education

FROM THE JOURNALS Edited highlights of weekly research reviews on https://bit.ly/2PLtil8

Adverse effects of methotrexate

Some drugs get an erroneous reputation for causing adverse effects. Methotrexate is a case in point because it is used to treat diseases which have myriad multisystem manifestations. The only way to know which events are attributable to methotrexate is from a double blind randomised controlled trial. Solomon and colleagues analysed 4786 participants without rheumatic disease who were randomised to methotrexate or placebo.

Active treatment was associated with moderate increases in rates of gastrointestinal, pulmonary, and haematological events with hazard ratios of 1.91, 1.52, and 1.15, respectively. The participants were quite a specific population of people with cardiovascular disease (in particularly 81% were male), but these data still provide a valuable insight into the true rates of adverse effects of methotrexate.

Ann Intern Med doi:10.7326/M19-3369

Lifting the polygenic cloud

Polygenic risk scores for coronary artery disease have turned out to have no incremental value beyond cohort equations (that is, existing risk calculators). Mosley and colleagues assessed the predictive accuracy of a polygenic predictor including millions of common mutations in two US (white, middle aged) cohorts and found it didn't add much.

Elliott and colleagues did essentially the same using the UK Biobank (middle aged and mostly European ancestry) and found a "modest improvement" in predictive accuracy that was statistically significant but which practically did not change most participants' predicted probabilities of coronary artery disease. Taken together, these studies suggest that genetic information does not add much in risk prediction and thus polygenic risk scores aren't clinically relevant in coronary artery disease risk prediction.

JAMA doi:10.1001/jama.2019.21782

JAMA doi:10.1001/jama.2019.22241

Making headway in a cure for HIV?

The RIVER trial, set at six sites in the UK, is a landmark randomised controlled trial in HIV infection. It is the first to test the effect of a "kick and kill" therapy on markers of the HIV reservoir. The existence of a reservoir of latent HIV is what has so far made it impossible to cure. You want to "kick" the latent HIV virus into expressing proteins, which can then be targets for the immune system, and provide antiretroviral drugs to "kill." The HIV reservoir was measured by number of HIV DNA copies from peripheral blood CD4+ T cells at 16 and 18 weeks after randomisation. Unfortunately the kick and kill therapy did not demonstrate any effect on the HIV reservoir markers in this 60 patient study. This is despite evidence that the kick had an effect and the kill had an effect. They just didn't result in the overall desired benefit. Maybe the dosing was wrong. Maybe some patients were resistant to this type of treatment. Maybe the stage of the disease in these patients was wrong—that is, these were patients with a recent diagnosis rather than chronic HIV infection, which may have quite different susceptibility to this therapy.

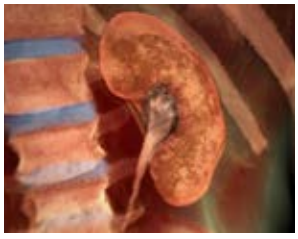
Lancet doi:10.1016/S0140-6736(19)32990-3

A no for no sedation in mechanical ventilation

Olsen and colleagues compared a plan of no sedation against a plan of light sedation with daily interruption in 710 mechanically ventilated patients in intensive care units. In the non-sedation group, mortality at 90 days was 42.4% compared with 37% in the lightly sedated group. There was also no difference in the number of ICU-free days or ventilator-free days. Perhaps the lack of difference could be attributed to the lack of difference in the amount of sedation between the non-sedated and lightly sedated groups, especially since the non-sedated group often ended up crossing over to sedation (27% in the first 24 hours). Nevertheless, I think the take-home message is a "no" for no sedation.

N Engl J Med doi:10.1056/NEJMoa1906759

Prehydration for contrast CT in chronic kidney disease



This Dutch randomised controlled trial compared prehydration with sodium bicarbonate with no prehydration in more than 500 people with stage 3 chronic kidney disease who were undergoing contrast enhanced computed tomography. The main outcome was the change in serum creatinine at two to five days after administration of the contrast medium compared with baseline. There was no difference between the two groups, allowing Timal and colleagues to conclude that it is safe not to prehydrate. I am not sure this is completely definitive because the prehydration fluid comparator was 250 mL of 1.4% sodium bicarbonate, and other prehydration strategies that were not assessed might be effective at reducing post-contrast acute kidney injury.

JAMA Intern Med doi:10.1001/jamainternmed.2019.7428

Alex Nowbar is a clinical research fellow at Imperial College London

Adult flatfoot

Chris Yuk Kwan Tang,[1] Ka Ho Ng,[1] Joyce Lai[2]

[1]Department of Orthopaedics and Traumatology, Queen Mary Hospital, Hong Kong
[2]Department of Family Medicine and Primary Care, Queen Mary Hospital, Hong Kong
Correspondence to: CYK Tang medic.chris.tang@gmail.com

CPD READING
0.5 HOURS

A 45 year old woman who is overweight presents with pain in the medial ankle radiating to the medial foot arch area. She reports a noticeable flattened foot arch.

Flatfoot is a deformity associated with collapse of the medial longitudinal foot arch, valgus deformity of the heel, and abduction of the forefoot. It is commonly encountered in primary care settings. The prevalence of flatfoot is estimated between 3% and 25% in the general population based on studies in healthy volunteers.[1 2] The most common cause in adults is posterior tibial tendon dysfunction.[6] It is associated with obesity, diabetes mellitus, hypertension, trauma, and corticosteroid injections.[7] The symptomatic flatfoot presents with medial arch pain and can affect gait.[8] Untreated symptomatic flatfoot in the long term can become a rigid deformity, which will further affect the patient's quality of life. Despite this, there are few publications or guidance for management of adult flatfoot in primary care. This article aims to give a systematic approach to manage a patient presenting with symptomatic flatfoot.

HOW THIS ARTICLE WAS CREATED

- We used clinical experience gathered from clinical practice with literature review in PubMed.
- Backed up by underlying patho-anatomical knowledge and recent studies regarding the efficacy of non-operative treatment.
- This aligned with published guidelines from the British Orthopaedic Association

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

We invited people at our clinic who had symptomatic flatfeet to be interviewed about the content of this article. They raised the following important points:

- Orthosis is sometimes too rigid to wear initially. We added a paragraph that highlights that orthosis support should be carried out in phases and started with softer materials.
- Instructions for home exercise programmes should be easy to understand and acceptable to patients. Patients found it easier to carry out exercises if the doctor demonstrated them in the clinic. With this in mind, we included in the article photos and instructions for foot exercises, which the healthcare provider can demonstrate to their patients during consultation.

WHAT YOU NEED TO KNOW

- Flatfoot is a deformity associated with the collapse of the medial longitudinal foot arch, valgus deformity of the heel, and abduction of the forefoot
- Patients with symptomatic flatfoot usually present with pain in the medial sided foot arch and ankle
- Offer instructions for home exercise programmes and a referral for foot orthosis for flexible flatfoot. Patients with a rigid flatfoot need referral to an orthopaedic team

What you should cover

History

What and where are the symptoms?

Flatfoot can be a normal physiological variant and many people will be asymptomatic—with decreased medial foot arch but without pain or gait disturbance—and therefore do not require treatment.[9] Where flatfoot is symptomatic, the most common symptoms include pain in the medial sided foot arch and ankle, and progressive flattening of the foot arch.[10] In more advanced cases, lateral foot pain can also be present (fig 1).

What is the impact on daily activities?

Ask the person if they have reduced their walking distance because of foot symptoms and if they have experienced any subjective feeling of imbalance, unsteadiness, or giving way during walking. Assess the impact of these on the person's daily living. Untreated symptomatic flatfoot can become rigid and affect gait mechanics in the long term.

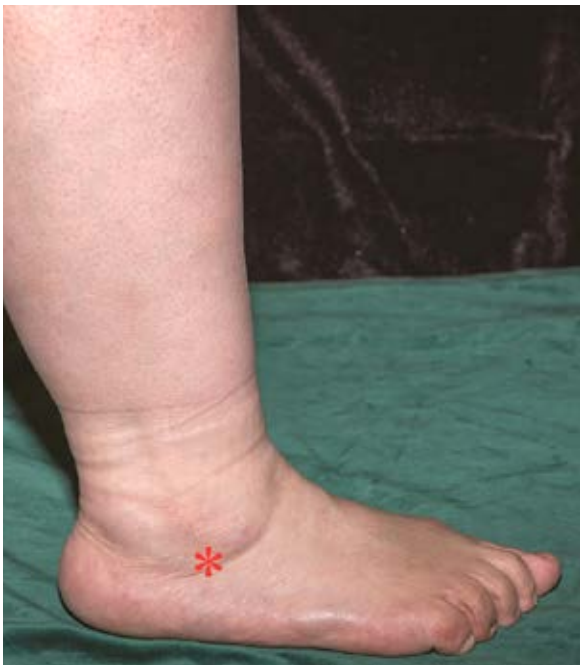


Fig 1 | Right adult flatfoot. The patient may experience lateral pain in the ankle (sinus tarsi, shown by the red asterisk) resulting from subfibular impingement in advanced cases

Physical examination

The purpose of physical examination is to assess whether flatfoot deformity and posterior tibial tendon pathology are present. If flatfoot is present, examination can also help determine whether it is flexible or rigid. A rigid flatfoot requires referral to secondary care and an orthopaedic surgeon.

Look

Assess the foot from three different views

With the patient in a standing position, look for a decrease in height of the foot arch (lateral view), abduction of forefoot (above down view), and valgus deformity of the heel (posterior view) (fig 2). Unilateral flatfoot is more common than bilateral flatfeet in adults,[5] so the alignment of the asymptomatic contralateral foot can be used as a reference in unilateral pathology.

Current literature does not define normal height of the foot arch. Decrease in foot arch is a qualitative and morphological description. Diagnosis of flatfoot does not depend solely on foot arch height, but also change in the deformity and associated symptoms. Inspect for associated forefoot deformity, such as hallux valgus and bunion. Assess the patient's gait pattern and excessive wear on the medial aspect of footwear or shoes. Calculate the patient's body mass index and establish whether they are overweight or obese as these are risk factors for flatfoot.

Feel

Palpate the posterior tibial tendon

Most cases of adult flatfoot are caused by posterior tibial tendon pathology, which is associated with tenderness.[6] Palpate the tibialis posterior tendon to elicit tenderness along its distal course: starting from navicular tubercle (tendon insertion), going proximally posterior to the medial malleolus of the ankle, to the medial distal leg (fig 3).

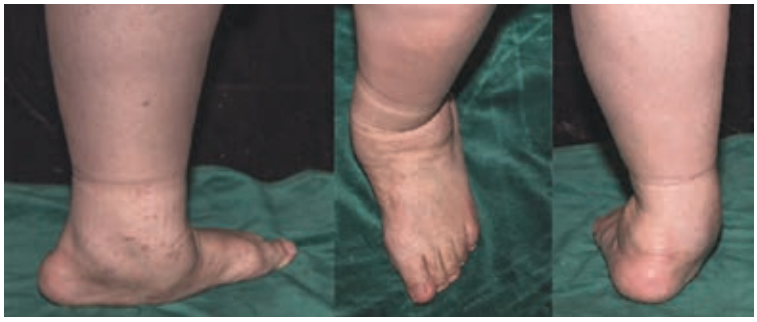


Fig 2| Deformity in flatfoot seen from three views: decrease in height of the foot arch (lateral view), abduction of forefoot (above down view), and valgus deformity of heel (posterior view)

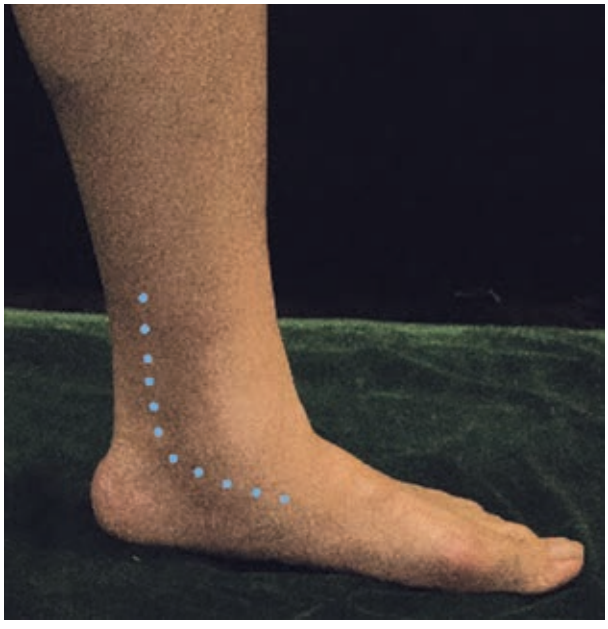


Fig 3| Left adult flatfoot, showing a decrease in foot arch. The course of posterior tibial tendon is illustrated by the blue dotted line

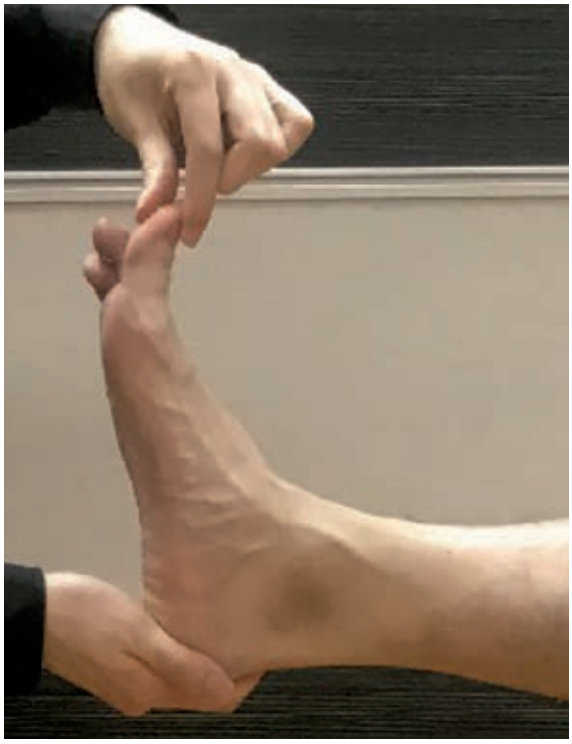


Fig 4| Jack's test: the clinician passively dorsiflexes the great toe, which re-creates the medial arch in a passively correctable foot

Move

Check whether the flatfoot is flexible or rigid

The flexibility of the flatfoot is assessed using Jack's test and single heel rise.

With Jack's test the clinician passively dorsiflexes the great toe, which will re-create the medial arch in a flexible foot (via the windlass mechanism), but not with rigid flatfoot (fig 4).[6]

The heel rise test checks the flexibility of the flatfoot by assessing the mobility of the subtalar joint. Ask the patient to stand on tip toe. In a flexible flat foot the heel goes from valgus to a varus position.

Check whether the hindfoot valgus is mobile or fixed

A fixed hindfoot valgus signifies a more severe deformity than a mobile one. Ask the patient to do a double heel raise and inspect from behind. The hindfoot should passively correct into the varus if the subtalar joint is still mobile (fig 5).

Move the posterior tibial tendon

Check the muscle power of the posterior tibial tendon by resisted inversion with the ankle in plantarflexion with examiner's hand. The examiner should be unable to resist the movement if the muscle power of the posterior tibial tendon is normal. Also, a posterior tibial tendon with normal power should enable a patient to do single leg heel raise without any difficulties. If the power is not normal, it indicates dysfunction of posterior tibial tendon. Home exercise of double leg heel raise is advised to strengthen the posterior tibial tendon.

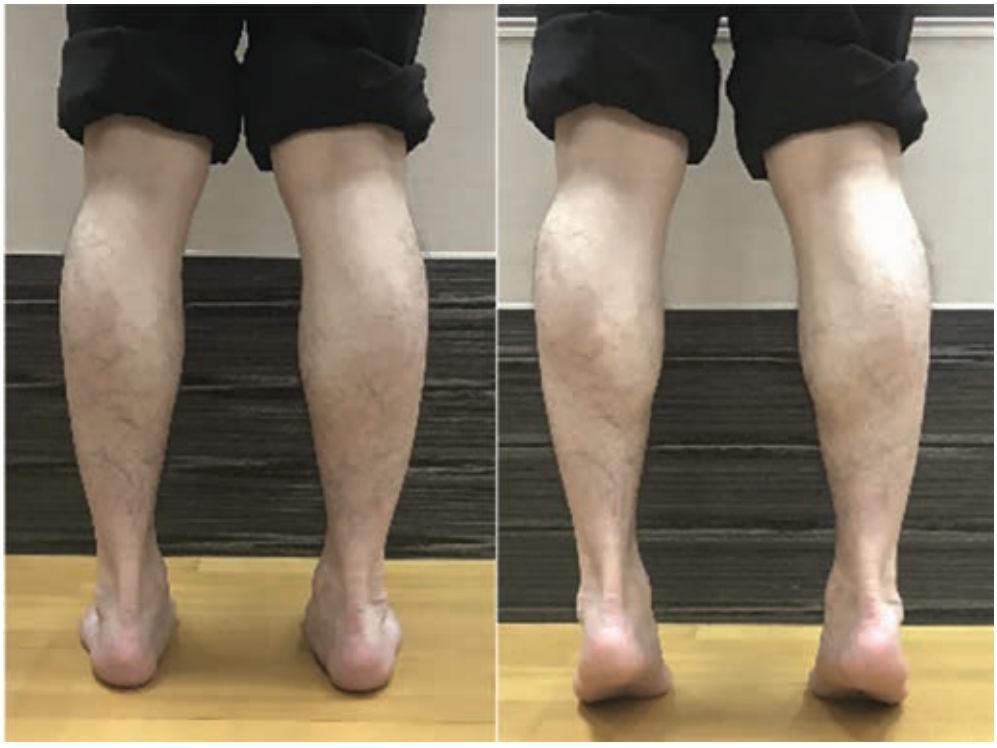


Fig 5 | Double heel raise: note the passive varus correction of the hindfoot during the double heel raise, if the subtalar joint is still mobile

What you should do

Pain in the medial sided arch and ankle with progressive flattening can be treated in primary care and podiatry. Timely (less than two weeks') referral to an orthopaedic foot and ankle surgeon is required in acute deformity, pain after trauma, or in diabetic patients (when the foot collapse was suspected to be caused by Charcot arthropathy).

Explanation

Explain to the patient that the symptoms are caused by loss of normal foot shape, which leads to pressure overloading the medial side of the foot over the arch. Explain that it can be treated with non-surgical options which can decrease symptoms, but these won't restore the decreased medial arch height.[13] There are surgical options available which have a longer recovery time but can correct the deformity.

Advice

Offer advice on measures to prevent further overloading: avoid wearing poorly fitted shoes and walking barefoot. Advise and support the patient with weight reduction if they are overweight or obese.

Investigation

Weight bearing radiograph of the foot is used to exclude the possibility of

- stress fracture of the tarsal bones
- subtle Lisfranc's injury (tarsometatarsal joint injuries) after a foot sprain
- Charcot neuroarthropathy in people with diabetes, or
- osteonecrosis of the navicular bone in an adult (Mueller-Weiss syndrome).

Treatment

Treatment is either conservative or surgical.[9]

Non-surgical (conservative) treatment

Symptomatic flatfoot can be successfully treated non-surgically in up to 87.5% of cases.[14] Home exercise programmes followed by foot orthosis prescribed in primary care are effective treatments.[15-18]

Instruction for home exercise programmes

Home exercises aim to maintain joint mobility and recondition and strengthen the posterior tibial tendon. Randomised controlled studies showed that posterior tibial strengthening and heel cord stretching improved pain for mild flatfoot.[15 16]

Home exercises (appendix, see bmj.com) include

- Hindfoot inversion
- Double leg heel raise
- Foot arch exercises (active toe dorsiflexion)
- Heel cord stretch if the gastrocnemius is tight.

A series of three sets of 15 repetitions should be performed twice daily on the involved side, with rest for one or two minutes between sets.[15]

Offer foot orthosis

Foot orthosis lifts the medial arch to reduce the stress on the posterior tibial tendon. The orthosis is best fitted by a podiatrist or orthotist and helps reduce the deforming force in a flexible flatfoot and prevents progression of the deformity into a rigid flatfoot.

• For patients with severe symptoms, using a resting ankle splint over four to six weeks to reduce movement while at rest can alleviate the symptoms. A previous study showed that the success rate of this option ranged from 67% to 90%.[6]

Medication

Analgesics and anti-inflammatory drugs can be prescribed for pain relief in flatfoot, but its underlying pathology is a biomechanical issue and should be addressed by other treatment modalities in addition.

Referral to physiotherapy

This has two aims:

1 Strengthening of the posterior tibial muscle complex

2 Stretching of the gastrocsoleus.

Observation and follow-up

Follow up in about three months to check how the patient is managing with the orthosis and exercises.

Surgical treatment

Surgery can include soft tissue and bony procedures to restore the normal biomechanics of the foot and ankle, restore the medial arch height, and alleviate the symptoms. The exact surgical procedure depends on the stage of the flatfoot and will be determined by the orthopaedic specialist. The duration of recovery will depend on the type of surgical procedure performed, but most require a period of non-weight bearing for six to eight weeks.

Consider referral within six to eight weeks to an orthopaedic surgeon for the following situations:

- Patient with rigid foot deformity
- Patient with flexible foot deformity that is progressive while on non-operative treatment for six months
- Patient's activities of daily living are severely affected by gait disturbance, irrespective of whether the deformity is flexible or rigid
- Patient with a history of trauma or diabetes and a new onset of flatfoot deformity requires urgent assessment within two weeks.

Prevention

Foot exercises done at home are recommended for symptomatic flatfoot. The purpose is to maintain joint mobility and avoid progression into rigid flatfoot.

Competing interests: None declared.

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EDUCATION INTO PRACTICE

- What diagnoses might you consider in a patient who presents with medial ankle pain and flatfoot?
- What other symptoms related to flatfoot would you bear in mind?
- How would you explain the diagnosis to a patient presenting with flatfoot?
- What is the process of referral for foot orthosis in your local community services?

UNCERTAINTIES

Is streaming patients in emergency departments to primary care services effective and safe?

Alison Cooper,[1] Andrew Carson-Stevens,[1] Thomas Hughes,[2] Adrian Edwards[1]

[1]Division of Population Medicine, School of Medicine, Cardiff University

[2]John Radcliffe Hospital, Oxford

Correspondence to: A Cooper CooperA8@cardiff.ac.uk

CPD READING 0.5 HOURS

This is one of a series of occasional articles that highlight areas of practice where management lacks convincing supporting evidence. The series advisers are Sera Tort, clinical editor, Nai Ming Lai, clinical editor, and David Tovey, editor in chief, the Cochrane Library. You can read more about how to prepare and submit an Education article on our Instructions for Authors pages:https://www.bmj.com/about-bmj/resources-authors/article-types

Between 10% and 43% of patients presenting to emergency departments can be managed in primary care, according to estimates from observational studies.[1-4] Increasing demands on emergency healthcare systems have led to the development of different healthcare models, including "streaming" patients presenting with non-urgent conditions to primary care services.[5] These strategies are intended to improve patient flow and reduce crowding in the emergency department. In 2017, NHS England made substantial investments for all emergency departments to have co-located primary care facilities so they are "free to care for the sickest patients."[6 7]

However, the evidence for this initiative is weak.[8-12] Different service models are described in different contexts using ambiguous terminology. A "co-located" primary care service may deliver patient care in a separate unit to the emergency department, without access to acute diagnostics, thus similar to normal general practice settings. Alternatively, general practitioners may work within the emergency department, with responsibilities beyond usual primary care.[13] It is uncertain if national implementation of streaming, considering the heterogeneous nature of emergency medicine case mix and practice, improves patient care and safety.

WHAT YOU NEED TO KNOW

- Co-located primary care services have been introduced in emergency departments in the UK to reduce crowding and improve patient care and safety
- There is limited, outdated evidence to show whether streaming emergency department patients to primary care services improves patient flow and reduces costs; and evidence is lacking for patient safety outcomes
- Commissioners and service providers should consider whether governance systems are clear and reflect whether general practitioners in emergency departments are encouraged to function more as primary care or emergency medicine clinicians to suit the local patient demographic profile, demand patterns, and staff recruitment needs

Summary of evidence for effectiveness and safety of primary care services co-located with emergency departments

Review	Published	Included studies	Intervention	Quality of evidence	Evidence of effectiveness	Evidence of safety
Goncalves et al.[8] (updated Khangura 2012[9]) Cochrane review	2018	1 non-randomised UK study (4641 patients) 2 non-randomised Irish studies (1878+4684 patients)	GPs providing care for non-urgent patients in the ED	Very low certainty evidence High heterogeneity across studies precluded pooling data	Uncertain if GPs reduce time to clinical assessment and ED length of stay, admission to hospital, or referral to hospital based specialists, use of diagnostic tests, or costs	No data were reported on adverse events (such as ED returns and mortality)
		1 randomised Australian trial (258 patients)	Standard ED medical care *v* emergency NP care			
NICE assessment[10]	2017	2 non-randomised UK studies (4641+1996 patients)	GPs providing care for non-urgent patients in the ED	Very low quality due to risk of bias	GPs may provide benefit in reduced number of diagnostic investigations. No effect on patient satisfaction observed. No relevant economic evaluations identified	No evidence found for mortality, quality of life, time to admission/ discharge, avoidable adverse events, readmission
Ramlakhan et al.[11] Narrative review	2016	20 primary studies from the Netherlands (n=8), England (n=4), others were from Australia, Ireland, Spain, Sweden, and Switzerland	Primary care professionals managing non-urgent ED patients	All evidence included to search for explanations loosely based on a realist approach. No formal individual study quality assessment	A paradoxical increase in attendances described, likely to be attributable to provider induced demand. The evidence for improved throughput is poor. Marginal savings may be realised per patient, but this is likely to be overshadowed by the overall cost of introducing a new service	No increase in patient reattendance described in two studies
Cooper et al.[12] Rapid Realist Review	2019	96 articles, largely primary research studies, most from the UK (n=44), Netherlands (n=17), others were from Ireland, Belgium, Switzerland, Sweden, Italy, Finland, Australia, USA, Canada, Singapore, and New Zealand	Mostly GPs seeing non-urgent patients in the emergency department	Extracts included that offered explanatory power why and how the services worked. No formal individual study quality assessment	The effectiveness of emergency department streaming to primary care services may be influenced by how staff interpret the streaming system and the roles GPs adopt. Little evidence that GPs directly or indirectly affected the care and throughput of the sickest patients	Minimal data on the safety implications of GPs working in EDs. Five studies showed no increase in reattendance rates and a Dutch study showed no increase in mortality rates

NICE=National Institute for Health and Care Excellence; GP=general practitioner; NP=nurse practitioner; ED=emergency department

What is the evidence of uncertainty?

There are few large scale evaluations of healthcare delivery models offering co-located primary care services. Studies have heterogeneous designs, making it difficult to draw conclusions on safety and effectiveness. Table 1 describes findings from reviews.[8-12] A Cochrane review found few studies on effects of general practitioners seeing non-urgent patients in hospital emergency departments. The results were inconsistent and highlighted a lack of evidence for effectiveness outcomes, with no data available for mortality or safety events.[8]

A narrative review described an increase in attendances at emergency departments with co-located primary care services that was attributed to the service creating its own demand—provider induced demand. There was little evidence on improved patient flow or costs to recommend this model.[11] A rapid realist review found little evidence that general practitioner services in emergency departments influenced the care and throughput of the sickest patients, with other factors, including delayed patient transfers to wards and inadequate staffing, also contributing.[12] Factors such as how staff interpret the streaming system and the roles adopted by general practitioners (whether they function in their traditional role or adopt an emergency medicine approach) could influence effectiveness of service models.

Given the limited, outdated clinical and cost-effectiveness evidence and concerns about the feasibility of staffing the workforce, the National Institute for Health and Care Excellence (NICE) chose not to make a recommendation for general practitioners to work within or on the same site as emergency departments, and called for further research in this area.

NHS England hospital episode statistics data show increasing numbers of hospital admissions from emergency departments rather than general practices (fig 1). Should interventions in emergency departments, which may include the expertise of general practitioners focussing on specific patient groups, prioritise preventing these admissions rather than treating patients with non-urgent conditions?

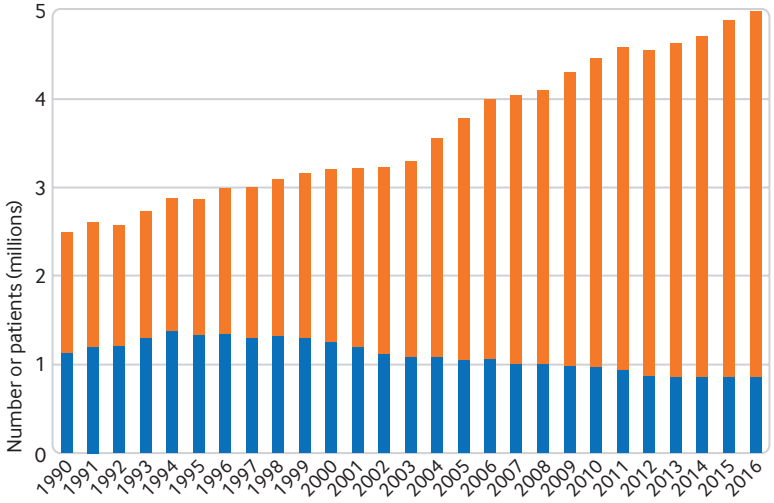

Fig 1 | UK National Health Service hospital episode statistics showing emergency admissions from emergency departments (orange) and general practice (blue). Source: NHS Digital, 2017.

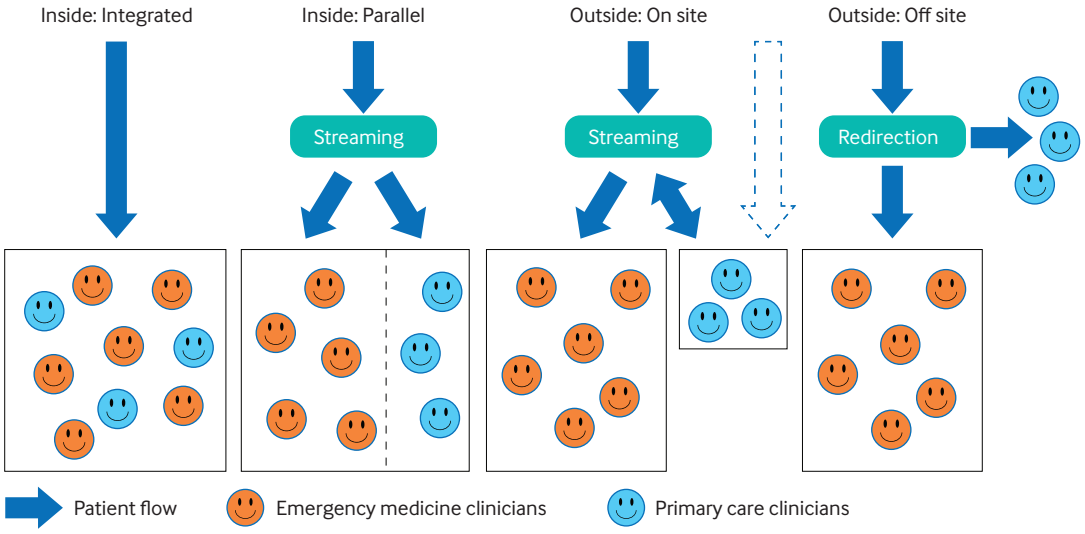

Fig 2| The form of primary care service models in or alongside emergency departments. Adapted from *Emerg Med J* 2019;36:625-30

Is ongoing research likely to provide relevant evidence?

We searched EU Clinical Trials Register, ISRCTN Registry, ClinicalTrials.gov, and ICTRP, and identified a Belgian study on effectiveness of a triage system for patients presenting to emergency departments with referral to primary care.[14]

The UK National Institute for Health Research has also commissioned two observational studies (HS&DR Projects 15/145/04 and 15/145/06) to evaluate the effectiveness, safety, patient experience, and system implications of the different models of general practitioners working in or alongside emergency departments.[15 16] Effectiveness will be evaluated by waiting times, admission rates and (re)attendances, patient satisfaction, and cost-consequence analysis, with patient safety incident reports analysed for patient safety outcomes. The teams have collaborated to update the taxonomy, to ensure consistency of terminology and classification of models, and include the wider primary care workforce. Figure 2 shows the form these service models take, inside and outside emergency departments.[13] The studies, due to be completed in 2021, will attempt to identify service models that may be better suited depending on local demographics and contexts.

What should we do in the light of the uncertainty?

Clinicians, service directors, managers, and commissioners should acknowledge the current evidence gap for effectiveness and safety in this area. The function of these service models is complex and influenced by many variables at multiple levels including: wider system determinants (eg patient demand, staffing); department level (eg access to investigations, governance); and individual level (eg experience, skill set and interest).[13]

Emergency department and primary care clinical leads should jointly consider which patients are appropriate for their local primary care service model and whether their model functions as usual primary care or is more integrated with the emergency medicine service. Clarification of governance processes is important. Where these models are operational, staff can reflect on how and why the service works well, for example through the patient safety incident reporting system, and identify what can be learned from this to continue to deliver safe patient care. .

How staff interpret the streaming system may influence effectiveness of service models

RECOMMENDATION FOR FURTHER RESEARCH

- How can individual primary care services at emergency departments be most effective for local needs?
- What are the patient safety implications for primary care services co-located with emergency departments? How can these be mitigated?
- What interventions in emergency departments can safely reduce the number of acute hospital admissions?

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

Patient representatives were involved as public collaborators and co-applicants on the NIHR funded study of GPs in emergency departments. They have given feedback on findings from the realist review and on taxonomy. No patients were directly involved in writing or reviewing this article.

EDUCATION INTO PRACTICE

- In what ways does your emergency department primary care service meet the needs of the local population and local context?
- What data do you already have to support this assessment?
- What data do you need to identify how to improve the service?

Competing Interests AC, AC-S, TH and AE are all co-applicants on the NIHR HS&DR study Project: 15/145/04—A realist evaluation of effectiveness, safety, patient experience and system implications of different models of using GPs in or alongside Emergency Departments.

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Find the full version with references at http://dx.doi.org/10.1136/bmj.m462

WHAT YOUR PATIENT IS THINKING

How can I explain my high blood pressure to you?

Evelyn Lawrie describes what it is like to live with white coat syndrome and how health professionals can help her manage this

CPD READING
0.5 HOURS

WHAT YOU NEED TO KNOW?

- It might not be possible to reduce the blood pressure readings of a patient with white coat syndrome
- Recording and acknowledging a diagnosis of white coat syndrome on medical records can help reduce patients' anxiety
- Providing distractions, such as music or small talk, for patients with white coat syndrome can help

ROSE LLOYD

For most people, having their blood pressure checked is a relatively straightforward process, but for me, living with white coat syndrome, it is more complicated. The syndrome feels like an uncontrollable physical or mental reaction when I am faced with medical professionals. Many people laugh when I try to explain my condition or think I am making it up, but it has caused me many challenges throughout my life.

A shock diagnosis

I had never felt anxious or worried about appointments—whether planned procedures or general check-ups; that is, until my blood pressure was checked for the first time. As soon as I was attached to the monitor my blood pressure went out of control. Initially I was given many different drugs to treat my unexplained high blood pressure but nothing helped. When my GP asked me to record my blood pressure at home for a month, the results showed that I had white coat syndrome.

I had never heard of this diagnosis before and it came as a shock. I consider myself a calm person and I have never felt worried at appointments. It was a relief to have an explanation for my blood pressure readings, but I worried about how to explain the diagnosis to health professionals and whether the condition would cause problems in the future.

Putting my diagnosis to the test

During a hospital visit for a routine procedure, my diagnosis was put to the test. As soon as the blood pressure monitor started to beep, my stomach muscles tightened. The healthcare team

Knowing the test was being repeated increased my worry

said I seemed to be fit and well—how was I going to explain about my high blood pressure readings? With the monitor attached and beeping uncontrollably, I blurted out my diagnosis. They saw my panic and reassured me that they would try again later.

Knowing the test was being repeated increased my worry because I knew the reading would still be high. Would the procedure be cancelled because of the high readings? I was becoming anxious. One of the team tried to reassure me, singing and dancing in front of my bed. Although it reduced my worry, the readings didn't improve. Fortunately, as I was being prepared for the procedure my blood pressure wasn't mentioned again and I was taken into surgery.

Reducing the worry

I still feel apprehensive about encounters with health professionals. I haven't found anything that stops my blood pressure from increasing around them, but doing my readings at home helps. Because of my high readings I worry that procedures might be cancelled, or that I might be put on a cardiac ward. I still struggle to know how to tell health professionals the reason for my high blood pressure. The most reassuring thing for me is that the diagnosis is on my medical records. This doesn't stop my blood pressure from increasing, but it does mean that I won't have to initiate this difficult conversation in the future.

Competing interests: None.
Provenance and peer review: Commissioned, based on an idea from the author; not externally peer reviewed.
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EDUCATION INTO PRACTICE

- What would you do, in your practice, to support and manage a patient with white coat syndrome?
- How else could you obtain a patient's blood pressure if you suspect they have white coat syndrome?
- How might you help a patient with white coat syndrome feel more comfortable during an appointment?

SPOT DIAGNOSIS **New onset weakness in the face and arm**

A 32 year old woman with a seven year history of discoid lupus erythematosus (a form of cutaneous lupus erythematosus) was admitted with one week of anisocoria (unequal pupil size), left sided facial weakness, and weakness in her left arm. She had areas of hair loss—a long term sign of her discoid lupus erythematosus—but no other symptoms, infections, toxicological habits, or history of thrombotic events or obstetric complications.

Blood tests results are in the table.

Her anisocoria and weakness of the face and arm prompted further central nervous system investigations:

- Cerebrospinal fluid analysis was normal
- Brain computed tomography scan showed bilateral cortical and subcortical frontal lesions of a potentially ischaemic nature
- Brain magnetic resonance imaging (MRI) was requested to look at the lesions in more detail (figure).

Echocardiogram, doppler ultrasonography of the carotid arteries, electrocardiogram, and screening for cardiac risk factors were all normal.

Renal involvement was ruled out with a 24 hour urine test and serum renal function testing.

What is the most likely diagnosis?

Submitted by Luisa Brites, Marília Rodrigues, Mariana Luis, and Mariana Santiago

Patient consent obtained.

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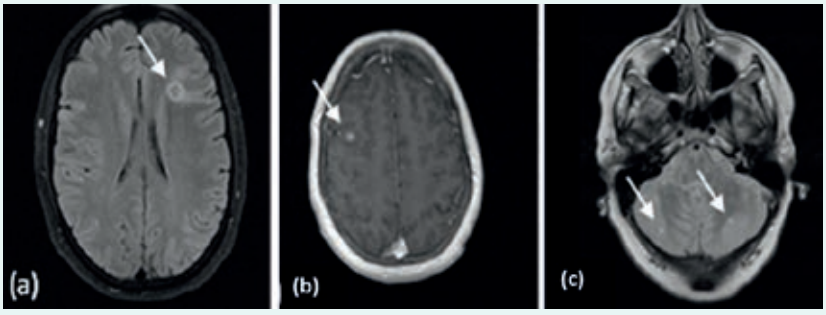
Relevant blood test results

Test	Result	Normal range
Haemoglobin	9 mg/dL	12-16 mg/dL
Platelet count	35 G/L	150-400 G/L
Lymphocyte count	0.9 G/L	1.0-3.0 G/L
Activated partial thromboplastin time	80 seconds	25-34 seconds
Acute-phase reactants	Normal	
Antinuclear antibodies	1:640, dense fine speckled pattern	
Antibodies to double-stranded DNA (anti-dsDNA)	12.7 IU/mL, immunoradiometric assay	<7.0 IU/mL
Lupus anticoagulant	Positive (2.63)	0.9-1.2
IgG anticardiolipin antibodies	High-titre (67.5 U/mL)	<20 U/mL
IgG anti-β2-glycoprotein 1 antibodies	Low-titre (29.9 U/mL)	<20 U/mL
Complement	Normal	
Blood tests for syphilis, human immunodeficiency virus, cytomegalovirus, Epstein Barr virus, rubeola, toxoplasma, brucella, rickettsia, coxiella, hepatitis B and C, tuberculosis	Negative	



Axial fluid attenuated inversion recovery brain magnetic resonance imaging showing (a) a left fronto-opercular lesion; (b) a right frontal lesion with gadolinium enhancement in T1 (c); T2 weighted sequence infratententorial lesions

answers

SPOT DIAGNOSIS **New onset weakness in the face and arm**

What is the most likely diagnosis?

Systemic lupus erythematosus (SLE) with central nervous system vasculitis, a form of neuropsychiatric systemic lupus erythematosus (NPSLE).

This patient met four of 11 clinical criteria for SLE (neurologic involvement, haemolytic anaemia, lymphopenia, and thrombocytopenia) and three of six immunologic criteria (positive antinuclear antibody, anti-dsDNA, and antiphospholipid antibody).[3] SLE is diagnosed when four criteria (at least one clinical and one immunological) are satisfied. Brain biopsy is the gold standard for diagnosing CNS vasculitis but, given the associated risks, SLE-associated CNS vasculitis is usually diagnosed from clinical, laboratorial, and imaging findings.

The distribution pattern of brain lesions on computed tomography and MRI (multiple bilateral lesions in various stages of healing) are suggestive of CNS vasculitis (figure).

NPSLE is diagnosed when a patient has neurological or psychiatric manifestations of SLE (after excluding malignancy, trauma, vascular malformation, hypoglycaemia, and infection). Other causes of cerebrovascular disease were ruled out with echocardiogram, doppler ultrasonography of the carotid arteries, electrocardiogram, and cardiac risk factor screening.

CNS vasculitis is the first manifestation of SLE in up to one third of cases.

People with discoid lupus erythematosus have a 5-10% risk of developing SLE within five years.

LEARNING POINTS

- Cutaneous lupus erythematosus can evolve to SLE, and NPSLE can be the first manifestation.
- Regularly review inflammatory markers of people with cutaneous lupus erythematosus and look for features that make them susceptible to SLE progression, and advise patients to be aware of systemic involvement and disease progression.

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.

LEARNING MODULE

Articles with a "learning module" logo have a linked BMJ Learning module at http://learning.bmj.com.

MINERVA

Spade phalanx sign of acromegaly

This is a radiograph of the spade phalanx sign of acromegaly. The patient was a 32 year old woman with a history of acromegaly, presenting with enlarged digits that were swollen and tender. Radiographs revealed the classic "garden spade" distal phalangeal tuft widening, typical of a patient with excess growth hormone.

Carpal tunnel syndrome is another example of growth hormone induced osteopathy. The characteristic paraesthesia observed in carpal tunnel syndrome associated with acromegaly occurs secondary to growth hormone related synovial oedema, which leads to median nerve compression. Such articular manifestations may be the presenting signs of excess growth hormone, with up to 70% of patients with acromegaly experiencing some degree of arthropathy.

Julia Lake (julia.c.w.lake@hitchcock.org), Sushela Chaidarun, Dartmouth-Hitchcock Medical Center–Endocrinology, Lebanon, New Hampshire, USA

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If you would like to write a Minerva picture case, please see our author guidelines at http://bit.ly/29HCBAL and submit online at http://bit.ly/29yyGSx

Physical activity

Physical activity prevents obesity, reduces levels of cardiovascular risk factors, and improves health in many other ways too. Even so, in developed countries only two in three adults achieve the minimum recommended 150 minutes of moderate activity every week. A longitudinal study of 200 000 adults in Taiwan reports that smaller amounts of exercise are also worth while. People who managed around half the recommended amount of physical activity had a substantially lower incidence of hypertension, dyslipidaemia, and diabetes over six years of follow-up than people who took no exercise at all (*Br J Sports Med* doi:10.1136/bjsports-2018-099740).

Acute gout

Colchicine interferes with microtubule formation and down regulates multiple inflammatory pathways. Although long established as an effective treatment for acute gouty arthritis, colchicine has a narrow therapeutic window and adverse reactions, particularly abdominal pain and diarrhoea. A multicentre randomised trial in general practice compares colchicine with naproxen in the treatment of a gout flare (*Ann Rheum Dis* doi:10.1136/annrheumdis-2019-216154). Both drugs were effective in reducing pain but adverse effects were twice as common in people given colchicine.

Beta 2 agonists and Parkinson's disease

The discovery that the β2-adrenoreceptor was a regulator of the α-synuclein gene generated interest a few years ago. Accumulations of α-synuclein occur in the brains of people with Parkinson's disease and it was hoped that β2 agonists such as salbutamol might slow progression of the disease. Studies from Norway reported a lower incidence of the disease in people who had ever used salbutamol, but a large UK investigation with more detailed information on duration of use finds that the protective effect is limited to short term use (*Am J Epidemiol* doi:10.1093/aje/kwaa012). This suggests that the explanation is something other than a biological effect of β2 agonists on brain pathology.

Colouring books in the emergency department

Colouring books for adults have become popular in recent years. They're promoted as a way of reducing feelings of stress and as an aid to relaxation. A small randomised trial finds, remarkably enough, that they work for anxious patients waiting in emergency departments (*Acad Emerg Med* doi:10.1111/acem.13838). Evaluated by the Hospital Anxiety and Depression Scale, a validated self-reporting score, patients who were given a colouring book and coloured pencils experienced a moderate drop in anxiety levels, while those given a blank notebook and pen did not.

The replication crisis

Researchers at the Berlin Institute of Health have been offered €1000 (£840; $1100) to put towards future research projects if they publish a paper with null results, or a replication study, or a paper that reuses data previously published by others. It's part of a programme to boost research transparency and confidence in science. Minerva thought this was the best response to the replication crisis that she had yet heard. Instead of handwringing about the state of medical science, how much smarter to offer an incentive to behave better (https://www.timeshighereducation.com/news/scientists-offered-eu1000-publish-null-results).

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