by 260% and if you take a [tumour necrosis factor] TNF blocker too it might go up by over 600%." Help: I am going to die. Or alternatively, "Taking azathioprine carries a very small risk of getting a type of cancer that is usually very treatable. It's the difference between about a quarter of a percent in 1000 years if you don't take azathioprine and half a per cent in 1000 years if you do. If you need a combination treatment it goes up to about 1% in 1000 years." Stares out of window: why is she telling me this?

JAMA doi:10.1001/jama.2017.16071



Breast cancer recurrence

We now know that there are about 20 different breast cancers, and each is likely to have its own response pattern to treatment and risk of distant recurrence. Twenty years ago, we understood less: the main characteristics measured then were oestrogen receptor status and tumour diameter and nodal status. More tumour information could no doubt be gathered retrospectively if someone did modern molecular analysis of all the histology samples from the 63 000 women in the 88 long term follow-up studies meta-analysed here.

This is not going to happen. But let's not worry: at least the *New England Journal of Medicine* has at last published a systematic review—is this a first? We are left with confirmation that oestrogen receptor positive breast cancer behaves as a systemic disease, with a steady incremental risk of recurrence from the end of hormone therapy (98% tamoxifen in these studies) up to the 20 year mark. This is strongly associated with original tumour diameter and nodal status, varying between 10% and 41% with tumour size, grade, and lymph node involvement.

N Engl J Med doi:10.1056/NEJMoa1701830

Does warfarin prevent cancer?

Now let's look at this shared decision making/risk communication stuff from another angle. Lots of decision aids have been produced to help people decide whether to take warfarin or a direct oral anticoagulant for say, stroke prevention in atrial fibrillation. This takes us to a central problem of therapeutics: it takes a long time to know the true effects of drugs. The direct oral anticoagulants are new, divided into two broad classes, but there are within-class differences too, which are just beginning to emerge. And we don't know the long term risks and benefits because they simply haven't been around long enough. Warfarin has been in medical use since 1954, but it has taken until now to discover its use is associated with a fall in the total incidence of cancers, especially of the lung, prostate, and breast. These data are robust and come from a whole-population Norwegian database, easily checked by using other whole-population databases. So, suddenly a decision aid comparing warfarin with direct oral anticoagulants should perhaps have a box saying, "Effect on cancer. Warfarin: reduces risk of several common cancers. Direct oral anticoagulant: effect not known."

JAMA Intern Med doi:10.1001/jamainternmed.2017.5512

Diabetic foot

Satish Chandra Mishra,[1] Kunal C Chhatbar,[2] Aditi Kashikar,[3] Abha Mehndiratta[4]

[1]Department of Surgery, Bhabha Atomic Research Centre Hospital, Mumbai, India
[2]KHM Hospital, Mumbai, India
[3]Seth Gordhandas Sunderdas Medical College and King Edward Memorial Hospital, Mumbai, India
[4]Global Health and Development Group, Imperial College London, St Mary's Hospital, London, UK

Correspondence to: A Mehndiratta abha@mail.harvard.edu

Foot disease affects nearly 6% of people with diabetes[1] and includes infection, ulceration, or destruction of tissues of the foot.[2] It can impair patients' quality of life and affect social participation and livelihood.[3] Between 0.03% and 1.5% of patients with diabetic foot require an amputation.[4] Most amputations start with ulcers and can be prevented with good foot care and screening to assess the risk for foot complications.[5] We provide an update on the prevention and initial management of diabetic foot in primary care.

What causes diabetic foot?

Uncontrolled diabetes contributes to the development of neuropathy and peripheral arterial disease by complex metabolic pathways.[6] Loss of sensation caused by peripheral neuropathy, ischaemia due to peripheral arterial disease, or a combination of these may lead to foot ulcers. A systematic review (78 studies from 84 cohorts) reports a prevalence of 0.003-2.8% for diabetes related peripheral neuropathy and 0.01-0.4% for diabetes related peripheral arterial disease.[4] Figure 1 depicts factors that contribute to foot complications.

Diabetes is also implicated in Charcot arthropathy, which involves progressive destruction of the bones, joints, and soft tissues, most commonly in the ankle and foot. Diabetes related Charcot's arthropathy has a reported prevalence between 0.08% and 13%, but there are no high quality epidemiological studies on Charcot's foot.[7 8] A combination of neuropathy, abnormal loading of foot, repeated micro trauma, and metabolic abnormalities of bone leads to inflammation, causing osteolysis, fractures, dislocation, and deformities.

In low and middle income countries barefoot walking, lack of awareness, delay in seeking care, and shortage of trained healthcare providers and foot care services are common factors that add to the burden of foot disease.

How is it diagnosed?

A thorough foot examination is important to detect the disease early. Screening for peripheral neuropathy and peripheral arterial disease can help identify patients at risk of foot ulcers. A history of ulcers or amputations and poor glycaemic control increase the risk.

Assess the patient's general condition for signs of toxicity or sepsis. The patient may report feeling unwell, may appear sick or have altered behaviour. Record vital parameters and check for a fever. Examine the feet for active disease such as ulceration or gangrene (fig 2). Look for lesions such as fungal infection, cracks and skin fissures, deformed nails, macerated web spaces, calluses, and deformities such as hammer toes, claw toes, and pes cavus, which increase the risk of ulceration (fig 3). Feel the temperature of the feet with the dorsum of your hand. A cold foot might suggest ischaemia, and increased warmth with redness and swelling might suggest inflammation such as acute Charcot foot or cellulitis.

Peripheral neuropathy

The aim of screening is to identify patients with loss of protective sensation in the feet. Most guidelines recommend the 10 g monofilament for neuropathy assessment (fig 4) in people with diabetes.[9 10] This monofilament exerts a 10 g buckling force when it bends. An inability to sense a 10 g pressure is the current consensus definition of loss of protective sensation. The test is portable, cheap, and easy to perform (box).[12 15] Despite the widespread use of the monofilament test, its accuracy in diagnosing neuropathy is variable.[16] The test may be combined with another test to screen for neuropathy, such as a biothesiometer or a graduated tuning fork (Rydel Seiffer) to assess vibration perception threshold.[17 18]

Peripheral arterial disease

Ask for a history of intermittent claudication and rest pain, which suggest peripheral arterial disease.[19] Palpate the posterior tibial artery and dorsalis pedis artery in both feet and record pulsations as absent or present.[20]

The ankle brachial index is an adjunct measure to diagnose peripheral arterial disease.[19 21] Availability of equipment, time constraints, and lack of training are reported as major barriers to ankle brachial index testing in primary care.[23-25]

On the basis of this initial assessment, patients can be categorised as having a low, moderate, or high risk of diabetic foot (see infographic).[9]

WHAT YOU NEED TO KNOW

- Diabetic foot can be prevented with good glycaemic control, regular foot assessment, appropriate footwear, patient education, and early referral for pre-ulcerative lesions
- Examine the feet of people with diabetes for any lesions and screen for peripheral neuropathy and peripheral arterial disease, which can lead to injuries or ulceration
- Refer patients with foot ulceration and signs of infection, sepsis, or ischaemia immediately to a specialised diabetic foot centre for surgical care, revascularisation, and rehabilitation

CPD READING
0.5 HOURS

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

No patients were involved in the creation of this article.

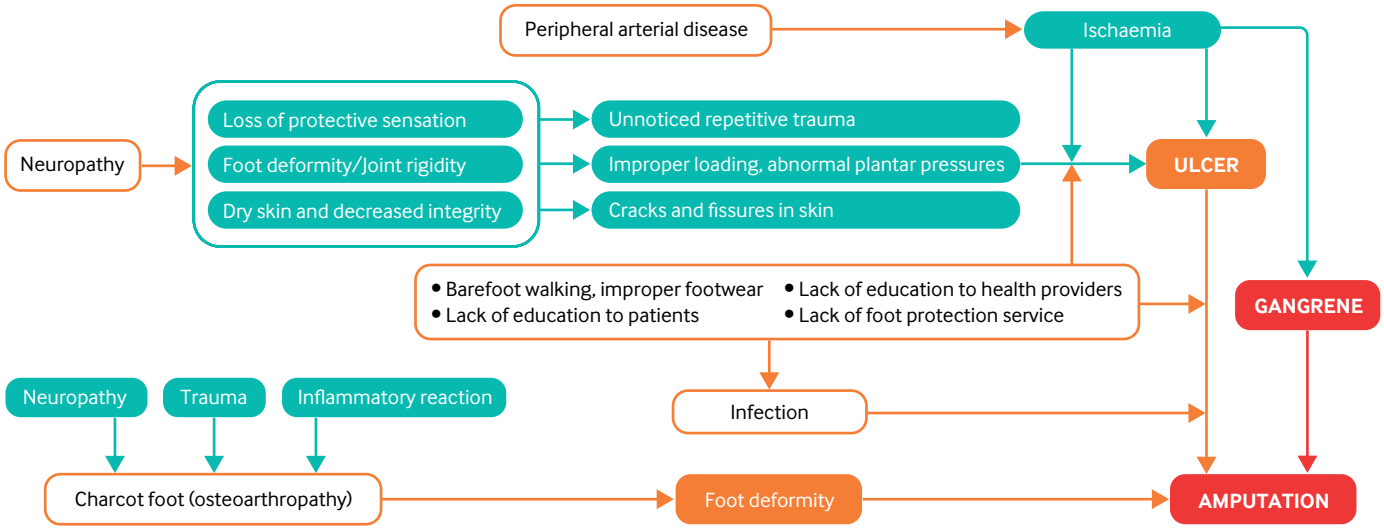


Fig 1| Risk factors and mechanism for foot ulcer and amputation

How can it be prevented?

Regular foot examination

The suggested frequency for follow-up is based on expert consensus (infographic, overleaf). For people at low risk, continue annual foot assessments as they could progress to moderate or high risk. Emphasise the importance of foot care and monitoring glycaemic control.

More frequent follow-up is advised in patients at moderate or high risk, such as those with a foot deformity or with a diagnosis of peripheral neuropathy or peripheral arterial disease at initial assessment. Repeat testing for neuropathy is not necessary if diagnosed previously.

Fig 2| Gangrene and ulcer in foot at high risk (previous toe amputation)

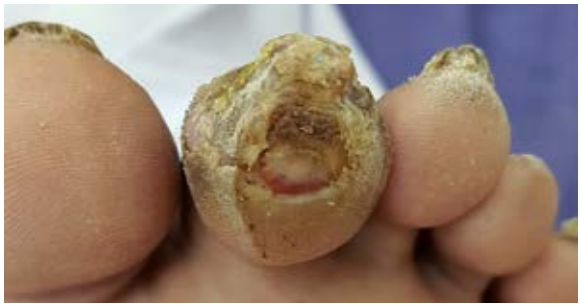


Fig 3| Hammer toe deformity with callus and ulcer. Hammer toe is caused by weakened muscles in the foot. The joint connecting the foot with the toe bends upwards and the joint in the middle of the toe bends downwards towards the floor. This results in the toe curling under the foot and being subjected to excessive ground reaction forces during walking

Neuropathy reversal is not established in studies. A quick inspection for a breach in skin integrity or ulceration should suffice. Patients with asymptomatic peripheral arterial disease may be followed up in primary care and managed as in guidelines for peripheral arterial disease.[21]

Refer patients with calluses and deformed toenails to preventive podiatry services for basic nail and skin care, including debridement of calluses. Timely referral to foot protection services for control of risk factors in patients with diabetes prevents infection, gangrene, amputation, or death, and reduces hospital admissions and costs.[9]

Glycaemic control

Early and good glycaemic control is effective in preventing neuropathy but there is a lack of studies to show that glycaemic control reverses neuropathy.[26] Discuss optimal blood sugar and glycated haemoglobin (HbA_{1c}) targets with patients and monitor these as per standard guidelines for diabetes care to prevent or slow the progression of peripheral neuropathy.[27 28]

Patient education

Offer people with diabetes or their caregivers, or both, oral and written information on:

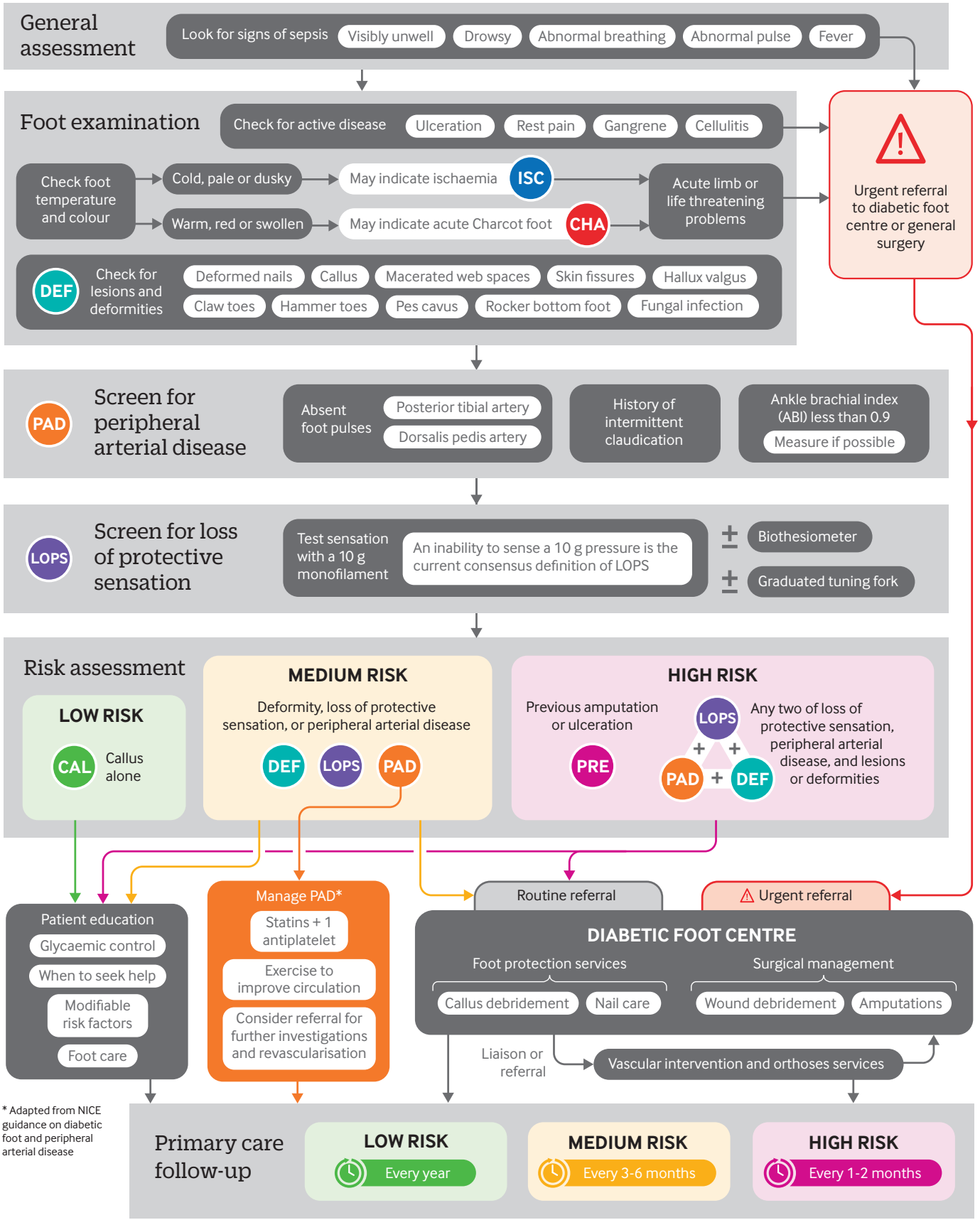
- The importance of blood glucose control and modifiable cardiovascular risk factors such as diet, exercise, body weight, and cessation of smoking.
- The importance of foot care and advice on basic foot care. While offering advice consider the patient's cultural practices and religious beliefs as well as social and family support.
- The person's current risk of developing a foot problem.
- When to seek professional help and whom to contact in foot emergencies.

Footwear

Occlusive footwear causes sweating and can predispose to fungal infection,[30 31] particularly in tropical countries. Ideally, footwear for people with diabetes should have a wide toe box, soft cushioned soles, extra depth to accommodate orthoses if required, and laces or Velcro for fitting and adjustments. A new pair of shoes can be worn for a short while daily until

Diabetic foot

Primary care assessment and monitoring



* Adapted from NICE guidance on diabetic foot and peripheral arterial disease

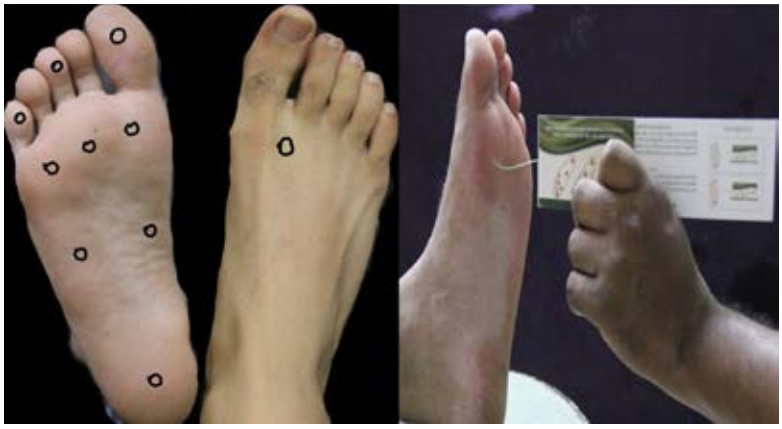


Fig 4| Monofilament test: testing sites and application. The nine plantar sites are the distal great toe; third toe; fifth toe; first, third, and fifth metatarsal heads; medial foot, lateral foot, and heel; and one dorsal site

Monofilament test

Procedure—Ask the patient to sit or lie down with both legs stretched out and soles exposed. Explain the procedure and make him or her familiar with the sensation by applying the monofilament on a sensitive area such as the palm. Ask the patient to close his or her eyes and to say "yes" every time touch is felt on the soles, no matter how lightly it is perceived. Place the monofilament at 90° to the skin and press it till it buckles to 1 cm, then hold there for 1-2 seconds and remove.[11] Test different sites in a random sequence with a pause (sham application) to prevent the patient from guessing the next application. If the patient fails to respond at a site, revisit the same site two more times in a random sequence during the assessment. If the patient does not perceive the sensation all the three times, then record the result as loss of protective sensation.[11] Loss of protective sensation even at a single site puts the patient at risk for foot complications.

Test sites and threshold—Most studies recommend testing at 10 sites. Inability to perceive a 10 g monofilament three times at even a single site means the patient has loss of protective sensation.[11 12]

Inter-observer variability—This is reported to be more on the heels, with a higher chance of a false positive result.[13] Exercise caution before labelling a heel as insensate, especially if screening a population where barefoot walking is common.

Durability of monofilaments—Monofilaments tend to fatigue with repeated use, and a 24 hour recovery period is recommended after 100 compression cycles.[14] Replace a monofilament after three months of regular use.

TIPS ON FOOT CARE FOR PEOPLE WITH DIABETES[19]

- Inspect both feet daily, including the area between the toes. Ask a caregiver to do this if you are unable to.
- Wash the feet daily with water at room temperature, with careful drying, especially between the toes.
- Use lubricating oils or creams for dry skin, but not between the toes.
- Cut nails straight across.
- Do not remove corns and calluses using a chemical agent or plaster. They should not be excised at home and must be managed by trained staff.
- Always wear socks with shoes and check inside shoes for foreign objects before wearing them.
- Avoid walking barefoot at all times.
- Ensure a qualified healthcare provider examines your feet regularly.
- Notify the healthcare provider at once if a blister, cut, scratch, or sore develops.

EDUCATION INTO PRACTICE

- In your practice, what proportion of people with diabetes have had a foot evaluation in the past 12 months?
- Describe how you would screen patients with diabetes for peripheral neuropathy and peripheral arterial disease.
- How would you advise a patient with diabetes about foot care?

comfortable. Patient compliance with prescribed footwear is usually poor, particularly at home where they are more active.[29] Patients with plantar ulcers at forefoot or heel may be offered offloading footwear to allow ulcer healing and prevent recurrence.

When to refer?

Refer immediately patients with a life threatening or limb threatening problem such as foot ulceration with fever or any signs of sepsis; ulceration with limb ischaemia; gangrene, or a suspected deep seated soft tissue or bone infection usually indicated by either a grossly swollen foot with shiny skin and patches of discoloration or a gritty feel to the bone during a probe to bone test in an open wound.[9] Refer to a specialised diabetic foot centre or to general surgery for wound care, offloading, revascularisation if needed, and rehabilitation.

Explain to patients the need to seek specialist care to limit complications. Provide detailed and clear communication before patients are referred so that multidisciplinary care can be facilitated at the earliest opportunity.

Before referral, wash the ulcer with clean water or saline and apply a sterile inert dressing such as a saline soaked gauze to control exudates and maintain a warm, moist environment for healing. Avoid microbicidal agents such as hydrogen peroxide, povidone iodine, or chlorhexidine to clean or dress the ulcer as these are cytotoxic. Costly antimicrobial dressings are not recommended.[9] Adjust dressings, footwear, and ambulation to avoid weight bearing on an ulcerated foot.[32] Early and aggressive treatment to control infection is important, especially in the presence of an ulcer. Start antibiotic treatment according to antibiotic policy based on local resistance patterns. Before starting antibiotics, take a piece of soft tissue from the base of the ulcer for culture and sensitivity, or take a deep swab for culture.[9] Refer urgently, within one or two days, patients with a history of rest pain, uncomplicated ulcer, or acute Charcot foot.[9] For patients with rest pain or intermittent claudication, offer referral to vascular intervention services for further investigations such as Duplex ultrasonography, and consideration for revascularisation.[21]

The management and referral pathways between primary care, specialty diabetic foot centres, and multidisciplinary foot care services need to be integrated (see infographic).

Competing interests: We have read and understood BMJ policy on declaration of interests and declare the following: SM, KC, and AK were members of the guideline development group for the standard treatment guideline on the diabetic foot: prevention and management in India, 2016 published by the Ministry of Health and Family Welfare, government of India. AM provided technical input on methodology to this guideline development group.

Cite this as: *BMJ* 2017;359:j5064

Find the full version with references at http://dx.doi.org/10.1136/bmj.j5064

EASILY MISSED?

Acute respiratory distress syndrome

John G Laffey,[1 2 3] Cheryl Misak,[4] Brian P Kavanagh[2 5]

[1]Departments of Anesthesia and Critical Care Medicine, Critical Illness and Injury Research Centre, Keenan Research Centre for Biomedical Science, St Michael's Hospital, Toronto, Canada
[2]Departments of Anesthesia, Physiology and Interdepartmental Division of Critical Care Medicine, University of Toronto, Canada
[3]Anaesthesia, School of Medicine, National University of Ireland, Galway, Ireland
[4]Department of Philosophy, University of Toronto, Canada
[5]Department of Critical Care Medicine and Anesthesia, Hospital for Sick Children, Toronto, Canada

Correspondence to: J G Laffey john.laffey@nuigalway.ie

CPD READING
0.5 HOURS

A 40 year old woman presented to the emergency department with fever, dyspnoea, and coughing purulent sputum. Chest radiograph revealed bilateral infiltrates, and peripheral capillary oxygen saturation (breathing 50% oxygen) was 92%. Antibiotics and a trial of non-invasive ventilation were commenced. The patient was admitted to a medical ward and then to intensive care for vasopressor infusion and invasive ventilation. The ventilation was weaned at 3 weeks, but rehabilitation was slow. Case review showed exemplary management of sepsis but noted a week's delay in the diagnosis of acute respiratory distress syndrome (ARDS) because of incorrect initial interpretation of the chest radiograph—despite all criteria for ARDS being present in the emergency department.

What is ARDS?

Acute respiratory distress syndrome was first described in 1967[1] and has become a defining condition in critical care. It is an acute inflammatory lung injury, often caused by infection, which increases lung microvascular permeability, resulting in hypoxaemic respiratory failure. It presents with dyspnoea, predominantly in the emergency department or hospital ward,[2 3] and requires assisted ventilation. Around 40% of patients with ARDS will die,[2] and survivors experience long term sequelae. No drug treatments exist for ARDS; however good supportive management reduces harm and improves outcome. Early diagnosis—ideally before admission to intensive care—maximises benefit.[4] Most cases of ARDS are diagnosed in hospital, but up to one third of patients with ARDS fulfil diagnostic criteria in the emergency department.[3] It is helpful, therefore, for primary care clinicians to be aware of ARDS and have a low threshold for rapid referral of patients who have an evolving illness associated with breathlessness and hypoxia to an emergency setting. Patients might also need additional support in the community with longer term complications.

WHAT YOU NEED TO KNOW

- Consider the possibility of acute respiratory distress syndrome (ARDS) in any sick patient with respiratory distress, especially in the presence of risk factors such as pneumonia, sepsis, trauma, or aspiration of gastric contents.
- Perform a radiograph of the chest and arterial blood gas sampling for all patients with acute respiratory distress to aid early recognition of ARDS.
- Timely diagnosis of ARDS facilitates implementation of simple measures that can reduce mortality, morbidity, and financial cost.

EDUCATION INTO PRACTICE

- Are you aware of the diagnostic criteria for ARDS and when to have a high degree of clinical suspicion in patients presenting in primary or secondary care?
- Are you aware of the potential longer term complications for patients who develop ARDS? How might you address these in your local setting?
- What might you do differently as a result of reading this article?

How common is ARDS?

The incidence of ARDS is variable (7-70 per 100 000 person years),[5] reflecting in part differences in recognition. The LUNG SAFE study, a prospective observational cohort study (29 000 patients in 459 intensive care units in 50 countries), allowed for retrospective diagnosis of ARDS by researchers using clinical data, independent of the treating clinicians. In that study, more than 10% of patients admitted to intensive care units—and more than 20% of those requiring invasive ventilation—had ARDS.[2]

How is it diagnosed?

ARDS should be suspected in all patients presenting to primary care or the emergency department with recent onset of severe respiratory symptoms and with clinical signs of hypoxia (fig 1).

ARDS can be anticipated where a risk factor is present (eg, pneumonia, sepsis, aspiration of gastric contents, massive blood transfusion).

- *Clinical features*: respiratory symptoms and signs (elevated respiratory rate, lung crackles on auscultation); clinical signs of hypoxia (central cyanosis).
- *Investigations*: these clinical features mandate a chest radiograph and an arterial blood gas analysis.

The chest radiograph should show diffuse opacities over both lung fields (fig 2). The radiological criteria from the diagnostic definition of ARDS states that the chest radiograph findings show "bilateral opacities that are not fully explained by effusions, lobar/lung collapse, or nodules." The arterial blood gas analysis will show low arterial oxygen tension—ie, arterial hypoxaemia. As oxygen tension is dependent on inspired oxygen concentration, the ratio of arterial oxygen tension to inspired oxygen fraction is calculated. If this ratio is less than 40 (oxygen tension measured in kPa), then the oxygenation criterion for ARDS is fulfilled.

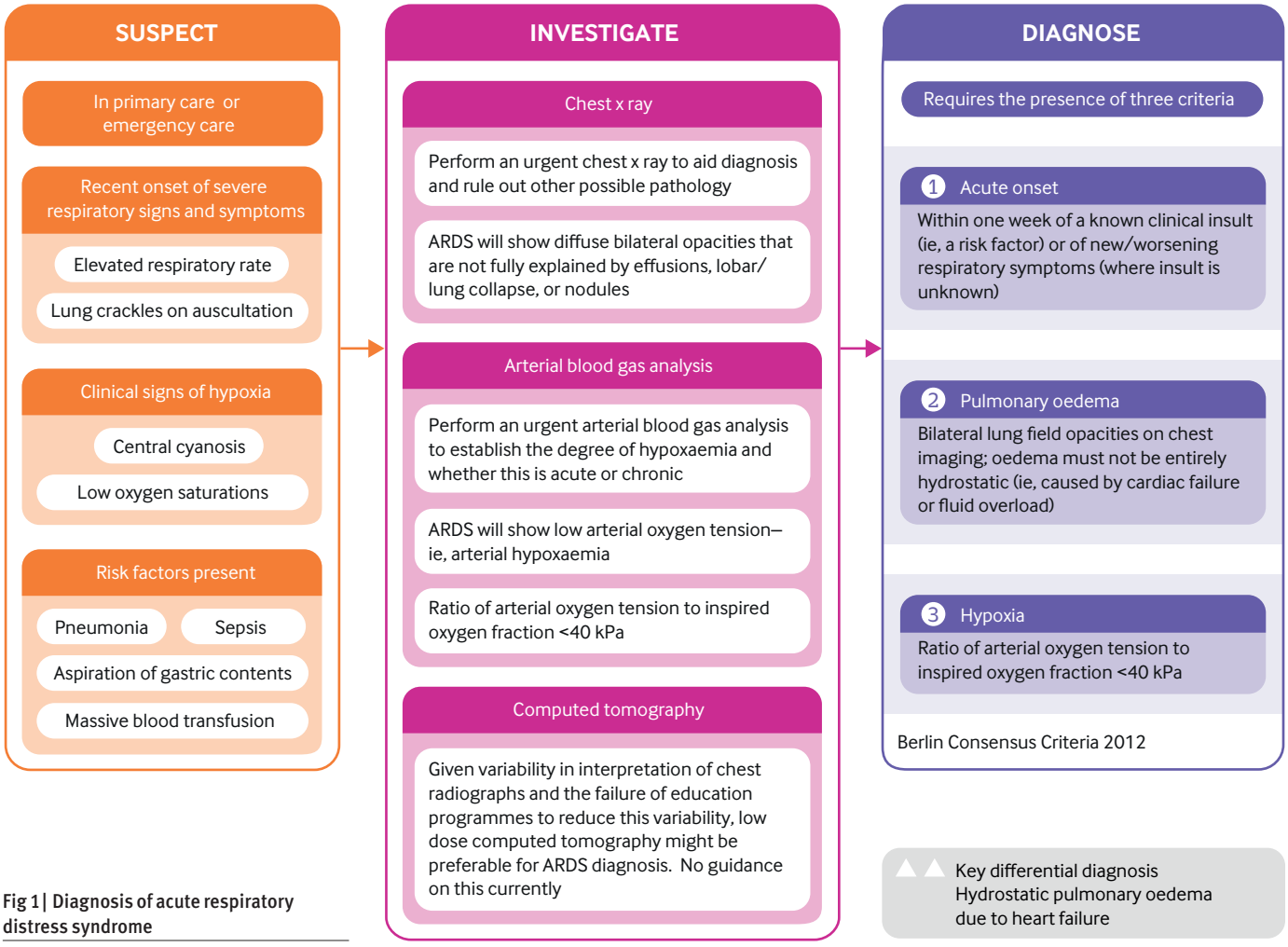


Fig 1| Diagnosis of acute respiratory distress syndrome

Diagnosis

The diagnostic criteria for ARDS have evolved since its first description in 1967, with the most recent criteria developed by a panel of experts following a consensus conference in Berlin in 2012 (convened by the European Society of Intensive Care Medicine, with the endorsement of the American Thoracic Society and the Society of Critical Care Medicine). The diagnosis of ARDS[6] requires the presence of three criteria:

- Acute onset: within one week of a known clinical insult (ie, a risk factor) or of new/worsening respiratory symptoms (where insult is unknown)
- Pulmonary oedema: bilateral lung field opacities on imaging; oedema must not be entirely hydrostatic (ie, caused by cardiac failure or fluid overload)
- Hypoxia: ratio of arterial oxygen tension to inspired oxygen concentration <40 kPa.

Cardiogenic pulmonary oedema is the main differential diagnosis, so when there is no clear predisposing cause for ARDS, patients need to be evaluated for heart failure. As both congestive heart failure and ARDS can coexist, the diagnosis of ARDS can still be made, as long as congestive heart failure is not the sole apparent cause of the hypoxia and chest radiograph findings.

What is the evidence that ARDS is missed?

The LUNG SAFE study reported that 40% of cases of ARDS were not recognised at any time during a patient's stay in the intensive care unit.[2] Delayed diagnosis was the norm, with <30% diagnosed on the first day that criteria were present.[2] Although this evidence is new and compelling, the issue is not new. A decade old study of ARDS proved through autopsy noted that <50% of cases were identified in the clinical notes,[7] while in a 2013 study <30% of patients with all criteria for ARDS had the condition recorded in their notes.[8]

Why is the diagnosis of ARDS usually missed?

Evolving illness in a complex environment

While the individual criteria are simple, the diagnosis relies on recognising patterns in patients with evolving illness and receiving complex care. Recognition is poor where doctor and/or nurse to patient ratio is low,[2] and, in contrast, is higher when attention is focused (eg, younger patients with single organ failure or more severe hypoxaemia).[2] The clinician might be caring for several patients with complex conditions, and information overload—pervasive in intensive care units[9 10]—occurs. Even experienced clinicians cannot process extreme volumes of information.[11] Thus, recognition might be delayed or missed.

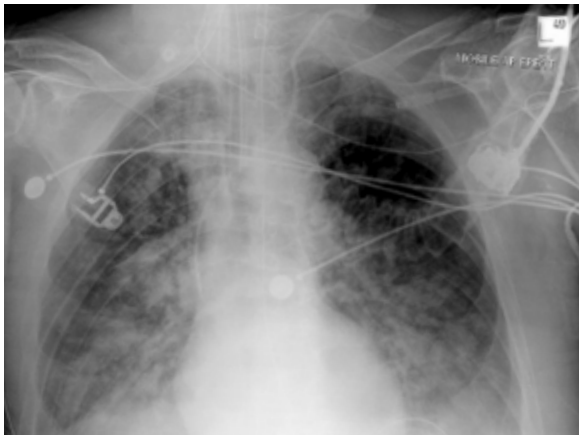


Fig 2 | Classic chest radiograph from a patient with acute respiratory distress syndrome, showing bilateral airspace opacities diffusely spread over both lung fields. (image provided courtesy of Frank Gaillard, Radiopaedia.org, rID: 35985)

Assumption of rarity

ARDS is incorrectly considered to be rare, especially by clinicians less familiar with intensive care units, who might even consider it restricted to the intensive care unit. The diagnosis requires chest imaging and arterial blood gas analysis; therefore ARDS can be suspected, but generally not confirmed, in the primary care setting. If the index of suspicion is low, the diagnosis will be missed even in high risk patients, and where fewer risk factors exist, the risk of missed diagnosis is increased.[2]

Misinterpretation of chest radiograph

The utility of chest radiography in ARDS can be poor, and substantial inter-observer variation has been documented.[12]

Limitations of ARDS consensus definition

The high sensitivity and low specificity of the ARDS consensus definition (sensitivity 89% and specificity 63% compared with histologic criteria)[13] is problematic; it is better for screening than for diagnosis, and clinicians might therefore take "positive criteria" less seriously.

Why does this matter?

Delayed or failed recognition of ARDS leads to delayed or non-implementation of beneficial treatment. Under-recognition is linked to under treatment.[2] Fewer than 50% of those with ARDS who died had received muscle relaxation, and <20% had a trial of prone positioning: two interventions with proven survival advantage.[14 15] In contrast, patients in whom ARDS was recognised were more likely to receive these interventions.

Early recognition in the community, in the emergency department, or on the ward can facilitate measures that increase the odds of survival (with fewer complications). Strategies to reduce iatrogenic harm include avoidance of excess intravenous fluid,[16] avoidance of high tidal volume[17] (breath size delivered by the mechanical ventilator), or the use of prone positioning,[18] and transient muscle relaxation.[15 19] Because high tidal volume is more injurious if used earlier, this underscores the need for early recognition.[4] These interventions are relatively simple in an acute care setting, easy to implement, and have excellent benefit/risk profiles.

Failure to recognise ARDS leads to failure to use proven treatments, and this translates into higher chances of death, worse quality of life (because of cognitive impairment, muscle wasting, and functional limitation[20]) among those who survive. These disabilities persist, with survivors of ARDS experiencing substantial limitations in physical function five years after their critical illness.[21] Only 48% had returned to work at one year, which increased to 77% by the end of year 5. Over half of ARDS survivors reported at least one episode of physician diagnosed depression, post-traumatic stress disorder, or anxiety in the five years after ARDS. In another study, 24% of survivors of critical illness showed impairment of cognitive function at 12 months similar to that seen with mild Alzheimer's disease.[22] Survivors require extensive rehabilitation in the community after ARDS, and guidelines have been developed by the National Institute for Health and Care Excellence in relation to this.[23]

How can we improve diagnosis of ARDS?

Simple steps could improve recognition. Increased awareness (clinicians, patients, relatives) elevates the index of suspicion and thus the likelihood of diagnosis. In one retrospective, single centre study, introduction of an ARDS standard operating procedure increased awareness of ARDS, leading to an increased frequency of ARDS diagnosis ($P<0.05$), increased application of early prone positioning ($P<0.05$), and use of neuromuscular blockers ($P<0.02$) in ARDS patients.[24] As >20% of ventilated patients in intensive care units have ARDS, it should be considered in any sick patient with respiratory distress—in the community, emergency department, or hospital ward.

Given variability in interpretation of chest radiographs[12] and the failure of education programmes to reduce this variability,[25] low dose computed tomography might be preferable for ARDS diagnosis.[26]

The discovery of biomarkers might help, but, given the high sensitivity of the consensus criteria, additional markers might be superfluous for detection—but could be of great use in confirmation (ie, to reduce "false positives").

How is ARDS managed?

Management of ARDS involves three complementary strategies.

- Measures are needed to sustain life; in particular, advanced support of oxygenation and organ function is required
- Underlying causes must be addressed (eg, antibiotic treatment and source control for sepsis)
- Hospital acquired harm must be prevented (eg, minimising lung injury caused by mechanical ventilation, avoidance of fluid overload).

In patients with more severe ARDS, early use of muscle relaxation[15] and prone positioning[18] can further improve outcome, and in rare cases, extracorporeal membrane oxygenation can be life saving in severely hypoxaemic cases unresponsive to conventional support.

HOW PATIENTS WERE INVOLVED IN THIS ARTICLE

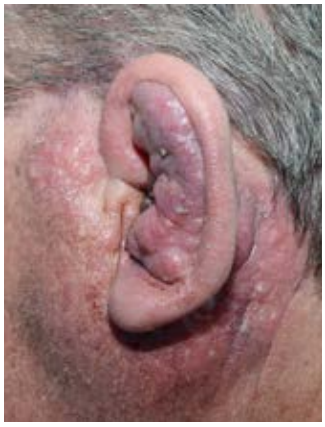
One of the authors (CM) is a survivor of sepsis and ARDS, and has published articles on her experience. She was consulted after the initial draft and edited the subsequent versions.

Competing interests: None declared.

Cite this as: *BMJ* 2017;359:j5055

Find the full version with references at http://dx.doi.org/10.1136/bmj.j5055

CASE REVIEW
The swollen pinna



A 65 year old man presented to his general practitioner with a one month history of pain and swelling of his left ear. His only comorbidity was a renal transplant 14 years earlier for immunoglobulin A nephropathy. His symptoms started with a small spot in the left external auditory canal and worsened after ear syringing. He was initially treated with two courses of oral antibiotics (co-amoxiclav). His symptoms failed to improve and he was admitted to hospital for intravenous antibiotics (tazocin and framycetin/gramicidin ear drops). On examination, the pinna was erythematous, warm, and swollen, without evidence of a discrete pus collection. Palpation of the neck revealed firm pre and post auricular nodes. He had no joint pains, respiratory, or eye symptoms, and was not diabetic. His inflammatory markers were not raised and he was discharged after 24 hours of intravenous antibiotics. The symptoms persisted and, after a further course of oral antibiotics in the community, he was again admitted (with increasing pain). The pinna was more painful, swollen, and erythematous on this admission (fig 1). He was managed with intravenous antibiotics, microsuction, and insertion of a pope wick. Swabs revealed no growth, and the appearance of the pinna did not improve with antibiotic treatment.

1 What are the differential diagnoses?
2 How should you investigate this patient's symptoms?
3 How will you manage this case?

Submitted by E Warner, C Weston, N Barclay-Klingle, and R Corbridge

Patient consent obtained.

Cite this as: *BMJ* 2017;359:j5073

If you would like to write a Case Review for Endgames, please see our author guidelines at http://bit.ly/29HCBAL and submit online at http://bit.ly/29yyGSx

SPOT DIAGNOSIS
A man with knee pain after a fall

A 47 year old man presented with a painful swollen right knee after a fall down stairs. On examination, his right knee was swollen, and there was infrapatellar tenderness, a palpable defect inferior to the patella, and a large effusion on palpation. He was unable to raise his leg or extend his knee. A radiograph of the knee was taken (fig 1). What sign can be seen on this radiograph and what diagnosis is it suggestive of?

Submitted by Tobenna J Oputa and Ronnie Davies

Patient consent obtained.

Cite this as: *BMJ* 2017;359:j5053

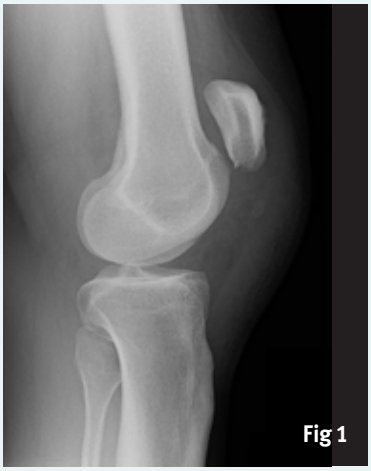


Fig 1

answers

CASE REVIEW
The swollen pinna

1 Perichondritis, relapsing perichondritis, necrotising otitis externa, malignancy (eg, Merkel's cell carcinoma).
2 Full blood count, urea and electrolytes, erythrocyte sedimentation rate, glycated haemoglobin, autoimmune screen, HIV, hepatitis screen, swab for microscopy, culture, and sensitivity; computed tomography and magnetic resonance imaging for bony and soft tissue anatomy.
3 For skin lesions, the treatment is primarily surgical if the lesion is resectable, with the option of further resection or radiotherapy where margins are close or the lesion is very large and extensive.

SPOT DIAGNOSIS
A man with knee pain after a fall

Patella alta (high riding patella) suggestive of a patellar tendon rupture (fig 2).

The patella is displaced superiorly by the unopposed pull of the quadriceps femoris (Q). The ratio between the patellar tendon length (TL) and the patella length (PL) is the Insall-Salvati ratio

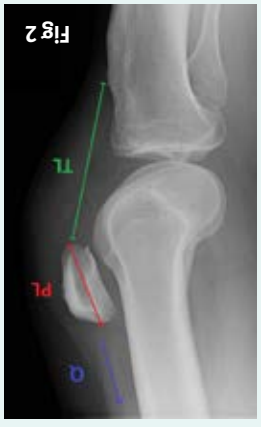


Fig 2

CPD READING
0.5 HOURS

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

Methotrexate induced labial haemorrhagic erosions

An 82 year old woman, who had been treated with valaciclovir for five days for a presumed diagnosis of labial herpes, was referred with a three day history of epithelial detachment with bloody crusts on the lips (figure) and macrocytic anaemia (haemoglobin level, 6.5 g/dL). No other skin or mucosal surfaces were involved. She had been receiving methotrexate (8 mg/week) and folic acid (5 mg/week) as treatment for rheumatoid arthritis for the preceding five months.

The methotrexate was suspended, and intravenous calcium folinate was administered at 36 mg/day. The labial erosions epithelialised in 10 days, and the anaemia improved in three weeks. Sulfasalazine and prednisolone were initiated in place of methotrexate.

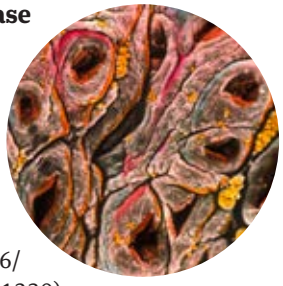
Mild mucositis and oral ulceration are well recognised adverse reactions with methotrexate treatment, but severe labial erosions such as this are rare. Adverse reactions to methotrexate are more common in patients with advanced age, renal failure, drug interactions, or folate dependent enzyme polymorphisms.

Yuka Maya, Mitsuhito Ota (ota@med.hokudai.ac.jp), Department of Dermatology, Chitose City Hospital, Chitose, Japan

Patient consent obtained.

Cite this as: *BMJ* 2017;359:j5063

Early growth in children with coeliac disease



A cohort study in Norway measured the growth of nearly 60 000 children on six occasions during the first two years of their lives (*Arch Dis Child* doi:10.1136/archdischild-2016-31230). This allowed investigators to look back at the early development of the 440 children who were later diagnosed with coeliac disease. They found that differences in growth rates emerged long before any gastrointestinal symptoms became apparent. As a group, the children with coeliac disease had been shorter from 12 months old and lighter from 18 months.

Psychological stress declines rapidly after middle age

Up to the age of 50, nearly half of American adults say yes if asked whether they experienced stress for a lot of the day yesterday. After 50 however, the proportion drops sharply, and, by the age of 75, only 1 in 5 responds positively. The pattern is unaltered by taking account of factors such as employment, social support, marital status, health conditions, health insurance, and church attendance (*J Psychosomat Res* doi:10.1016/j.jpsychores.2017.09.016). It seems that, regardless of their social situation and whether they are well or ill, Americans report stress much less frequently from middle age onwards.

Chronic widespread pain

Data from UK Biobank, a longitudinal study of half a million adults, show that mortality among people with chronic widespread pain syndromes is double that of people without chronic pain (*Ann Rheum Dis* doi:10.1136/annrheumdis-2017-211476). Deaths from cancer, cardiovascular, and respiratory diseases were all commoner in people who had reported "pain all over the body." However, adjustment for low levels of physical activity, high body mass index, poor quality diet, and smoking substantially reduced the excess risk. Doctors often find it hard to help patients with chronic widespread pain but it looks as if encouraging a healthier way of life would be worthwhile.

Best practice after cardiac arrest

National and international guidelines for advanced life support were updated in 2015. A survey of English National Health Service acute hospital trusts finds that the recommendations have been taken up variably and incompletely (*Postgrad Med J* doi:10.1136/postgradmedj-2016-134732). Waveform capnography and ultrasound were often unavailable and post-resuscitation debriefing occurred at only a few trusts. On the other hand, most hospitals were taking part in quality improvement strategies such as the National Cardiac Arrest Audit.

Reporting of interventions for patellofemoral pain

A recent Cochrane review found strong evidence that exercise therapy was effective for people complaining of patellofemoral pain (*Br J Sports Med* doi:10.1136/bjsports-2017-097547). It reduced severity of pain in both the short and long term, and improved function. But a review of the quality of reporting discovered that not a single study gave enough detail about their exercise programme to allow full replication. How can anyone implement exercise therapy for patellofemoral pain if they can't find out what it is?

Progress in acute myocardial infarction

In 1955 the US president Dwight Eisenhower complained of indigestion while playing golf. The next day, an electrocardiogram revealed an anterolateral infarction with ST segment elevation. At that time, no one knew that aspirin inhibited platelet aggregation, defibrillators had still to be invented, and beta blockers and statins weren't even twinkles in a pharmacologist's eye. However, it's probably prompt revascularisation that has made the biggest difference to outcomes after acute coronary events. SWEDEHEART registry data show that mortality almost halved between 1995 and 2014 (*Eur Heart J* doi:10.1093/eurheartj/ehx569).

Cite this as: *BMJ* 2017;359:j5196