education

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

Will ranitidine make a comeback?

When ranitidine was taken out of circulation it left an obvious void, as it was a popular drug for heartburn. But it was suspended for good reason. In 2019 a US citizen petition reported the carcinogen N-nitrosodimethylamine (NDMA) was found in specific lots of ranitidine, and it proposed that ranitidine could convert to NDMA in the body. This was investigated by the US Food and Drug Administration, but the analysis had several general study limitations.

In this new study, Florian and colleagues report on the effect of oral ranitidine on urinary excretion of NDMA. The study design was a randomised crossover of 18 healthy participants comparing 300 mg ranitidine with placebo and looking at 24 hour urinary excretion of NDMA. They tested a worst case scenario of people being able to convert more ranitidine to NDMA because of the presence of nitrites (through having a high nitrate diet from eating cured meat). The good news was that ranitidine did not increase NDMA in the urine. These data are reassuring for ranitidine, but they don't necessarily mean that the drug will be back when other H2 antagonists cause no such concerns.

JAMA doi:10.1001/jama.2021.9199

Amyloidosis gene editing

I'm surprised to read a paper about the first six patients in a phase I, single arm study. Generally it is not good practice to report interim results. But it is wonderful news that the intravenous gene editing approach reported by Gillmore and colleagues was not unsafe and reduced the concentration of transthyretin protein in people with hereditary transthyretin amyloidosis. Evidence for efficacy of the CRISPR/Cas9 strategy for genetic disorders is growing, so get ready.

I only hope that early promising results aren't followed by neutral or unfavourable ones. It will also be important to see if the protein modifications translate into tangible benefit. For example, after cardiomyopathy develops in amyloidosis, is it too late to meaningfully intervene with gene editing?

N Engl J Med doi:10.1056/NEJMoa2107454

Physical punishment in children

Heilmann and colleagues performed "a narrative review of prospective studies." If this were the title of a talk, I can picture the audience's attention dropping to their phones. But this is a valid approach to systematically studying physical punishment and child outcomes, and it yielded useful results. Not all issues, especially public health ones, can be analysed in a more traditional manner such as a meta-analysis.

The main conclusion of this review was "documented compelling evidence that physical punishment is harmful to children's development and wellbeing and has shown no evidence that it is beneficial." This supports the principle of policies prohibiting physical punishment, but such policies may not address the drivers of parental behaviour, so more work is needed to develop interventions to improve child outcomes. The review also went into depth about common beliefs (for example, are the child's behavioural problems eliciting the punishment, or the other way round?). I highly recommend reading this review for its thoughtful approach to unpicking this issue of potential reverse causality.

Lancet doi:10.1016/S0140-6736(21)00582-1

Examining autophagy disorders

Autophagy ("self-devouring") is the process of cells degrading unwanted parts. Done correctly, it's a crucial part of health. Done wrongly, it can be fatal or cause serious disease. A lot of work has been done in this area in animals. Collier and colleagues examine this process further in humans with a genetic, clinical, and neuroimaging analysis of five families who had members with developmental disorders. They found that defective autophagy underlies the profound neurodevelopmental impairment in 12 patients within these families through deleterious recessive variants in the ATG7 gene. This gene encodes a protein necessary for autophagy.

This work advances our knowledge of the role of autophagy in human disease, especially in neural and musculoskeletal integrity. And with that comes the mildest twinkle of hope that gene therapy could one day be a therapeutic strategy for this series of patients or others with diseases related to impaired autophagy.

N Engl J Med doi:10.1056/NEJMoa1915722

Clarity on antibiotics for rectal chlamydia

This large Australian trial randomised men to 100 mg doxycycline twice a day for seven days or a single 1 g dose of azithromycin for asymptomatic rectal chlamydia. The primary endpoint of this 625 participant, doubleblind trial was microbiological cure at four weeks. This was achieved in 20% fewer men in the azithromycin group compared with the doxycycline group—that is, the cure rate was 76% rather than 97%.

This is critical evidence for the treatment of rectal chlamydia, as the two treatments were previously considered interchangeable. Clearly it is important to be using a doxycycline strategy. Gastrointestinal side effects such as diarrhoea were also more common in the azithromycin group.

N Engl J Med doi:10.1056/NEJMoa2031631

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Virtual consultation for red eye

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This is part of a series of occasional articles on common problems in primary care. *The BMJ* welcomes contributions from GPs.

CPD READING
0.5 HOURS

LEARNING MODULE
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A 63 year old woman contacts her general practice, via a telephone call, reporting a left painful red eye since yesterday. Because of the covid-19 pandemic, she is booked in for a video consultation with a GP the same day.

Face-to-face consultations remain the optimal medium for ophthalmic consultation. Virtual consultations have been implemented as an alternative in the covid-19 pandemic.[1-9] In a recent London study of 854 patients, video consultations for emergency ophthalmology services in adults had similar safety to face-to-face consultations. There was a higher rate of unplanned reattendance, but most patients were satisfied with video consultations.[10]

Red eye is a common ophthalmic presentation in primary care, accounting for 2-3% of consultations.[11] Virtual consultation for red eye(s) follow the same format and principles as face-to-face consultations, but adaptation with specific instructions to the patient and/or family member will be required.

Box 1 | Red flag symptoms and signs for red eye

Symptoms
- Ocular pain (acute onset, progressive nature, pain not relieved by analgesia or keeping the patient awake at night)
- Headache on the same side as the affected eye
- Visual loss
- Photophobia
- Recent history of contact lens wear, trauma, or ocular surgery
- New onset of binocular double vision

Signs
- Periorbital swelling or rashes respecting the midline of the face
- Proptosis
- Eyeball tenderness on palpation
- Corneal whitening (due to infection, severe inflammation, or oedema)
- Non-reactive pupil
- Systemic signs of infection (such as fever, chills, and being generally unwell)

WHAT YOU NEED TO KNOW
- Virtual consultation, when performed in a systematic fashion, is a safe alternative to face-to-face examination to diagnose and manage patients with acute red eye(s)
- Advise the patient to report if symptoms remain unchanged or worsen, as important diagnoses may be missed at the initial virtual consultation
- Arrange prompt ophthalmology referral for patients with a red eye and symptoms such as ocular pain, ipsilateral headache, loss of vision or double vision, photophobia, history of trauma or surgery, corneal haziness, or systemic signs of infection

What you should cover

Telephone consultation

You may ask for history of presenting symptoms over a telephone consultation to determine the cause and severity of red eye. It also provides an opportunity to advise patients on any immediate measures that need to be taken while awaiting an appointment for a virtual video examination (such as not wearing contact lenses if conjunctival or corneal infection is suspected). Ask about red flag signs and symptoms (box 1), which should prompt ophthalmology referral.

Virtual examination

After a detailed history (box 2), a subsequent video consultation is desirable but may not always be necessary, for example, if a diagnosis can be made confidently (such as conjunctivitis) or if the patient warrants a face-to-face ophthalmic examination (such as corneal infection with severe painful red eye and visual loss).

Virtual examination can be performed either via a live interactive video consultation, or a store-and-forward, asynchronous review of patient's self-taken images.[3] Tailor the virtual examination according to the history.

Visual acuity—A crude estimation can be performed by asking the patient to read sentences of different font sizes (such as from a newspaper) at a fixed distance. Ask the patient to check one eye at a time, with the correct glasses (if any), as many patients are not aware of the loss of vision until they shut the unaffected eye. Comparing visual acuity between the eyes may provide additional information on level of visual impairment in the affected eye. In patients with diplopia (double vision), this helps to determine whether the diplopia is monocular (suggestive of ocular causes) or binocular (suggestive of ocular motility or neurological causes).

Free online mobile phone apps are available to check vision, but most of them have not gained regulatory approval.[17 18] In a prospective comparative study of app-based home vision testing in children, most families were able to generate results deemed useful for clinical decision making, but parental engagement was low.[19]

Face and eyelid—The presence of vesicles and rashes on the face or eyelids suggest herpes simplex infection or herpes zoster ophthalmicus (if the involvement respects the midline of the face and V1 dermatomal distribution).[20]

Box 2 | Pointers for history taking in a patient with red eye(s)

Is it painful? Red eye with no pain or only mild discomfort is usually self limiting (fig 2). Painful red eye usually points towards more serious causes such as corneal infection,[12] anterior uveitis,[13] acute angle-closure glaucoma,[14] or scleritis.[15] In the presence of ocular pain, explore the onset, duration, progression, laterality, and severity of the pain. Ask about red flag symptoms that indicate serious conditions (box 1). Photophobia is suggestive of corneal pathologies or anterior uveitis. Pain on eye movements suggests orbital pathology.

Is it unilateral or bilateral? Acute painful red eye conditions—including corneal infection, acute angle-closure glaucoma, and scleritis—often present unilaterally. These are potentially sight threatening. Anterior uveitis may present unilaterally or bilaterally. Unilateral conjunctivitis should be a diagnosis of exclusion, after ruling out any red flag symptoms and signs.

Is there any discharge? Ocular discharge and itching are commonly associated with conjunctivitis. Viral conjunctivitis usually produces watery mucous discharge, whereas bacterial conjunctivitis produces mucopurulent discharge with crusting of the eyelashes.[16]

Is the vision affected? Loss of vision in red eye suggests a more serious cause, particularly when the loss of vision is sudden, severe, or progressive. Reduced visual acuity with painful red eye, halos, and headache are indicative of acute angle-closure glaucoma. Serious red eye conditions such as anterior uveitis and anterior scleritis may not present with visual loss during the initial stage. New onset of double vision with red eye is suggestive of orbital pathologies, including orbital cellulitis and thyroid eye disease.

Any contact lens wear? A painful red eye in contact lens wearers should raise the suspicion of contact lens-related corneal infection. This requires an urgent ophthalmic assessment within 24 hours.

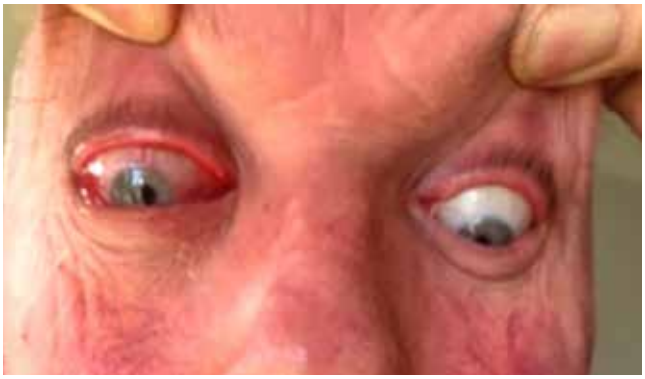

Fig 1 | A patient with a new onset of a right painful red eye with diplopia. Proptosis, restricted eye movement, and anisocoria (right pupil is larger than the left pupil) was detected on a live video consultation. Visual acuity was checked by a family member using an app. An inter-eye acuity difference supported a diagnosis of right optic neuropathy. The patient was urgently referred for further management.

Box 3 | Differential diagnosis of conjunctival hyperaemia

Diffuse superficial hyperaemia—Conjunctivitis, dry eye, blepharitis, corneal infection (mild)

Localised superficial hyperaemia—Subconjunctival haemorrhage, episcleritis

Diffuse deep hyperaemia—Corneal infection (severe), acute angle-closure glaucoma, scleritis

Circumcorneal hyperaemia—Anterior uveitis

Presence of Hutchinson's sign (involvement of the tip or lateral aspect of the nose) suggests a higher likelihood of ocular involvement in herpes zoster ophthalmicus.[20] Periorbital swelling may suggest preseptal (less serious) or orbital cellulitis (more serious), with the latter being associated with orbital involvement (for example, painful red eye, reduced visual acuity, proptosis, and restricted eye movement). Preseptal cellulitis can progress to orbital cellulitis, particularly in younger children where the orbital septum has not fully developed. Other aetiology such as angioedema, should be suspected when patient presents with periorbital swelling. Periocular eczema or inflammation is suggestive of allergic eye disease or contact dermatitis (when there is a recent history of using new eyedrops).

Conjunctiva—Look for localised or diffuse redness. Box 3 lists possible causes for conjunctival redness.

Cornea—Ask the patient or family member to shine a light (such as a pen torch) from the corner of the eye without obstructing the camera, whilst looking straight ahead. The cornea is normally smooth and transparent. Any haziness or whitening may indicate corneal opacity, infiltrate, or corneal oedema. This can be secondary to corneal diseases, intraocular inflammation, or elevated intraocular pressure. Corneal signs (unless severe) are not easily detectable on mobile technology. Refer patients with suspected corneal diseases, based on history, for a face-to-face examination.

Pupils—Inspect both pupils for size, shape, position, and symmetry by asking patient to bring the device close to their eyes. Any difference in size or symmetry of the pupils (anisocoria) warrants further ophthalmology assessment (fig 1).[21] Pupillary light responses, including direct and consensual light reflexes and relative afferent pupillary defect, should be checked when the vision is affected or orbital pathology is suspected. Examination can be performed by asking the patient to shine light into each eye in turn and swinging the light from one eye to another. A non-reactive pupil may be indicative of acute angle-closure glaucoma (tonic, mid-dilated pupil) or anterior uveitis (small, non-dilating pupil due to posterior synechiae). Relative afferent pupillary defect (RAPD) can be detected by moving the light between the two eyes quickly (spend about 3 seconds on each eye). Normally, both pupils should constrict when light is shone to either pupil. However, when RAPD is present in either eye, both pupils will dilate when light is shone to the abnormal eye (due to reduced afferent light impulse). This is indicative of significant disease at pre-chiasmal visual pathway, including retinal pathology or optic neuropathy.

Eyeball palpation—If there is no history of trauma to the eye, ask the patient to palpate the affected eyeball. Tenderness may suggest scleritis or acute angle-closure glaucoma. "Eyeball hardness" could be a sign of raised intraocular pressure, which warrants an urgent assessment by an ophthalmologist. However, this sign is very subjective and needs to be interpreted with caution.

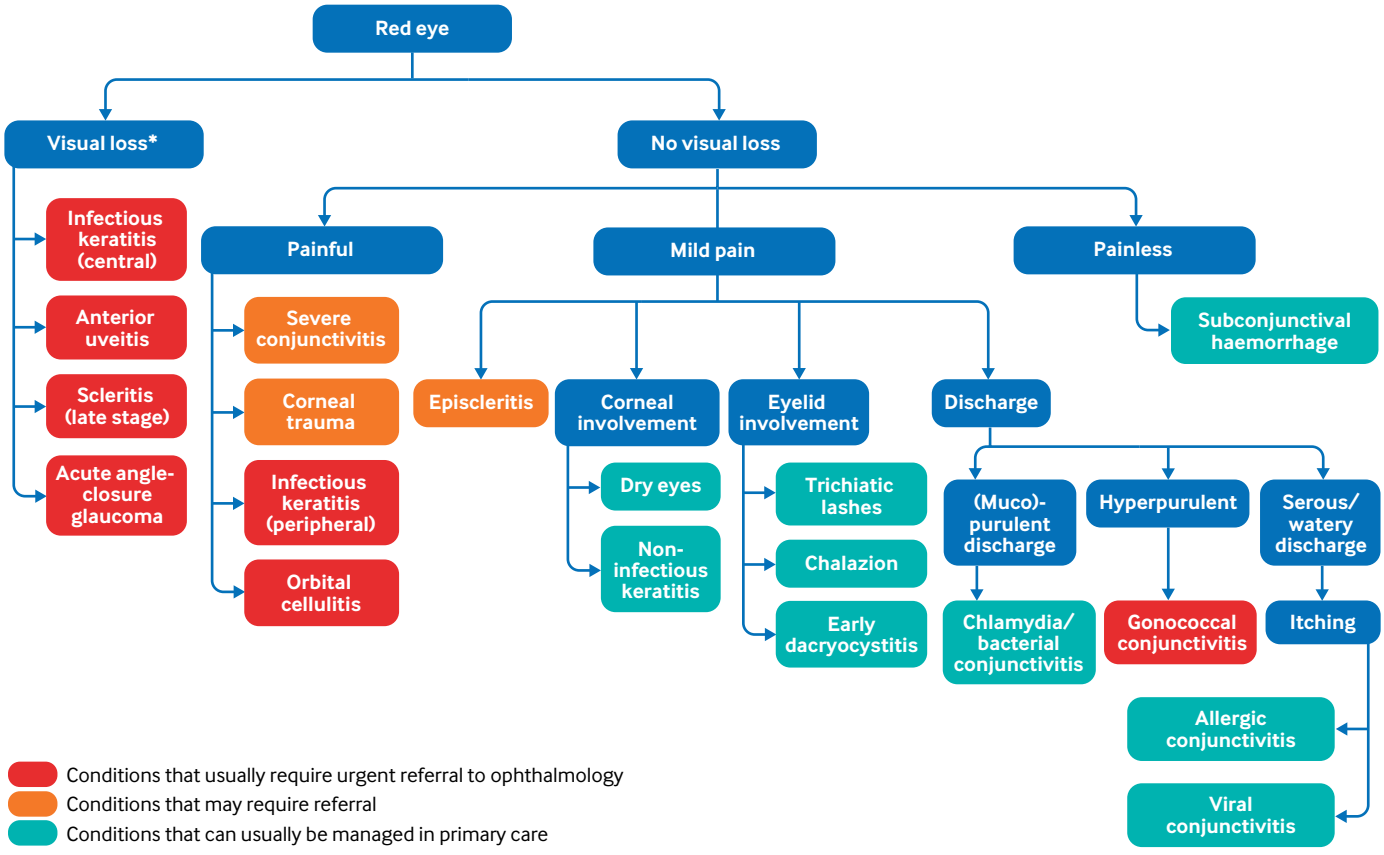

Fig 2 | Possible diagnoses based on initial assessment of red eye in primary care. *Red eye with visual loss is often caused by conditions that are painful and warrant an urgent ophthalmic examination. However, be aware that patients with neurotrophic keratopathy (reduced or absent corneal sensation) may not complain of any ocular pain despite having serious corneal pathology such as infectious keratitis

What you should do

Recognising when to refer patients with a red eye to ophthalmology services, whether it is routine or urgently, is crucial (box 4). Fig 2 represents possible diagnoses to consider based on initial assessment.

Patients with non-sight threatening red eye conditions—such as mild infectious or non-infectious conjunctivitis, subconjunctival haemorrhage, or mild corneal abrasion—can be reassured and managed in the community.[22] Advise that most cases of acute viral and bacterial conjunctivitis will resolve without any treatment in 5-7 days. A trial of topical antibiotics (such as chloramphenicol or fusidic acid) can be started if symptoms are not resolving within three days of onset in bacterial conjunctivitis.[23] If the condition does not improve or if it worsens after treatment, offer referral to an eye casualty unit for urgent ophthalmic assessment.

Box 4 | Conditions that require urgent referral to an ophthalmologist

- Severe "conjunctivitis" that does not improve after several days of antibiotic treatment ("bacterial conjunctivitis") or lubricants ("viral conjunctivitis")
- Corneal infection (especially in contact lens wearers)
- Anterior uveitis
- Acute angle-closure glaucoma
- Endophthalmitis (suspect if the patient had intra-ocular surgery within the past week)
- Ocular trauma (including mechanical, chemical, and thermal injury)
- Scleritis
- Orbital cellulitis

Suspected severe and potentially sight threatening conditions or the presence of red flags warrant prompt ophthalmology referral.[23] Patients with recurrent history of red eye related conditions warrant a face-to-face ophthalmology review. Ocular signs could be too subtle to be picked up by mobile imaging. Contact the local on-call ophthalmology team immediately for these patients.

Competing interests: None declared.

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EDUCATION INTO PRACTICE

- Recollect a patient with an eye condition you examined recently. What would you do differently based on reading this article?
- What red flags would you look for in a patient with red eye that would prompt an ophthalmology referral?

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

Recent examples of patients with a red eye who had been assessed by virtual consultation have informed the writing of this article. Images of patients with red eyes taken during virtual consultations have been used after obtaining informed consent. We are grateful for their contribution.

Lumbar spinal stenosis

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0.5 HOURS

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Lumbar spinal stenosis (LSS) affects about 11% of the population,[1] and primarily affects older adults.[1] Pain in legs and difficulty walking can limit function and participation in daily activities, which can have negative psychological effects.[2]

Diagnosis can be challenging because of the overlap of symptoms with other conditions that cause leg or low back pain in older adults. Lumbar spinal stenosis can usually be diagnosed clinically and managed conservatively in primary care. Patients with severe symptoms may require referral for imaging and intervention. The evidence for most treatment options is limited.[3-5] Shared decision making with patients must consider the severity of symptoms and their impact on the person's life, risks and benefits of treatments, and individual preferences.

WHAT YOU NEED TO KNOW

- Suspect lumbar spinal stenosis in people over 50 who describe leg pain or paraesthesia on walking or prolonged standing, and who are walking reduced distances as a result
- Imaging is not required during initial assessment as the correlation between imaging findings and symptoms is poor
- Conservative treatment, which includes supervised exercise and manual therapy, is advised as first line treatment; about 30-50% of patients with mild to moderate symptoms experience spontaneous improvement in pain and ability to walk greater distances
- Prescribe pain medication only for a short period and after careful consideration, taking into account the important side effects, especially in older people, and the absence of good evidence for efficacy
- Refer patients with severe symptoms, neurological deficits, or no improvement after 3-6 months of conservative treatment to a spine specialist for imaging and further intervention or surgery

What causes LSS?

Degenerative changes in the spine can cause narrowing of the central spinal canal, lateral recesses, or intervertebral foramen. Changes include disc degeneration and bony or soft tissue changes, such as facet joint arthrosis and hypertrophy of the ligamentum flavum, which occur with ageing[6] (fig 1). Narrowing may occur, which can cause compression and/or ischaemia of the associated neural and vascular structures.

How common is it?

About 11% (95% confidence interval (CI) 0.04 to 0.18) of adults in the general population experience symptoms of LSS, as per a systematic review (four studies, 6108 participants).[1] The pooled prevalence of clinical symptoms of LSS in primary care patients is 25% (95% CI 19 to 32%) (four studies, 171 157 patients). Prevalence varies between studies because of differences in population characteristics and diagnostic criteria used.

Prevalence increases with age. The mean age in the general population and primary care patient population is 62 and 69 years, respectively (age range 19-93).[1] Patients with congenital LSS are often younger.

How do patients present?

Patients describe being able to walk reduced distance because of pain in buttocks and/or legs (neurogenic claudication).[7] Symptoms are aggravated by walking or prolonged standing and relieved by forward bending (shopping cart sign) or sitting.[8] Pain is bilateral in central LSS and may be accompanied with paraesthesia, or weakness in the buttocks, thighs, or lower legs. Low back pain may be present.[9] The severity can range from mild discomfort when walking to being unable to walk. Impaired balance and forward flexion when walking may increase the risk of falling.

Lateral recess or foraminal stenosis can cause affection of single nerve roots (radiculopathy). Leg symptoms such as pain and paraesthesia will then often follow a more specific distribution and can be unilateral or bilateral. Combinations of these subtypes are common.

Key clinical features for a diagnosis of lumbar spinal stenosis, based on international consensus[9]

Patient over 50 with the following symptoms:

- leg or buttocks pain while walking
- forward flexion to relieve symptoms
- relief experienced when using a shopping cart or bicycle
- motor or sensory disturbance while walking
- normal and symmetric foot pulses
- lower extremity weakness
- lower back pain

How is it diagnosed?

History and examination help make a presumptive diagnosis of LSS to initiate management. Buttock and/or leg symptoms that worsen on extension postures and are relieved with flexion can guide diagnosis. The box lists clinical features that increase the certainty of a diagnosis of LSS. Patients may not always clearly distinguish buttock pain from low back pain. You may attempt to trigger the symptoms by asking the patient to walk or by standing in a lumbar spine extension posture for 30 seconds.[8 10] Neurological examination is usually normal. Neurological deficits (eg, muscle weakness, absence of tendon reflexes, and sensory deficit) indicate severe disease. Rarely a patient may present with urinary retention or decreased anal sphincter tone, which suggest cauda equina. This requires prompt referral.[11]

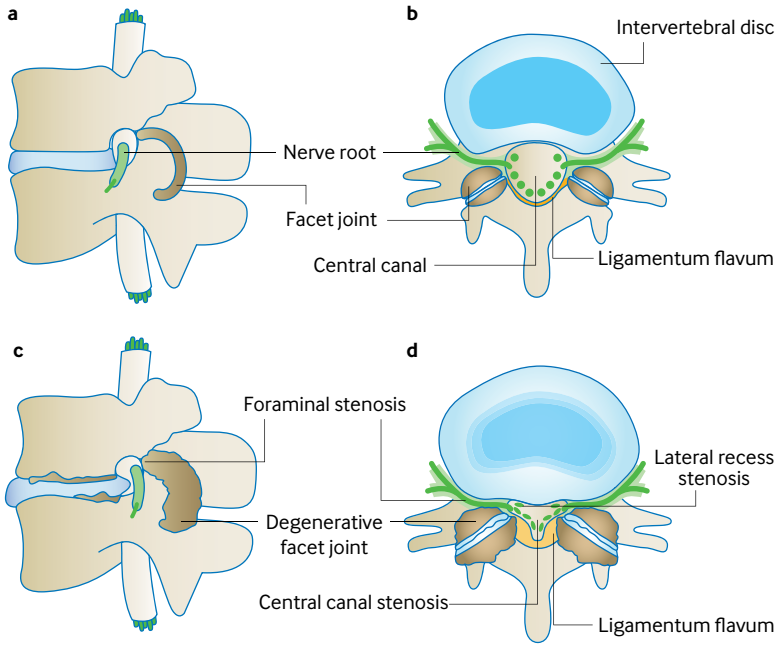

Fig 1 | Normal vertebral motion segment (a (sagittal) and b (axial)) and degenerative lumbar spinal stenosis (c (sagittal) and d (axial)). Narrowing of the central spinal canal and recess (d) is only minor to allow for visualisation of the nerve structures (small green dots). Sagittal (c) and the left side of the axial (d) illustrates a moderately compromised nerve root in the intervertebral foramen. Figure reproduced with permission from the Danish Chiropractors' Association

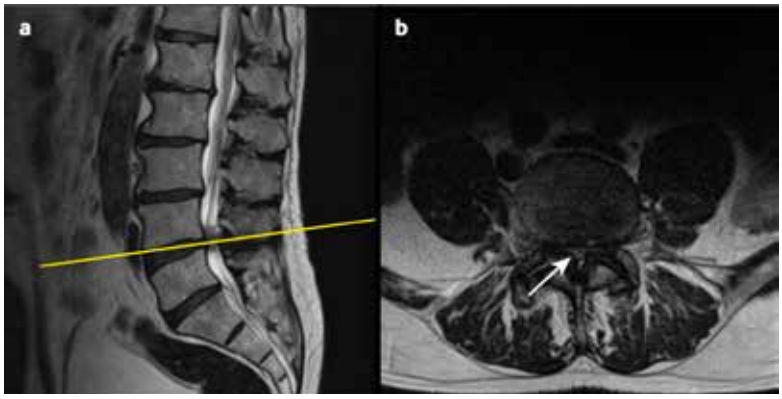

Fig 2 | Lumbar spinal central stenosis on MRI. Sagittal (a) and axial (b) T2-weighted MRI of the L4/L5 lumbar segment of a 57 year old patient. The yellow line on image a indicates the level of the visual slice shown in image b. The images are showing limited space in the central spinal canal (arrow) owing to disc bulge, hypertrophy of the ligamentum flavum, and osteoarthrosis of the facet joints

What differential diagnoses to consider?

Vascular claudication, a result of peripheral arterial disease, causes leg pain which—like LSS—is trigged by walking and relieved with rest. Forward flexion, using a shopping cart, cycle, or stationary bike, does not improve symptoms as is expected in LSS. Cycling is usually tolerated in people with LSS. In addition, patients may have cold feet or legs and skin discoloration on the feet. Check for foot pulses and abnormal ankle-brachial index.

Hip osteoarthritis also presents with buttock, groin, lateral hip, and leg pain provoked by weightbearing gait. Patients may limp and have limited walking ability. On examination, pain may be localised to one hip or both. Passive flexion and internal rotation of the hip are usually limited in range and painful.

Trochanteric bursitis presents with pain overlying the lateral aspects of the hip aggravated by lying on the affected side. Pain can extend along the lateral hip to the knee. You may elicit point tenderness with palpation of the trochanteric area. Pain is present on active and resisted abduction, passive adduction of the hip, and during combined passive flexion, abduction, external rotation, and extension.

What are the investigations?

Imaging is not recommended as part of the initial assessment.[12] Imaging can confirm a clinical diagnosis of LSS,[13] but it is not a strong diagnostic tool. The association between imaging findings and symptoms or severity is unclear.[4] A systematic review (nine studies, 714 participants) found that 11% (95% CI 0.11 to 0.18) of people with no symptoms had radiological signs of LSS, and this rose to 21-33% of people over 60.[1]

Magnetic resonance imaging (MRI) of the lumbar spine (or computed tomography (CT) if MRI is contraindicated), can be done in patients being considered for surgery[4 14] (fig 2).

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

We asked a patient about the impact of LSS on their daily living, diagnosis, and treatment, in an informal interview (box "Patient perspective"). A patient with LSS from primary care kindly reviewed an early draft of this paper and suggested alterations to the language style and inclusion of additional illustrations. In addition, a patient reviewer kindly reviewed this paper for *The BMJ* and suggested information resources for patients. We have revised the article to incorporate their suggestions and are grateful for their input.

PATIENT PERSPECTIVE

We asked a patient in her 70s to share her experience of living with lumbar spinal stenosis. The patient received intensive six week structured treatment with supervised mobilisation exercises and manual treatment twice a week. This was supplemented with self-managed daily mobilisation and strengthening exercises and a stationary bike workout.

I was in incredible pain every single day and sometimes had to walk bending forward. The thought of having to walk the 400 metres to the bus was a nightmare. Sometimes I could not walk more than 100 metres and I had to stand still for quite a long time before I was able to walk again. I was worried about whether I could reach the bus and I didn't like to feel pathetic.

I started treatment 2-3 months ago. For the first few weeks, I was in pain after the treatment. It was stressful with the cycling and exercises I had to do every day. Then it started to help. Now I can walk to the bus. I only have minor pain and it does not restrict my activity. Last Sunday, I took part in a city walk of two hours. I have gained a new independence.

EDUCATION INTO PRACTICE

- How would you discuss lumbar spinal stenosis and the available treatment options with a patient receiving a new diagnosis?
- At your practice, how are patients included in decision making when choosing treatment?

SOURCES AND SELECTION CRITERIA

We searched Medline and the Cochrane Database of Systematic Reviews from 2016 to 2020 to build on the literature used in a clinical review published in the *BMJ* in 2016. We used the search terms "lumbar spinal stenosis" and "neurogenic claudication" and focused primarily on clinical guidelines and systematic reviews.

How is it managed?

Assess the impact of symptoms on the patient's life. Pain, limited walking ability, and low mood can affect participation in recreational and social activities.[2] Consider severity of symptoms and the patient's preferences when deciding the approach to management. Evidence to recommend any single treatment approach is limited.[15]

Conservative management

LSS is degenerative in nature, therefore the aim of treatment is to improve or stabilise symptoms rather than cure. About a third to half of patients with mild to moderate symptoms experience improvement over time with reduced pain and increased walking distance.[4 16] Patients rarely experience rapid symptom deterioration.[16]

National Institute for Health and Care Excellence (NICE) has not published guidelines on LSS. Other guidelines recommend exercises and manual therapy involving spinal mobilisation and manipulation.[5 17] Systematic reviews find that a limited number of randomised controlled trials of low methodological quality explore exercises and manual therapy in patients with LSS.[18-21] Supervised exercise and manual therapy resulted in a higher proportion of patients seeing an improvement in symptom severity, walking distance, physical function, and pain at 6 to 8 weeks compared with home or self-directed exercise or group exercise in recent small trials.[22-24] It is unclear if benefits sustain longer.

The efficacy of pain medications for LSS is uncertain primarily owing to low quality studies or lack of studies.[25] Prescribe pain medications only sparsely and after discussing their side effects with the patient.[5] Pain medication may be used for short term pain relief or to bridge waiting time in patients undergoing surgery.

Spinal injections

Reviews of interventional pain medicine in 2019 give a weak recommendation that local epidural anaesthesia injections without steroids could be considered in LSS, based on low quality evidence.[26 27] Owing to the potential adverse effects of steroids and no evidence of benefit, epidural administration of local anaesthetics alone may be considered.[26] Inform patients about possible side effects, short term effects of treatment, and the need for repeat injections.

A large, well conducted, randomised controlled trial (400 patients) showed no difference at 6 weeks between patients receiving epidural injections of glucocorticoids plus lidocaine or lidocaine alone.[28] Approximately 30-40% of patients achieved a 30% reduction in disability and 50% achieved a 30% reduction in leg pain at 6 week follow-up. One or more adverse events such as pain, skin irritation, fever, headache, dizziness or numbness was reported by 15-20% of patients. This was higher in the group receiving steroids. No trials have compared the effect of local anaesthetics with placebo.

Surgery

A minority of patients in primary care need surgery. Surgery aims to increase the space around the compromised neural structures. This is commonly achieved by decompressive laminectomy, in which part or all of the vertebral lamina are removed. Lumbar fusion as an add-on can be necessary in case of instability. Surgery may be considered in patients with severe symptoms or neurological deficits or those with no improvement or worsening symptoms after 3-6 months of conservative management.[5 14] Offer referral to a spine specialist.

A cohort study published in 2020 (2559 patients) reported unsatisfactory outcomes with surgery for LSS in patients with mild leg pain (3 on a 10 point scale).[29] A Cochrane review (five randomised controlled trials, 643 patients) in 2016 reported no clear benefits with surgery compared with non-surgical treatment. In patients undergoing surgery, 10-24% experienced side effects compared with none with non-surgical management.[21] A systematic review (64 cohort studies, 3774 participants) found a 50% reduction in pain and disability three months post-surgery but mild-to-moderate pain and disability persisted at five years.[30]

Ongoing trials are evaluating surgery and physiotherapy interventions in patients with LSS, which will add to the evidence.[31 32]

Competing interests: None declared.

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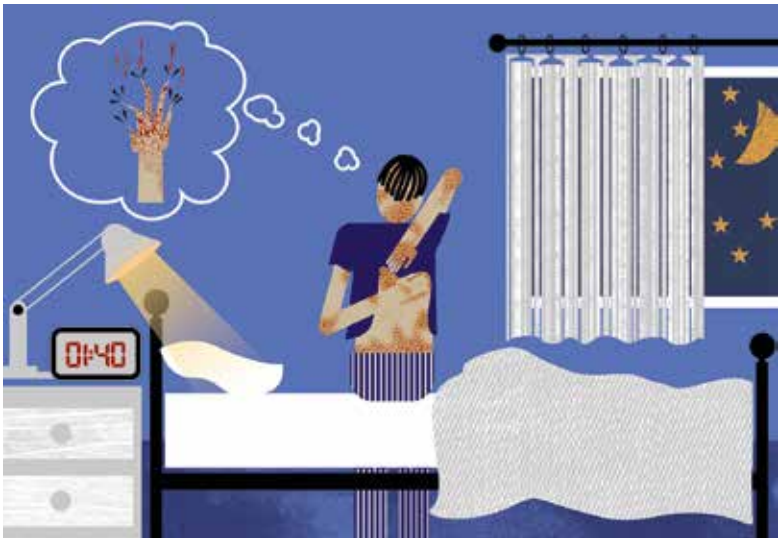
WHAT YOUR PATIENT IS THINKING

Just another eczema case for you, but to me it's the world

Wei Chern Gavin Fong describes what it is like to grow up with the skin condition and how health professionals could have supported him better



ROSE LLOYD

CPD READING
0.5 HOURS

I was diagnosed with eczema as a child and have lived with it for two decades. My journey has not been smooth sailing. The itch of atopic eczema was often debilitating, incessant, and intrusive. I remember clawing myself at night and struggling to sleep. At times I even physically hurt myself to distract from the itch. I was often told to simply "stop scratching," which was like being told not to sneeze.

At most of my eczema clinic appointments I felt like I was being giving instructions rather than having a conversation. Any discussions were brief and mainly with my parents. No one truly spoke to me about my condition. I was involved only during a quick examination of my skin. The lack of explanation resulted in my parents and I not understanding the condition nor the treatment. We began to worry about becoming dependent on topical treatment, especially the steroids. I started refusing to use these sticky and uncomfortable therapies as I did not understand how they would help. This led to me being admitted to hospital to manage my condition and I missed three months of school. It also led us to explore expensive and unhelpful alternative therapies, some of which made my skin worse.

I wish health professionals had explained what caused the itching and why the topical therapy would help. Having a better understanding of the biology of eczema would have helped me learn how to manage it and improve my adherence.

Psychological impact

With my skin so visible, the eczema had a profound impact on my mental health. Growing up, I remember receiving glances that were a mixture of pity and disgust. I hated my appearance and would not even look people in the eye because of how ashamed I felt of my skin. I had persistent low mood, low self-esteem, and even had some suicidal thoughts.

I wish clinicians realised the full impact of eczema on mental wellbeing. I want them to consider assessing the mental health of people living with eczema as well as their skin. I would have appreciated my doctor asking me how I was coping psychologically with my eczema. I also wish they had signposted me to support groups, as knowing that I was not alone would have given me hope and made me feel less isolated.

I wish clinicians realised the full impact on mental wellbeing

Setting realistic expectations

My parents and I were told by many clinicians that I would outgrow eczema. Yet with each "older" milestone, my eczema persisted. The unmet promises made me frustrated, desperate, and further distrust health professionals and their treatments.

I wish our expectations had been managed appropriately. Knowing that—while my eczema could not be cured—I could control the condition and live a normal life would have helped me hugely.

My eczema now

As an adult my eczema is well controlled and has a minimal impact on my daily life. This is thanks to those health professionals who did take the time to educate me about the biology of eczema.

Competing interests: None declared.

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WHAT YOU NEED TO KNOW

- Sharing details on the biology of eczema may help a patient accept and manage their condition
- Setting realistic expectations, such as explaining that creams won't work immediately, may help patients feel informed
- The impact of eczema is beyond just skin deep: the psychological burden of this highly visible disease is profound

EDUCATION IN PRACTICE

- When might you explain the biology behind eczema to a patient and their family?
- How could you explore the concerns of a patient living with eczema?
- When might you ask a patient living with eczema about their mental health?
- How could you support someone living with eczema who is struggling with their mental health?

SPOT DIAGNOSIS

A recurrent rash on the penis

An uncircumcised man in his late 70s presented with a six month history of recurrent asymptomatic patches on his penis. He had experienced three episodes of penile lesions, each lasting for about 7-10 days.

On examination, sharply demarcated, glistening, moist, erythematous patches were seen on the glans and opposing inner surface of the foreskin (fig 1). Subtle signs of male genital lichen sclerosus (constrictive posthitis, a preputial inflammation causing a band-like stenosis (sometimes called hourglass penis), and coronal adhesions, inflammatory obliterative fusions located at the mucosal part of the prepuce and corona of the glans penis) were also present (fig 2). No abnormal findings were seen on the penile shaft or scrotum.

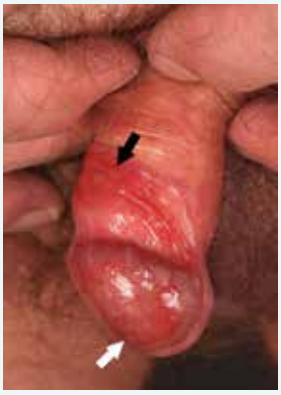

Fig 1 | Lateral part of penis showing sharply demarcated, glistening, moist, erythematous patches on the glans (white arrow) and opposing inner surface of the foreskin (black arrow)

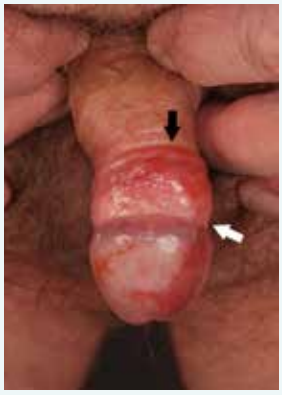

Fig 2 | Dorsal aspect of penis showing well demarcated, erythematous, shiny patches and plaques on the glans penis and prepuce. Clinical signs of male genital lichen sclerosus, such as constrictive posthitis (black arrow) and coronal adhesions (white arrow) are present

The patient reported no new sexual contacts, and screening for sexually transmitted infections (*Treponema pallidum*, *Chlamydia trachomatis*, *Neisseria gonorrhoea*, and *Mycoplasma genitalium*) was unremarkable. He had no previous exposure to potential contact allergens or local drug use. To exclude penile intraepithelial neoplasia, a 3 mm punch biopsy sample was taken from a lesion on the dorsal part of the prepuce. Histopathology showed epidermal atrophy and plasma cell-rich dermal inflammatory infiltrates, haemosiderin deposition, and extravasated red blood cells.

What is the most likely diagnosis?

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Patient consent obtained

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answers

SPOT DIAGNOSIS **A recurrent rash on the penis**

What is the most likely diagnosis?

Plasma cell balanitis (or Zoon's balanitis) associated with genital lichen sclerosus.

Sharply demarcated, moist, erythematous patches on the glans and opposing foreskin, together with the histopathology described above. Effacement of rete ridges and dilated capillaries might also be seen on histopathology.

Underlying lichen sclerosus is present in 60% of patients with plasma cell balanitis; psoriasis might also coexist.

Plasma cell balanitis is a benign inflammatory mucositis, primarily found in middle aged or older, uncircumcised men, and in about 10% of men presenting to UK genitourinary medicine clinics. The condition can persist or recur. It is usually asymptomatic, although pruritus, dysuria, pain, and a burning sensation might be present.

Predisposing factors include chronic irritation due to microincontinence, balanoreprepuitial dysfunction (eg induced by lichen sclerosus), trauma, and poor genital hygiene.

The main differential diagnoses are penile Bowen's disease and erythroplasia of Queyrat, penile intraepithelial neoplasias induced by high risk human papillomaviruses (HPVs) (eg HPV16). Erythroplasia of Queyrat can coexist with plasma cell balanitis.

Patients might initially be managed in primary care with good genital hygiene, protective ointments, and topical corticosteroids (alone or with antibacterial or antifungal agents). Avoid contact with urine. Potent topical corticosteroids are also the first line treatment for genital lichen sclerosus. Refer patients with any non-responsive, suspicious, persistent, or atypical penile lesions to a genitourinary specialist. Consider biopsy if the diagnosis is uncertain. Complete remission is most likely with circumcision.

PATIENT OUTCOME

The patient underwent complete circumcision followed by application of clobetasol-propionate on the glans penis for four weeks. This resulted in complete clearance of the plasma cell balanitis and underlying lichen sclerosus. At follow-up two years after circumcision, there was no recurrence.

LEARNING POINTS

- Plasma cell balanitis may be accompanied by underlying lichen sclerosus or psoriasis.
- Differential diagnoses include penile intraepithelial neoplasia.
- Refer patients to a genitourinary specialist with an uncertain diagnosis or poor response to genital hygiene and topical corticosteroids.

CPD READING
0.5 HOURS

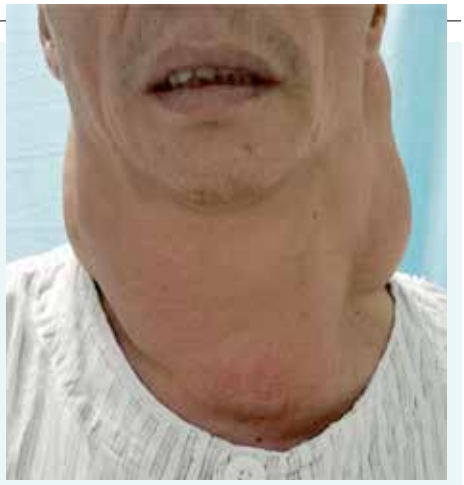
You can record CPD points for reading any article. We suggest half an hour to read and reflect on each.

LEARNING MODULE

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

MINERVA

A patient with an enlarged neck and alcohol excess



This is a man in his 60s with benign symmetrical lipomatosis. This rare (one in 25 000) lipid metabolic disorder is characterised by symmetrical, diffuse proliferation of non-encapsulated adipose tissue in the neck and upper trunk, which in 95% of patients is associated with excess alcohol intake.

The patient presented with dyspnoea. He reported a history of a painless mass on the neck that had increased over 10 years and an alcohol intake of 80 units/week for more than 30 years. Computed tomography showed characteristic non-encapsulated adipose tissue causing compression of the upper airway, clinically consistent with benign symmetrical lipomatosis.

Although this condition is benign, surgical intervention is indicated when the trachea or vena cava becomes compressed and causes dyspnoea or impaired venous return, respectively. The causative mechanism is thought to be mutation in the mitochrondrial DNA secondary to alcohol intake.

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Patient consent obtained.

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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

Exercise in people with AF

Physical exercise helps most things, but a retrospective study of 70 000 people living in Korea reports that the benefits in atrial fibrillation are small. People who initiated or continued regular exercise after an AF diagnosis had a slightly lower risk of heart failure over three years of follow-up compared with those who took no exercise. However, exercise brought no statistically significant reduction in risk of ischaemic stroke (*PLoS Med* doi:10.1371/journal.pmed.1003659).

Venous thromboembolism

Although long periods of immobility predispose to venous thromboembolism, a large US longitudinal study finds no evidence that people who spend a lot of time watching television are at increased risk (*JTH* doi:10.1111/jth.15408). Members of the cohort who reported high levels of physical activity showed a trend towards a reduced risk, however, no interaction was seen between TV viewing, physical activity, and risk of thromboembolism.

Complications of shingles

The commonest complication of herpes zoster is postherpetic neuralgia—pain at the site of the original rash persisting long after the rash has healed. Data from UK Clinical Practice Research Datalink show that other complications are numerous, but are all fairly uncommon (*BJD* doi:10.1111/bjd.19687). In the three months following diagnosis, around one in 75 patients had an ocular complication, such as keratitis, and around one in 200 had a neurological complication, such as facial palsy. Prescription of antivirals reduces the risk of complications by nearly half.

Collision sports

A longitudinal study from Sweden which followed a population based sample of older adults finds no evidence that playing collision sports in early life has long term adverse effects on cognition. Among 660 people, those who recalled taking part in sports such as football, ice hockey, or boxing were no more likely to be cognitively impaired at baseline or to develop cognitive impairment over 28 years of follow-up (*Am J Epidemiol* doi:10.1093/aje/kwab177).

Dementia risk in people with gout and hyperuricaemia

Epidemiological evidence links hyperuricaemia and gout to cardiovascular disease. However, a meta-analysis of four longitudinal studies from Europe, Taiwan, and the US discovers nothing to suggest that high levels of uric acid lead to an increased risk of dementia (*BMJ Open* doi:10.1136/bmjopen-2020-041680). Indeed, the data contained a suggestion that hyperuricaemia had a protective effect against Alzheimer's disease.

Phenotypes of MS

Whether the variable clinical course of multiple sclerosis reflects different pathogenic mechanisms or different manifestations of the same underlying pathology is debated. Two large studies from Sweden found that environmental and lifestyle factors associated with MS applied equally to both progressive onset and relapsing onset phenotypes (*Neurol Neurosurg Psych* doi:10.1136/jnnp-2020-325688). The investigators' interpretation is that the different clinical phenotypes share common underlying disease mechanisms.

Child mortality

Covid has probably had a hidden impact on the health of infants and children through reduced medical contacts, delayed presentation of serious illness, and increases in child maltreatment. However, the UK's national database reports that child mortality was lower in 2020 than it had been in 2019. During the first phase of the pandemic and in the first two periods of lockdown no excess child mortality was recorded (*Arch Dis Child* doi:10.1136/archdischild-2020-320899).

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Exercise did not reduce risk of ischaemic stroke in people with atrial fibrillation