education

RESEARCH REVIEWS Fortnightly round up from the leading medical journals

A vintage year for D-dimers?

An editorial about D-dimers likens the endlessly talked about blood test to a fine wine. It's nothing to do with its taste (I find it a bit claggy) or cost, but because it's matured with age to become an "indispensable tool in patients presenting in the outpatient setting with symptoms indicative of venous thromboembolism." The new ADJUSTED-DVT study explores whether age adjusted D-dimer thresholds can improve the sensitivity of the test in people with low to intermediate pretest probability of deep vein thrombosis. Among patients 75 years or older, using an age adjusted cut-off instead of a cut-off of 500 ug/L increased negative D-dimer results from 33 to 99 out of 379 (8.7% to 26.1%). Cheers.

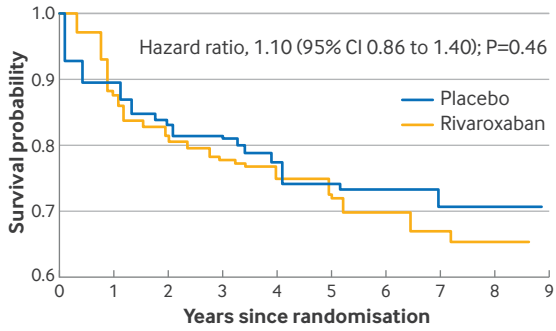
JAMA doi:10.1001/jama.2025.23182

Benefits of statins in people with low cardiovascular risk

For many people, deciding whether to start a statin depends on how much of a benefit they think they're going to get from it. An observational study using UK prescribing data puts some numbers on this: people with type

2 diabetes and a 10 year cardiovascular risk estimate of <10% who initiated a statin had a 0.53% lower absolute risk of death at 10 years, compared with those who didn't start taking one. Although the investigators used propensity scoring to match the characteristics of those who start taking statins with those that didn't, residual confounding might still explain some of the differences between the two groups.

Ann Intern Med doi:10.7326/ANNALS-25-00662



Primary efficacy outcome of cognitive decline, stroke, or transient ischaemic attack in participants with atrial fibrillation and low risk of stroke randomly assigned to rivaroxaban versus placebo

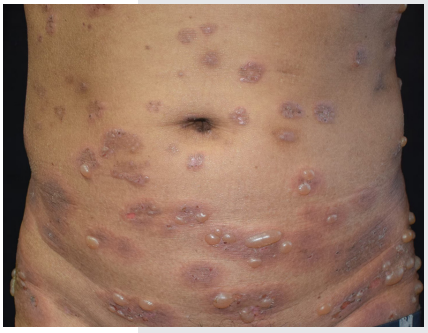
RIVARD L, KHAIRY P, TALAJIC M, *ET AL. NAT MED* 2026 DOI:10.1038/S41591-025-04101-Y

Anticoagulation to prevent dementia

Observational studies have found that people with atrial fibrillation are at increased risk of dementia, possibly caused by subclinical cerebral emboli. However, a new randomised control trial has found no benefit from anticoagulation on cognitive decline, stroke or transient ischaemic attack in people with atrial fibrillation and was terminated early. To enable a placebo controlled trial design participants were under the age of 62 and at low risk of stroke, so whether anticoagulation may reduce rates of dementia in higher risk, older patients with atrial fibrillation, is still unclear.

Nat Med doi:10.1038/s41591-025-04101-y

CLINICAL PICTURE



Bullous rash in end stage renal failure

A man in his 50s presented with a two week history of pruritic erythema and blisters on the trunk, which rapidly progressed across the body. He had a 10 year history of hypertension treated with metoprolol, and chronic kidney disease (CKD) for which he received long term haemodialysis. His medications had not been changed recently. Physical examination showed widespread tense blisters and bullae on an erythematous base. The Nikolsky sign was negative. Direct immunofluorescence showed linear IgG deposition at the basement membrane zone, and serology was strongly positive for anti-BP180 IgG antibodies. He was diagnosed as having bullous pemphigoid.

Bullous pemphigoid is a chronic blistering skin disease in which autoantibodies are directed against the dermal-epidermal junction. Renal insufficiency and haemodialysis are risk factors for developing bullous pemphigoid, with reported triggers including materials used in dialysis, medications, transplant rejection,

Rheumatoid arthritis nerve study stimulates interest

An implanted vagus nerve-targeted neuromodulation system could emerge as an effective treatment for rheumatoid arthritis. The implant is 2.5 cm long and weighs 2.6 g and is placed on the left cervical vagus nerve under general anaesthetic. Researchers recruited 242 patients with inadequate response to, or intolerance of, disease modifying anti-rheumatic drugs to the study, and allocated to active-vagal stimulation or a sham control. 35.2% of participants in the vagal stimulation group reached the primary endpoint (a 20% reduction in a composite disease severity score, the ACR20) after 3 months, compared with 24.2% of controls, rising to 50% at six months.

Nat Med doi:10.1038/s41591-025-04114-7

School of hard Roblox

As campaigns to limit or ban smartphone use in childhood continue to gain momentum, a research letter in JAMA sheds some light on what children use their phones for when they have access to them in school. The study, of 640 participants at schools in the United States, found that adolescents spent on average more than an hour a day using smartphones during school hours. They spent an average of around 30 minutes on social media apps, and about 15 minutes each on video apps and games such as Roblox. Self reported problematic mobile phone use was positively associated with more minutes using smartphones and social media during school hours.

JAMA doi:10.1001/jama.2025.23235

Tom Nolan, clinical editor, *The BMJ*, London; sessional GP, Surrey

Cite this as: *BMJ* 2026;392:s62

and CKD associated immune dysregulation. This patient was treated with oral prednisone (0.5 mg/kg/day), topical corticosteroids, and topical calcipotriol (a synthetic vitamin D analogue). After four weeks of treatment there was substantial clinical improvement with blister resolution, allowing tapered discontinuation of prednisone. At five month follow-up the patient remained in remission.

Patient consent obtained.

Cite this as: *BMJ* 2026;392:e084292

Xiaoqing Dong; Mengjun Xu (mjxu@zju.edu.cn), Second Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China

MINERVA From the wider world of research

Button batteries

Foreign body ingestion is common in young children, most often involving coins, magnets, or button batteries. Coins usually pass through the digestive tract without intervention. Magnets rarely cause harm unless more than one is swallowed. Button batteries, on the other hand, are dangerous because their size allows them to lodge in the oesophagus. Among 79 cases seen at a French paediatric centre, nearly 40% developed complications, including mediastinitis, oesophageal stenosis, perforation, and fistula formation (*Arch Dis Child* doi:10.1136/archdischild-2025-328576).

Epstein–Barr virus in systemic lupus erythematosus

Epstein–Barr virus has been implicated in multiple sclerosis and long covid and has now been linked with systemic lupus erythematosus. Many people carry B cells that weakly recognise self but are normally kept quiescent. An investigation using single-cell RNA sequencing showed that infection with Epstein–Barr virus reprogrammes these autoreactive antinuclear antigen B cells, turning them into antigen-presenting cells with the potential to promote systemic autoimmune responses (*Sci Transl Med* doi:10.1126/scitranslmed.ady0210).

Epileptic seizures in pregnancy

Epileptic seizures during pregnancy increase the rates of adverse outcomes for both mother and child, according to a systematic review of data from 25 000 pregnancies (*PLOS Med* doi:10.1371/journal.pmed.1004580). The risks of caesarean birth, preterm birth, low birth weight, and being small for gestational age were all increased by 30–50%. The risk of peripartum depression was doubled.

The Fermi paradox

Physicist Enrico Fermi famously asked, "Where is everybody?" Why can't we find other advanced civilisations in a universe as large and old as the one we inhabit? Perhaps intelligent life is vanishingly rare, or conditions on Earth are uniquely suited to it. Conceivably, no civilisation lasts for long because it destroys itself or exhausts its resources. Or maybe we cannot recognise them because they use advanced modes of communication that we are unable to detect (*Nautilus* https://nautil.us/we-might-not-be-so-strange-1242875).

Botulinum toxin in systemic sclerosis

Botulinum toxin inhibits sympathetic vasoconstrictor tone by blocking presynaptic neurotransmitter release from autonomic nerve endings. A review of more than 100 patients with acute digital ischaemia, digital ulcers, or gangrene—most of whom had systemic sclerosis as the underlying cause—reported high response rates to botulinum toxin injected around the intradigital neurovascular bundles (*JAMA Dermatol* doi:10.1001/jamadermatol.2025.4929). Adverse events, most often transient muscle weakness and injection-site pain, were mild.

Dairy consumption and risk of dementia

A large longitudinal study from Sweden with 25 years' follow-up reports that participants with high dietary intakes of high fat cheese and high fat cream experienced a 16% lower risk of dementia (*Neurology* doi:10.1212/WNL.0000000000214343). Consumption of other dairy products—including low fat cheese, low fat cream, milk (both high fat and low fat), fermented milk, and butter—showed no protective effect.

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Potassium sparing diuretics

Steven D Anisman, Stephen B Erickson, Ashley K Luneau

Full author details on bmj.com

Correspondence to: S D Anisman sanisman@nmcinc.org

Practical Prescribing is a series produced in conjunction with the *Drug and Therapeutics Bulletin* to highlight important issues for prescribers to consider and prompts for shared decision making between prescribers, patients, and their carers. Targeted at all medical and non-medical prescribers, particularly doctors in training, the series covers medicines commonly prescribed in primary and secondary care.

Advisers to this series are Fraz Mir, consultant physician at Addenbrooke's Hospital, Cambridge, and David Phizackerley, former deputy editor of *Drug and Therapeutics Bulletin*.

CPD READING

0.5 HOURS

A 73 year old woman presents to her GP after checking her blood pressure at home. Her average reading over the past few months is 140/80 mmHg. She is taking a thiazide-type diuretic, a calcium channel blocker, and an angiotensin converting enzyme (ACE) inhibitor. Her ejection fraction is 60%, with diastolic dysfunction on echocardiogram. She has noted some mild lower extremity oedema. She has normal renal function and no other comorbidities of note. She exercises regularly and eats a Mediterranean-style diet. You consider offering a potassium sparing diuretic to treat her heart failure, hypertension, and mild oedema.

How often are potassium sparing diuretics prescribed and how do they work?

Potassium sparing diuretics are widely used in the management of hypertension, heart failure, and oedema. All potassium sparing diuretics increase sodium and water excretion while retaining potassium by blocking sodium channels in the distal portion of the nephron—specifically, the late distal tubule and collecting duct (figure). Compared with loop diuretics, they are considerably weaker (approximately 5% to 10% as potent), although urine output is dependent on dose.[1] Potassium sparing diuretics can be combined with other diuretics (such as torasemide, furosemide, and/or chlortalidone) to enhance fluid removal while preventing potassium loss.

WHAT YOU NEED TO KNOW

- Potassium sparing diuretics are effective in treating resistant hypertension, and are useful for mild hypervolaemia, or for potentiating other diuretics
- Monitor potassium levels and renal function for the risk of hyperkalaemia
- The aldosterone inhibiting potassium sparing diuretics (spironolactone, eplerenone, finerenone) offer improvement in outcomes for patients with heart failure with reduced ejection fraction and heart failure with preserved ejection fraction, but also require monitoring
- Variations in subtype (aldosterone inhibiting *v* epithelial sodium channel blocker) might have an impact on drug selection

There are two classes of potassium sparing diuretics. The first class is pure epithelial sodium channel blockers such as amiloride and triamterene. They cause mild diuresis by direct inhibition of sodium channels, which is helpful in managing hypertension, especially when used in combination with thiazide diuretics (such as hydrochlorothiazide/triamterene).

The second class of potassium sparing diuretics is mineralocorticoid receptor antagonists such as spironolactone, eplerenone, and finerenone. They block sodium channels in the same way as pure epithelial sodium channel blockers,[2] but also suppress the renin-angiotensin-aldosterone system, providing additional therapeutic benefits. By blocking aldosterone receptors, mineralocorticoid receptor antagonists not only reduce sodium reabsorption and conserve potassium but also counteract the maladaptive effects of chronic renin-angiotensin-aldosterone system activation. This article focuses on mineralocorticoid receptor antagonists because they are the most widely used potassium sparing diuretics in practice.

Clinical uses

Spironolactone is the most frequently prescribed mineralocorticoid receptor antagonist, with approximately 340 000 prescriptions in the UK and 12.5 million in the US in 2022.[7,8]

Heart failure

Mineralocorticoid receptor antagonists are used to treat heart failure with reduced ejection fraction (defined as left ventricular ejection fraction <40%). Evidence supports a reduction in mortality,[9] and a reduction in the length of associated hospital stays.[10] Evidence is growing for use in heart failure with preserved ejection fraction (defined as left ventricular ejection fraction >50%), demonstrating that mineralocorticoid receptor antagonists reduce heart failure events and cardiovascular deaths,[11,12] regardless of the age of the patient.[13] These benefits appear to be additive in patients who are taking other drugs for heart failure, as directed by guidelines.[14] International guidelines that recommend the use of mineralocorticoid receptor antagonists in heart failure include National Institute for Health and Care Excellence,[15] American College of Cardiology/American Heart Association/Heart Failure Society of America,[16] and European Society of Cardiology.[17]

Eplerenone has identical dosing to spironolactone for this indication but is not associated with gynaecomastia or other endocrine related side effects seen with spironolactone. Some patients might prefer it for this reason.

The recent FINEARTS-HF trial showed that finerenone reduced worsening heart failure events and cardiovascular death, compared with placebo,

thebmj Visual summary

Prescribing considerations: Potassium sparing diuretics

This graphic summarises the main indications for potassium sparing diuretics, as well as some important considerations when prescribing these drugs. This information is not exhaustive and prescribers are encouraged to consult local guidance.

Drug classes

ENaC blockers — **Pure epithelial sodium channel**

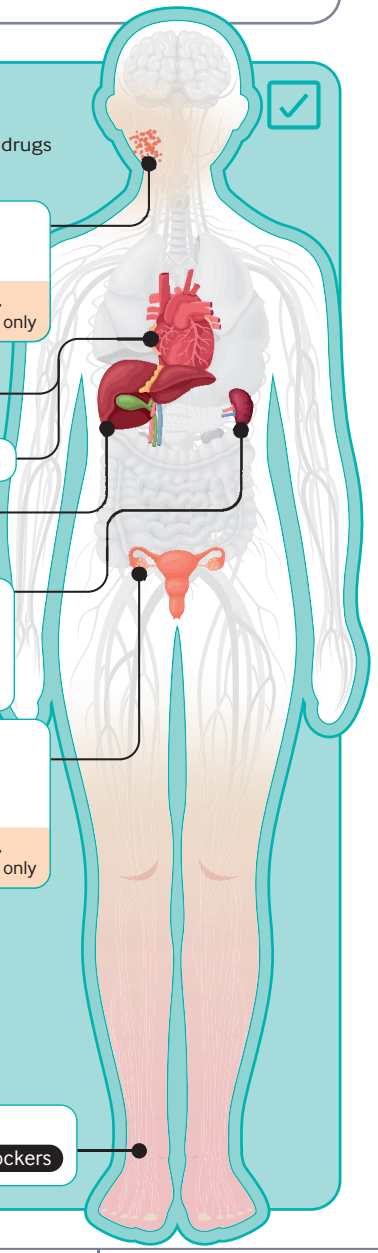
- Amiloride
- Triamterine

MRAs — **Mineralocorticoid receptor antagonists**

- Spironolactone
- Eplerenone
- Finerenone

Indications

Indications and licensed drugs may vary by location



- **Acne**
  - Spironolactone
  - ⚠ Unlicensed indication, specialist prescription only
- **Heart failure**
  - MRAs
- **Resistant hypertension**
- **Ascites**
  - Spironolactone
- **Chronic kidney disease associated with type 2 diabetes mellitus**
  - Finerenone
- **Polycystic ovarian syndrome**
  - Spironolactone
  - ⚠ Unlicensed indication, specialist prescription only
- **Oedema**
  - MRAs
  - ENaC blockers

Important adverse effects

- ⚠ Hyperkalaemia — Greater risk in acute or chronic renal impairment. Consider discontinuation if serum potassium ≥5.0 mmol/L, and/or ECG changes
- Hypotension
- Gastrointestinal side effects
- Endocrine issues (gynaecomastia/impotence) — Spironolactone

What should I monitor?

- Creatinine
- Serum electrolytes

Check at baseline, at one week, and one month after initiation

Monitoring requirements vary in the NICE and European Society of Cardiology guidelines

Further checks if needed, particularly with potential of higher risk

- Advanced age
- Heart failure
- Renal impairment
- Concurrent use of potassium raising drugs

Consider discontinuation if serum potassium level exceeds 5.0 mmol/L

Interactions

Certain drugs can exacerbate the risk of hyperkalaemia when combined with potassium sparing diuretics such as:

- Angiotensin converting enzyme inhibitors
- Beta blockers
- Angiotensin II receptor blockers
- Cyclosporine
- Heparin
- Non-steroidal anti-inflammatory drugs
- Pentamidine
- Renin inhibitors
- Tacrolimus
- Trimethoprim

⚠ **Caution and close monitoring are essential when these drugs are used together**

Avoid if

- Baseline potassium ≥4.5 mmol/L
- Pregnant
- Breastfeeding — Might be safe: Spironolactone

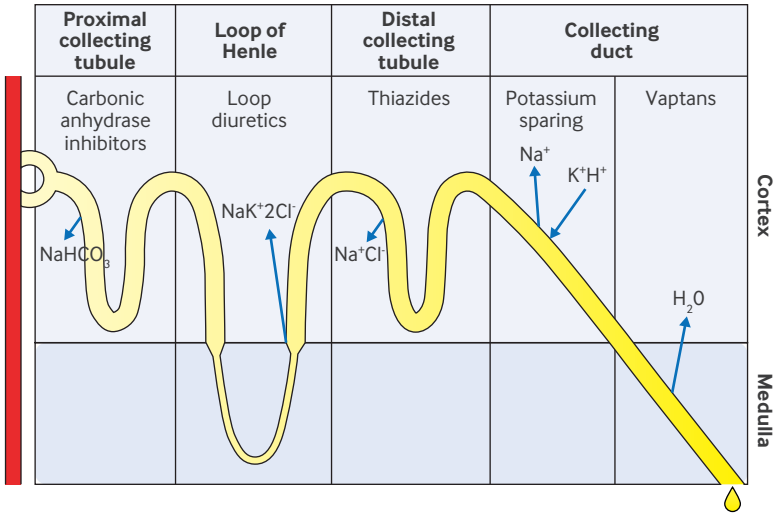
Contraindicated in

- Addison's disease — MRAs

Caution if

- Adjust dosage according to estimated glomerular filtration rate in renal impairment

No changes recommended for hepatic failure, although these drugs are typically metabolised in the liver



Sites of action of common diuretics within the nephron. For full description see bmj.com

in patients with heart failure with preserved ejection fraction and heart failure with mildly reduced ejection fraction (defined as left ventricular ejection fraction 40% to 50%).[20] This benefit is seen early in treatment,[21] particularly in patients with worsening disease,[22] without adversely affecting long term renal function,[23] and irrespective of age.[24] However, as yet, there are no head-to-head trials comparing the newer finerenone with well established spironolactone or eplerenone, suggesting that this may be a class effect.

Resistant hypertension

Resistant hypertension is defined as uncontrolled blood pressure despite three preferred agents such as a thiazide-type diuretic, a calcium channel blocker, and an ACE inhibitor or angiotensin II receptor blocker (ARB). The role of mineralocorticoid receptor antagonists in treating resistant hypertension is well established in multiple guidelines,[25] although this is an off-label indication in the UK. While potassium sparing diuretics provide only weak blood pressure reduction on their own, spironolactone and eplerenone are effective in resistant cases,[26] with studies showing clinically meaningful reductions in systolic blood pressure. Mineralocorticoid receptor antagonists have demonstrated reductions in systolic blood pressure of 9 mmHg compared with placebo in one study and 22/10 mmHg in another.[27 28]

Renal effects

Because their diuretic potency is modest, potassium sparing diuretics are not usually used alone for oedema, but they can augment loop or thiazide diuretics by limiting sodium reabsorption later in the nephron. This combination can help overcome diuretic resistance in heart failure when further fluid removal is required. In nephrotic syndrome, potassium sparing diuretics combined with loop diuretics improve diuresis while mitigating potassium loss, though head-to-head comparisons between diuretic classes are lacking for this indication.[33] Finerenone provides a benefit for patients with chronic kidney disease,[34] including diabetic kidney disease.[35]

Ascites

Spironolactone has also shown greater efficacy in managing ascites in cirrhosis when compared with loop diuretics.[36 37] Although the trials were conducted using spironolactone and it is the most widely used clinically, this is likely to be a class effect.

Other indications

Spironolactone has been used to manage acne, with mechanisms of action involving multiple pathways,[39 40] including decreasing sebum excretion by 30% to 50%.[41] In addition, spironolactone has shown benefits in the treatment of polycystic ovarian syndrome owing to antiandrogen effects.[42]

What should I discuss with patients before starting treatment?

Begin by asking whether the patient has previously taken a potassium sparing diuretic, the reason it was prescribed, and how effective it was.

Explain that patients might not notice immediate changes, because it can take days to weeks for the full effect of the drug. Depending on the indication, benefits might include improvement in symptoms (such as reduced shortness of breath or swelling for patients with heart failure) or lowered blood pressure for patients with hypertension.

It is important to emphasise that even if symptoms do not noticeably improve, these drugs reduce risks such as hospital admissions for heart failure, and cardiovascular events including heart attacks and strokes.

NICE recommends that patients starting these drugs should be counselled about the increased risk of renal failure and hyperkalaemia in the context of dehydration, for example, secondary to an acute illness causing diarrhoea and vomiting. If patients develop diarrhoea and vomiting, they should consult their healthcare provider to consider stopping the treatment.[43]

What are important side effects to discuss?

Hyperkalaemia is an important side effect, although its incidence and severity are reported with high variability.

Fewer than 15% of hospital admissions for hyperkalaemia overall are attributed to potassium sparing diuretics, highlighting that while hyperkalemia is important, it can be effectively managed with appropriate care.[50]

Despite the increased risk of hyperkalaemia from the combined use of potassium sparing diuretics and ACE inhibitors or ARBs,[52] this combination delivers the greatest mortality benefit in heart failure, especially in patients with reduced ejection fraction.[53]

The risk of hyperkalaemia is greater in patients with acute or chronic renal impairment,[54] and guidelines often recommend not initiating treatment if baseline potassium exceeds 4.5 mmol/L, with discontinuation advised if levels surpass 5.0 mmol/L.[55]

Steroidal mineralocorticoid receptor antagonists (eg, spironolactone) additionally interact with androgen and progesterone receptors, with the potential to cause endocrine side effects such as gynaecomastia,[58] impotence, and menstrual irregularities. Gynaecomastia caused by spironolactone is related to the dose, affecting about 9% of patients taking the recommended dose (25 mg daily) and up to 52% at high doses (150 mg daily), and is generally reversible after discontinuation.[59] Hormonal side effects are substantially less common with eplerenone and particularly rare with non-steroidal agents such as finerenone,[60] which are more selective for mineralocorticoid receptors and carry a lower risk of hyperkalaemia and hormonal effects.

Other side effects shared by antihypertensive or diuretic classes include hypotension (including orthostatic hypotension),[63] erectile dysfunction (thiazides, beta blockers, and spironolactone),[64] and gastrointestinal symptoms, including nausea, diarrhoea, and constipation, although these tend to be mild and short lived.

In practice, awareness of these side effects, particularly hyperkalaemia and hormonal effects, alongside diligent monitoring, enables patients and clinicians to balance risks and benefits effectively, optimising patient safety and clinical outcomes.

This list of side effects is not exhaustive, and we encourage prescribers to refer to local guidelines.

What to consider when prescribing?

Potassium sparing diuretics are generally avoided in pregnancy, but spironolactone is considered acceptable for use during breastfeeding in several settings, including the UK,[66] despite a small amount of transfer into breast milk. There are no breastfeeding data for eplerenone, and finerenone is contraindicated in breastfeeding. Mineralocorticoid receptor antagonists are contraindicated in Addison's disease, a condition already marked by aldosterone deficiency.[67]

What should I monitor during the course of the prescription?

Serum electrolytes should be monitored when taking any diuretic drug, but special attention should be paid when taking potassium sparing diuretics with other drugs that can increase potassium. Many American guidelines recommend checking serum electrolytes and creatinine at baseline, at one week, and one month after initiation of these diuretic drugs because hyperkalaemia typically occurs within four weeks of initiation.[16] All current guidelines also recommend longitudinal lab work at least every six months, or if there is reason for clinical concern, particularly concerns regarding renal function. There are some additional variations in frequency of monitoring between the NICE and European Society of Cardiology guidelines.[75-77]

Potassium supplementation can be given to patients who experience hypokalaemia while taking loop diuretics, although supplementation may no longer be necessary when potassium sparing diuretics are added. The British National Formulary states that potassium supplementation should be avoided with potassium sparing diuretics, however, we recognise in our own practice that this is sometimes required to treat clinically important hypokalaemia, in which case we would suggest additional monitoring.

Although possible with any diuretic medication, hyponatraemia appears to be less common with potassium sparing diuretics—most severe cases of hyponatraemia are associated with thiazides.[78]

EDUCATION INTO PRACTICE

- In what clinical situations might you consider a potassium sparing diuretic for your hypertensive patients?
- How do you decide between potassium sparing diuretics and other diuretics for oedema? If you prescribe a potassium sparing diuretic, how do you select between the various options?

When should I stop the prescription?

These drugs are used for the duration of the underlying condition; in most cases, this would be lifelong. However, it is reasonable to consider discontinuation when the drug is no longer needed, for example, for control of hypertension or oedema, if there are adverse effects, or for end-of-life care.

Hyperkalaemia is a reason to stop potassium sparing diuretics. There is no clear consensus regarding a threshold value that should prompt discontinuation, but a pragmatic approach would be to stop them if there is persistent hyperkalaemia that causes electrocardiogram changes or prompts hospital admission. Some guidelines recommend discontinuation on a 30% decrease in estimated glomerular filtration rate, although those same guidelines also recommend continuing the combination of ACE I and/or ARB in patients with chronic kidney disease, even if the estimated glomerular filtration rate falls below 30 mL/min per 1.73 m^2.[79] Note that some degree of increase in potassium is expected, as is a decrease in glomerular filtration rate, and an increase in creatinine. These result from direct drug effects, as well as indirect effects of the intended relative hypovolaemia with effective diuresis. In the SOGALDI-PEF trial, one in five patients had a drop of >30% in estimated glomerular filtration rate when combining mineralocorticoid receptor antagonists and sodium-glucose co-transporter 2 inhibitors, although this combination yielded noticeable benefits.[80] Stopping medication might undermine the benefits of this combination therapy, a particular problem given that these medications are subsequently often not restarted.[81]

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Competing interests: None declared.

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10-MINUTE CONSULTATION

Vulvodynia (chronic vulval pain)

Michal Braunstein,[1] Michal Sheinis,[2 3] Jeremiah Laktabai,[4] Amanda Selk[2 3]

Full author details on bmj.com

Correspondence to: A Selk Amanda.selk@utoronto.ca

This is part of a series of occasional articles on common problems in primary care.

CPD READING
0.5 HOURS

Vulvodynia is chronic, unexplained vulval pain lasting more than three months without an identifiable cause.[1 2] It can be classified as primary—present from the first physical contact—or secondary, arising after an initial pain-free period.[2] The pain may be localised to a specific area, such as the vestibule or clitoris, or generalised, involving the entire vulva.[2] The exact mechanism of why vulvodynia occurs is not fully understood and is likely multifactorial.

Multiple, large population based surveys in the US estimate the lifetime prevalence of vulvodynia to be 10-28% in the general population. Qualitative studies show themes of delayed diagnosis (in many cases years), symptoms minimised by healthcare providers, and a lack of awareness and understanding by health professionals.[3 4] For example, in interviews with 10 women aged 25 to 57 in the UK diagnosed with vulvodynia, participants reported experiencing disbelief, stigmatisation, and minimisation of their symptoms by physicians.[4] Interviews of eight women aged 23 to 32 in Norway revealed similar patient experiences.[3]

Vulvodynia, like other vulval pain, often affects quality of life, causing extreme discomfort or pain while wearing underwear, trousers, or while sitting. It can also hinder intimate relationships, gynaecological examinations and procedures, and can negatively impact daily activities.

What you should cover

Take a focused history:

- Where is the pain located? For example, is it localised to a certain portion of the genitalia? Pain confined to the clitoris is clitorodynia, and to the vestibule or vaginal entrance is vestibulodynia.
- How long has the vulval pain been present? The minimum duration required for a diagnosis of vulvodynia is three months.[1]
- Has there always been pain with sexual activity or with other physical contact (primary vulvodynia) or have symptoms developed after a period of pain free contact (secondary vulvodynia)? For secondary vulvodynia, ask about possible triggers, such as a traumatic injury and sexual assault.
- What does the pain feel like? Patients often describe the pain as burning, but it can also be sharp, pricking, or irritating.[5]
- Is the pain provoked? For example, by touch or insertion, unprovoked (appears spontaneously with no trigger), or mixed?[6]
- Are there situations or activities that exacerbate the pain, such as intercourse, wearing tight clothes, touching the affected area, riding a bicycle, using tampons, or prolonged sitting?[6 7]
  - Pain that appears while riding a bicycle may suggest nerve compression.
  - Pain that arises or worsens with sitting down may suggest recent trauma (eg, obstetric perineal trauma, genital mutilation), infected or inflamed Bartholin's cyst or other vulval or vaginal cysts, neuropathic pain syndromes (eg, postherpetic neuralgia, pudendal neuralgia, neuroma), or iatrogenic pain (eg, postoperative scarring or nerve damage, chemotherapy, radiation).
  - Intermittent pain affected by position or situation may suggest pelvic floor dysfunction—a common and often underrecognised condition, which may occur as a result of previous sexual assault, endometriosis, or arise after any pain has occurred (eg, with sexual intercourse or a gynaecological examination).
- What does the pain feel like on a scale of 1-10? This can be useful in quantifying the pain and assessing response to treatment. Moreover, greater pain severity correlates with an elevated risk of additional comorbidities, however it does not help differentiate cause.[8]
- Do you have a history of cervical cancer or dysplasia? This is a risk for vulval dysplasia and should prompt you to assess for vulval lesions.[1 7]
- Have you had any recent genital infections such as chlamydia, yeast and bacterial vaginosis, or obstetrical lacerations or tears to the area affected? If the patient has had recent infection, rule out recurrence of infection.[7]
- Do you use any hormone-containing medications, including contraceptives, fertility medications, or endometriosis medications such as gonadotropin releasing hormone agonists? These may lead to vaginal atrophy and pain.[9 10]

WHAT YOU NEED TO KNOW

- Vulvodynia is chronic, unexplained vulval pain lasting more than three months without an identifiable cause, and is classified as primary (present from the first physical contact) or secondary (arising after an initial pain-free period)
- A variation of the cotton swab test can be used to localise and quantify pain
- Treatment includes pelvic floor physiotherapy, psychotherapy, and oral neuropathic pain medications

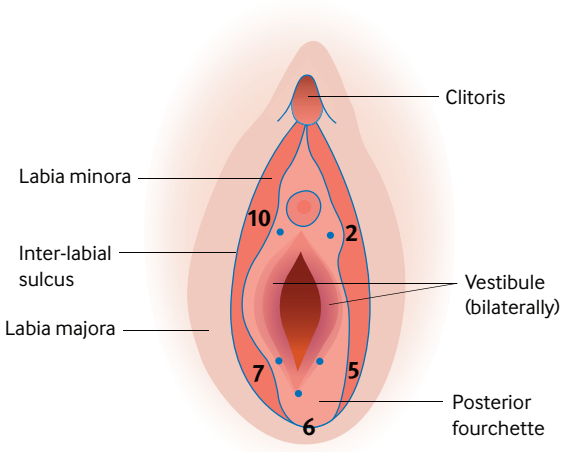

Fig 1 | Assessing pain using a variation of the cotton swab test. Begin on the inner thigh to assess the labia majora, inter-labial sulcus, labia minora, clitoris, and vestibule bilaterally.[6] When assessing the vestibule (just distal to the hymen), assess at 2:00, 10:00, 5:00, 6:00, and 7:00 positions systematically.[5] According to expert opinion, major and minor vestibular gland areas (ie, 2, 10, 5, 7) and posterior fourchette (6, 5, and 7) represent the major vestibular glands, and 2 and 10 are the minor vestibular glands

- Are there any associated symptoms beyond pain, which may suggest an alternative or comorbid diagnosis (table 1, bmj.com)?

Examination

Before an intimate examination, explain the procedure to the patient using a trauma-informed approach. Ensure the patient feels safe and in control, for example by asking their permission prior to touching them.[12] We recommend adequate lighting, visualisation, draping, documentation of consent, and presence of a qualified chaperone.[12] Based on our experience, common accepted practice, and methodology in vulval pain studies,[6] we suggest:

Assess

- If the clitoris is visible (fully, partially, or not visible)
- Whether labia minora are present, resorbed, or absent
- Whether there are inflammatory skin changes and, less often, severe atrophy.

Skin colour changes (dermatoses), texture changes (dermatoses, dysplasia), and focal masses, erosions, or ulcers, may suggest infection, dysplasia, or malignancy.

A variation of a cotton swab test can be used to localise and quantify the pain. This test also assesses whether the pain is localised or generalised, provoked, unprovoked, or mixed:

- Using a cotton swab, demonstrate, in a neutral area (eg, the inner thigh), that you will be pressing it gently on various points of the vulva
- Ask the patient to rate their pain on a scale of 0-10 with 0 being no pain and 10 being the worst possible pain and examine structures systematically (fig 1)[6]
- Document the location and severity of pain.

Pelvic floor muscle overactivity is a common sequela of vulval pain and can also cause vulval pain, therefore should be treated in parallel. In our experience, it is reasonable to conclude that pelvic floor muscles are highly contracted in the following clinical scenarios:

- The vaginal opening appears small or narrowed
- The patient's bottom raises at the beginning of the examination
- The patient retreats when examination is attempted, or
- The patient blocks the option of examining with their hands or thighs.

In these cases, internal examination is not necessary. Targeted treatment with pelvic floor physiotherapy, where techniques differ based on severity of pelvic floor muscle overactivity and from that offered to patients with vulvodynia alone, may help alleviate symptoms.

In all other cases of suspected pelvic muscle overactivity, discuss the option of performing a vaginal examination in order to assess and document the degree of pelvic floor muscle tension and response to treatment:

- If indicated, and after consent, assess tone by gently inserting a single gloved index finger with lubricant into the vagina and applying mild pressure to the centre of each muscle before sweeping along its length (fig 2)
- A narrow or tight vaginal introitus typically reflects increased tone of the levator ani muscles, whereas a more compliant and flexible opening is palpated when these muscles are relaxed. The obturator internus muscle is most effectively palpated while the patient actively abducts the thigh, which increases tension in the muscle. When the patient relaxes the leg, the muscle softens, allowing clearer assessment of its baseline tone[13]
- Low-osmolarity, pH balanced lubricants are preferred to minimise discomfort and protect the vaginal epithelium.[14]

What you should do

Making a diagnosis

Since the definition of vulvodynia is vulval pain without an identifiable cause, assess for and treat other causes of pain as mentioned in table 1 before considering a diagnosis.

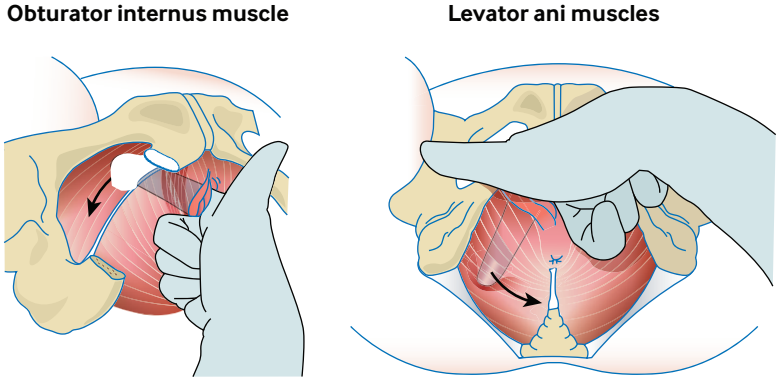

Fig 2 | Assessing pelvic floor muscle tension. The levator of the pelvic floor, stabilising the bladder and rectum. The obturator internus muscle, located in the pelvic wall, connects to this group via the arcuate tendon. Both muscles should be palpated using gentle, even pressure with the index finger during examination. Tight and stiff muscles are considered hypertonic[13]

Vulvodynia is a diagnosis of exclusion, but women can still present with both vulvodynia and another concurrent pathology (eg, recurrent vulvovaginal candidiasis or lichen sclerosus), and sometimes more than two conditions at once.[1] A cross sectional multicentre study of 1183 women with vulvodynia showed that 37.4% of women had concomitant urinary symptoms: 19.5% had recurrent urinary tract infections and 17.9% had post coital urinary tract infections.[15]

Provoked vestibulodynia (that is, pain confined to the vestibule triggered by touch) and generalised vulvodynia (spontaneous pain that affects more than just one specific area of the vulva) are the two most common types of vulvodynia.[1] According to a population based survey study of more than 19 121 women[17] as well as a subset of 371 women in a prospective cohort study in the US,[18] provoked vulvodynia is more common in women under 30 and generalised vulvodynia is more common in peri- or post menopause.

Overall, the yield of viral and bacterial swab and culture is not usually informative and is used mostly to rule out other possible diagnoses.[6] Swabs may be warranted in specific scenarios—for example, microbiological confirmation of candidiasis may be necessary if this was the suspected diagnosis and treatment has not led to substantial improvement.[5] With the exception of suspected malignancy, some dermatoses, and concerning lesions (such as chronic eczema or dermatitis that do not respond to treatment, which could for example be the manifestation of intraepithelial Paget's disease),[16] biopsies are usually reserved for refractory cases of vulvodynia. Their purpose is to rule out any other conditions that might have been missed. Otherwise, the diagnostic utility of biopsies remains uncertain.[7]

Management

Manage any identified pathologies related to vulval pain in line with local protocols and guidelines. For example, granulation tissue can occur as part of the healing process in perineal or vaginal tears postpartum, and can cause vulval pain. Therefore, if granulation tissue is found on examination, it can be cauterised with silver nitrate to treat the pain.[19] Another common clinical scenario is when a patient experiences hormonal changes secondary to oral contraceptive use, leading to atrophy, and subsequently, pain.[9] The standard treatment for this, other than stopping oral contraception use, is outlined in box 1 (bmj.com). These treatments may be needed even after stopping contraception. Topical oestrogen alone can be used in cases of genitourinary syndrome of menopause or lactational amenorrhea.

International guidelines recommend that treatment of vulvodynia is individualised and multimodal, including lifestyle modifications, psychotherapy, psychosexual counselling, pelvic floor physiotherapy, medications, and rarely, surgical interventions.[5-21]

Inform the patient that vulvodynia is common and that multiple treatments are available. Teach patients appropriate vulval care such as avoiding irritants like scented or dyed products, soaps, and douches. Advise them to wash with water only, pat dry gently, and avoid clothing that worsens pain. Recommend use of plain cotton underwear, no synthetic fabrics, and sleeping without underwear.[16] Inform them of patient support groups and resources locally and online.

Vulvodynia often affects quality of life, causing extreme discomfort or pain

Lubricants are recommended for patients who have pain during sexual intercourse. Oil based lubricants are not condom compatible, and silicone may be preferred to water based because many water based lubricants have high osmolality that can cause mucosal damage, irritation, itching, and burning.[22]

The 2014 American Society for Colposcopy and Cervical Pathology guidelines and 2021 European guidelines recommend a multi-modal approach, with pelvic floor physiotherapy alongside psychosocial therapies as first line treatment for patients with vulvodynia. These treatments are recommended for both localised and generalised vulvodynia, although the benefits of pelvic floor physiotherapy in unprovoked vulvodynia are not established.[5 16]

A 2025 systematic review, including eight RCTs of 689 participants with any type of vulvodynia, evaluated various forms of psychotherapeutic interventions.[26] Six studies compared cognitive behavioural therapy (three individually, two in groups, and one in couples) with either drug treatments (topical corticosteroids, topical lidocaine), or with other methods of psychotherapy. Within this review, four studies showed a statistically significant reduction in vulval pain in the intervention groups (types of psychotherapy) compared with the control groups (two drug treatment groups, and two different types of psychotherapy groups).

Table 2 (bmj.com) shows oral neuropathic pain medications recommended as second-line treatments in international guidelines.[5 16] However, if vulvodynia is generalised, we advise that they be considered first line.

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

A patient with vulvodynia who runs an international patient podcast and support network reviewed this article. Their feedback led to an emphasis on how vulvodynia affects quality of life, and that urinary symptoms are commonly associated with vulval pain. We emphasised hormonally mediated vulval pain because, as the patient noted, it is often missed and treatable. Furthermore, the patient advised to remove recommendations for topical lidocaine as its use has become controversial.

EDUCATION INTO PRACTICE

- How do you investigate for possible causes of vulval pain in your history and examination?
- How often do you refer patients which chronic vulval pain to pelvic floor physiotherapy or any kind of psychotherapy?
- In whom might you offer a trial of amitriptyline or gabapentin?

Competing interests: None declared.
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STATE OF THE ART REVIEW

Advances in the management of acute decompensated heart failure

Michael Gottlieb,[1] Fernanda Bellolio,[2] Brit Long,[3] Alan Storrow[4 5]

Full author details on bmj.com

Correspondence to: M Gottlieb michaelgottliebmd@gmail.com

This is a summary of Clinical Review Advances in the management of acute decompensated heart failure. The full version can be read here: https://www.bmj.com/content/391/bmj-2025-084242

0.5 HOURS

Heart failure affects millions of people worldwide. Progressive worsening of symptoms, referred to as acute decompensated heart failure (ADHF), is associated with a high rate of unplanned emergency department visits, frequent hospital admissions, and increased risk of mortality.[1] The approach used to diagnose and manage ADHF has evolved over time, and there is a need to understand the current state of the science.

Epidemiology

Approximately 1.4% of all emergency department visits are for heart failure, and nearly three quarters are admitted to hospital.[7] Most people admitted to hospital with ADHF will have a known history of heart failure, with one study reporting an outpatient heart failure diagnosis in 73.4% and treatment for heart failure in 64.9%.[8] Inpatient mortality ranges from 4% to 12% and can be as high as 25% in patients at high risk.[6 9 10] The one year survival rate among those with heart failure is 86.5%, which has been declining over the past decade.[11 12] Among those requiring hospital admission for ADHF, the mortality rate markedly increases.[5]

Diagnosis

History and physical examination

The evaluation should begin with a detailed history and focused physical examination. This should include history of heart failure (including ejection fraction and last echocardiogram, if known), drugs taken (including recent changes or missed doses), dietary changes, weight changes, and relevant associated medical conditions (such as coronary artery disease, hyperthyroidism). Assessment of acute symptoms should include the time course and severity, comparison with previous episodes of ADHF, and any interventions attempted (such as increases in diuretic dosing). Dyspnoea should be quantified using changes in level of orthopnoea and distances walked before experiencing dyspnoea. Changes in total body weight and urine output should also be noted. Because heart failure can overlap with other conditions, it is important to use a combination of historical features, physical examination findings, and testing to determine whether ADHF is the cause of the patient's symptoms. Importantly, existing clinical decision tools for ADHF are intended to assess the risk of adverse outcomes to inform admission or discharge decisions, rather than to diagnose heart failure.[33]

There is no single diagnostic test for ADHF; the diagnosis is based on the combination of all clinical data. The presence of a third heart sound or ventricular filling gallop (S3) helps to rule in ADHF (positive likelihood ratio 4.0), while the presence of fever assists with ruling it out (negative likelihood ratio 0.4).[34]

Evaluation for precipitating factors could also help diagnosis. These factors include drug or diet non-adherence (excess salt or fluid intake, unable to fill the drug prescriptions or take as recommended), renal failure (especially missed dialysis), poorly controlled hypertension, iatrogenic (recent addition of negative inotropic drugs, starting salt retaining drugs such as non-steroidal anti-inflammatory drugs, steroids, thiazolidinediones, inappropriate treatment reduction, or new dysrhythmic agents), and substance abuse (cocaine, methamphetamines, ethanol).

Electrocardiography

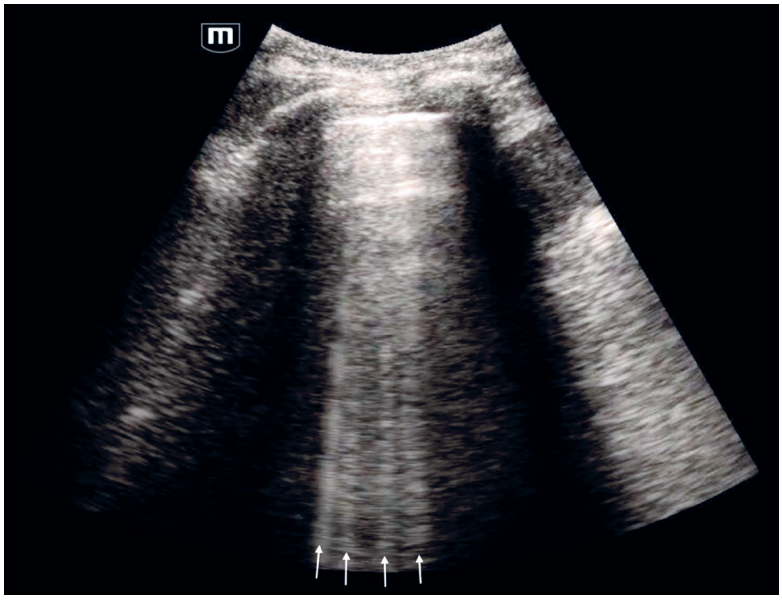
Although electrocardiogram findings are not effective in confirming or excluding ADHF, all patients with ADHF should undergo electrocardiography to help identify alternative causes (such as ST elevation myocardial infarction, pericarditis) and evaluate for dysrhythmias requiring targeted interventions for heart rate control.

Chest radiography

Chest radiographs are commonly ordered in ADHF to identify the presence and degree of pulmonary oedema, and to assess for alternative causes (such as pleural effusion, pneumonia, pneumothorax).[7] The progression of heart failure on chest radiography has been proposed to follow three stages.[36] Stage 1 consists of redistribution of pulmonary vessels, increased cardiothoracic ratio,

WHAT YOU NEED TO KNOW

- Heart failure affects millions of people worldwide and is associated with substantial morbidity and mortality
- Acute decompensated heart failure refers to the worsening of symptoms that requires changes in drugs or the start of new treatments such as non-invasive positive pressure ventilation
- This review summarises the current data and provides an evidence based approach to the diagnosis and management of acute decompensated heart failure



B-lines (white arrows) on point-of-care lung ultrasound suggestive of pulmonary oedema

and a broad vascular pedicle. Stage 2 involves interstitial oedema, which includes Kerley B-lines, peribronchial cuffing, hazy contour of vessels, and subpleural oedema. Stage 3 involves alveolar oedema, which can present with consolidations, a butterfly appearance, cotton wool appearance, and pleural effusions. Although these findings are modestly specific for ADHF, their absence does not exclude the diagnosis.[34] Approximately 20% of patients attending the emergency department subsequently diagnosed with ADHF have chest radiographs without evidence of congestion.[37]

Point-of-care ultrasound

Point-of-care ultrasound (POCUS) is a valuable tool allowing for rapid bedside diagnosis. This test can help to reveal emergent causes of dyspnoea, such as cardiac tamponade or pulmonary embolism (such as evidence of acute right heart strain), and can estimate left ventricular function and volume status.[38]

Lung POCUS involves assessment for pulmonary oedema, which is defined as the presence of at least three B-lines (hyperechoic imaging artefacts extending two thirds of the length of the ultrasound screen) involving at least two areas bilaterally (figure).[39] A systematic review and meta-analysis showed lung POCUS is more sensitive (91.8% *v* 76.5%) and more specific (92.3% *v* 87.0%) than chest radiography for detecting pulmonary oedema in ADHF.[40] Because bilateral B-lines can be found in conditions not caused by pulmonary oedema (such as pulmonary fibrosis, pulmonary contusion, bilateral pneumonia), rapid assessment for raised central venous pressure may follow. An inferior vena cava diameter >2 cm or collapsibility index of <30% is indicative of raised central venous pressure.[42]

Cardiac POCUS can be used in a complementary manner to determine the ejection fraction and diastolic dysfunction, as well as alternative causes such as pericardial effusion or right ventricular dysfunction from pulmonary embolism.[43-45] With training, emergency physicians have reasonable agreement with cardiology interpretations by classifying a visual POCUS estimation of left ventricular ejection fraction into broad categories of normal, moderately reduced, and severely reduced.[46]

Laboratory testing

Laboratory testing can be beneficial to identify potential causes and complications of ADHF. Common testing includes a complete blood count, electrolytes, creatinine, liver function testing, troponin, and B-type natriuretic peptide (BNP) or N-terminal proB-type natriuretic peptide (NT-proBNP). The complete blood count can evaluate for anaemia as a potential mimic of ADHF. Electrolytes are valuable because many diuretics can cause electrolyte imbalances. Creatinine and liver function testing can identify renal impairment and hepatic congestion, respectively, which can influence the differential diagnosis and inform prognosis. Troponin can be useful for determining whether acute coronary syndrome is present in patients with suggestive symptoms, as well as for prognosis.

Management

The initial management of ADHF should include haemodynamic stabilisation and symptom relief.[32 56] Delays in diagnosis and treatment can worsen morbidity and mortality, with data suggesting an adjusted odds of death increasing by 6.8% for each six hour delay in treatment.[57]

Non-invasive ventilation

Evaluation of oxygenation and respiratory status should be the immediate first step in the emergency department. Non-invasive positive pressure ventilation (NIPPV, including continuous positive airway pressure and bilevel positive airway pressure) should be started rapidly in those presenting with acute respiratory distress to improve oxygenation and reduce work of breathing.[58] Successful NIPPV requires haemodynamic stability, facial anatomy allowing a facemask seal, monitoring, and patient cooperation. In patients who cannot tolerate NIPPV, high flow nasal cannula may be considered.[61]

Nitroglycerin

For patients with adequate blood pressure, intravenous vasodilators should be used to reduce afterload and optimise preload, thereby improving symptoms and reducing congestion. These agents are particularly useful in patients with severe hypertension or acute pulmonary oedema.[63-65] Nitroglycerin is the drug of choice in patients with ADHF and hypertension (defined as a systolic blood pressure >160 mm Hg).[65-67] An initial dose of 400 µg sublingually (tablets or spray) can be given while obtaining intravenous access. Once intravenous access is established, a nitroglycerin infusion should be started. Studies have shown that an initial high dose bolus of 1000-2000 µg is well tolerated and can lead to improved patient symptoms and oxygen saturation, and reduced rates of intensive care unit admission.[65-68]

A starting intravenous infusion dose of 0.5-0.7 µg/kg/min is common and titrated every few minutes up to 200 µg/min based on blood pressure and symptoms. Patients should be closely monitored to prevent hypotension. Flow limiting, preload dependent states such as aortic stenosis, right ventricular infarction, and hypertrophic cardiomyopathy, and patients with volume depletion are at increased risk of vasodilator associated hypotension.[69]

Other vasodilators

If additional arterial vasodilation is needed despite high dose nitroglycerin and NIPPV, intravenous angiotensin converting enzyme inhibitors or dihydropyridine calcium channel blockers may be considered. However, these agents should remain second line only after sufficiently titrated doses of nitroglycerin and NIPPV have been administered.[72] Nitroprusside dilates venous and arterial vessels, but is less preferred than the other agents because of increased risks of hypotension.[73 74] Owing to their mechanisms, intravenous vasodilators may be more effective than diuretics for patients with acute pulmonary oedema caused by increased afterload and fluid redistribution to the lungs, even when there is minimal total body fluid accumulation.[32 64 65 75]

Diuretics

Among patients with fluid overload, diuretics increase the excretion of water and salt. Intravenous loop diuretics, such as furosemide, are usually administered. If a patient is already on a diuretic regimen, the intravenous equivalent of double their home dose is a reasonable first approach, with adjustment based upon clinical response. If the patient presents with new onset heart failure or is not on maintenance diuretic treatment, intravenous furosemide 40 mg is an acceptable starting dose.[63]

Diuretic response should be evaluated by monitoring urine output or urine sodium with a goal of 100-150 mL/h during the first six hours, or urine sodium content 50-70 mEq/L at two hours.[86 87] If response is inadequate, doses can be doubled, and if still insufficient, additional diuretics acting at different sites of the renal system (such as thiazides, metolazone, acetazolamide) can be considered with careful monitoring of electrolytes and renal function.[63 88-90]

Mineralocorticoid receptor antagonists such as spironolactone or eplerenone are commonly used for chronic management because of their role in fluid retention and cardiac remodelling. Mineralocorticoid receptor antagonists can also be added in the acute setting as long as potassium and renal function are not a concern, although the evidence of effectiveness is mixed.[96]

Importantly, a subset of patients with ADHF may be isovolaemic with the primary driver of symptoms being sympathetic overactivation resulting in acute hypertension and pulmonary oedema. This group, referred to as sympathetic crashing acute pulmonary oedema (SCAPE), benefits primarily from vasodilation and NIPPV and may not require routine diuresis.[97]

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS MANUSCRIPT

We discussed this review with patients with lived heart failure experiences. They emphasised the importance of clear communication and explaining information in patient centric terminology and language.

Ultrafiltration

In the setting of diuretic refractory fluid overload, ultrafiltration or renal replacement therapy may enable more rapid weight loss and reduce readmission for heart failure.[98] Ultrafiltration also carries an increased risk of adverse events, including raised creatinine, bleeding complications, and intravenous catheter related complications.[100]

Vasopressors and inotropes

A subset of patients will have ADHF with cardiogenic shock, resulting in poor forward flow and hypotension. Cardiogenic shock is a life threatening hypotension with rapidly escalating inotropic or pressor support, and critical organ hypoperfusion (often confirmed by worsening acidosis and lactate levels).[26] Other criteria can include hypotension (systolic blood pressure <90 mm Hg or mean arterial pressure <60 mm Hg), low cardiac output (cardiac index <2.2 L/min/m^2), or pulmonary capillary wedge pressure >15 mm Hg.[26]

These patients often require a combination of vasopressors and inotropic agents.[75] Noradrenaline can be used as a first line agent based on its ability to provide vasoconstriction and inotropic benefits. Inotropes may improve haemodynamics, reduce congestion, and increase cardiac output, thereby improving peripheral perfusion. Among patients with low cardiac output and peripheral hypoperfusion, requiring inotropic support, data suggest that milrinone or dobutamine are reasonable, with milrinone showing a slightly lower mortality rate in overall ADHF compared with dobutamine; however, no difference was seen in the subgroup with ADHF and cardiogenic shock.[102] Close communication with a heart failure specialist is recommended in patients with ADHF requiring vasopressors or inotropes.

Guidelines

Several clinical practice guidelines and consensus documents exist for the management of ADHF. Overall, these recommend the use of biomarkers; chest radiographs and echocardiography to assess heart size, pulmonary congestion, and rule out other causes; use of risk scores to estimate mortality risk; maintenance or optimisation of guideline directed medical treatment during hospital admission; use of intravenous diuretics for fluid overload; venodilators or vasodilators for afterload; and inotropic support or temporary mechanical circulatory support to maintain systemic perfusion and end organ function among those with cardiogenic shock.[26 28 125 126]

Competing interests: None declared.

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Find the full version with references at doi: 10.1136/bmj-2025-084242

CPD READING
0.5 HOURS

Help me piece my story together

Jen Higgs describes her experience of delirium and how healthcare professionals can advocate for patients unable to speak up for themselves

In December 2021 I tested positive for covid-19. I knew I was vulnerable as I take medication to suppress my immune system, but nothing could have prepared me for the fight I was about to enter. As I slowly deteriorated, breathing became increasingly difficult until I developed respiratory failure. I was admitted to the intensive care unit and for seven weeks I remained on a ventilator in a coma. I experienced sepsis, multiple organ failure, and had a stroke.

PRIYA SUNDRAM

Moving between different levels of care, I found that some health professionals didn't know my full history

Trapped and helpless

One day, out of the darkness, I heard someone talking to me. They explained I was in hospital but was safe. I launched into a state of delirium where I experienced going on safari in Africa and travelling to the depths of the ocean, all the while I was in bed and wondering why I was there at all. And then one that still haunts me. All dressed in black with only their eyes showing, they floated around the ward staring at me menacingly. I knew they were going to kill me and I was terrified. I felt trapped and unable to ask for help. It was a terrifying place to be and will stay with me for the rest of my life.

As I began to emerge from the delirium, I found it helpful to learn why I was feeling what I was describing. The team in ICU had kept a diary of my time there, which, when I was able to read it, became essential in putting together what had happened to me. This enabled me to reorientate myself back into the world, before I started my long, complex recovery.

Time to listen

The most distressing times during recovery were when I didn't have someone to offer me reassurance and protection. I moved between different levels of rehabilitation care, and found that often the health professionals didn't know my full history. They were tasked with focusing on one area of my health or recovery, and I wish someone had been able to give me their time to discover what was important to me, to view me as a whole person and not just a "stroke survivor."

Recovery doesn't end when you leave hospital. My world will never be the same, and having health professionals acknowledge this and focus on what is important to me makes such a difference. I need to have a say in what my recovery looks like.

Support from those who were there

I am adapting to my new normal. I will forever be thankful to the team in ICU who saved my life. Their kindness and compassion supported me when my family couldn't be with me, and the follow-up service continues four years later. I can seek support and comfort from people who were with me when I was completely unable to speak for myself, and who remind me how far I have come.

WHAT YOU NEED TO KNOW

- Delirium can be frightening, and the effects can stay with patients for an extended period
- Patients in ICU and recovery value healthcare professionals who will advocate for them by offering comfort and stability
- Patients in rehabilitative care may appreciate being able to determine their own recovery

EDUCATION IN PRACTICE

- What support could you give someone who may have experienced delirium?
- How could you help a patient who may not be able to advocate for themselves?

Patient author

Cite this as: *BMJ* 2026;392:r2624

Jen Higgs is author of *Can You Tell Me Where You Are?: my incredible story of ICU and surviving COVID-19*

SPOT DIAGNOSIS

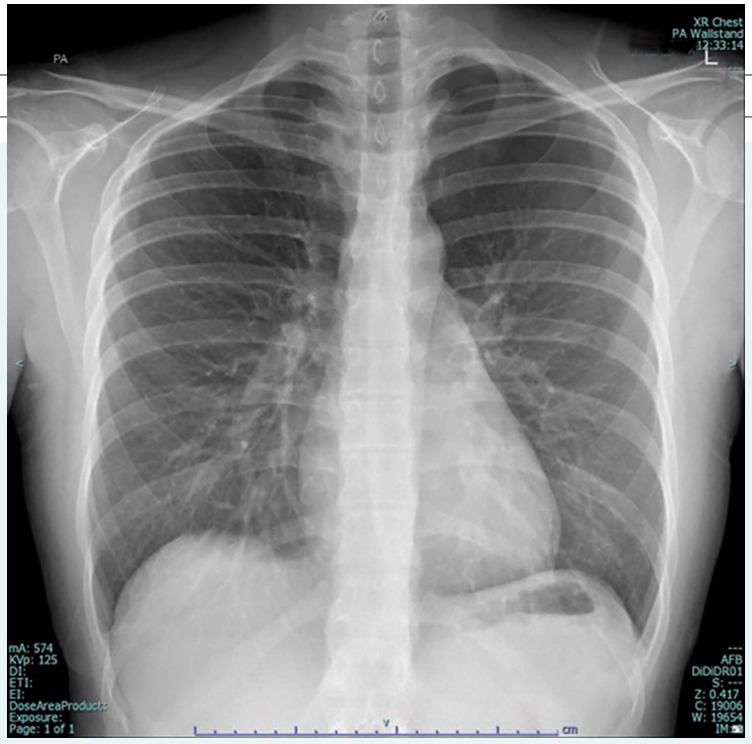
Chest pain and shortness of breath after acupuncture

A woman in her 20s presented to the emergency department with acute onset of shortness of breath and chest pain after removal of an acupuncture needle from the posteromedial aspect of her left scapula. She used acupuncture as part of her pain management strategy for chronic back pain.

At the time of assessment, her symptoms had eased with only mild residual pleuritic chest discomfort persisting. On physical examination, there were no obvious clinical findings and observations including heart rate, respiratory rate, and oxygen saturations were within normal ranges. The patient had never smoked, did not vape, and had no history of respiratory disease.

What is the diagnosis?

Submitted by Samantha A Jones and Helen E Davies
Patient consent obtained.
Cite this as: *BMJ* 2026;392:e085651



Chest radiograph on presentation

If you would like to write an Endgames article, please see our author guidelines at bit.ly/29HCBAL and submit online at bit.ly/29yyGSx

answers

SPOT DIAGNOSIS **Chest pain and shortness of breath after acupuncture**

Left apical pneumothorax secondary to acupuncture needle.

Pneumothorax is a recognised but rare complication associated with acupuncture. It is the most common serious adverse event. The true incidence is unknown owing to delayed presentation and a lack of appreciation of a link.

Pneumothorax occurs more commonly when acupuncture is performed near parasternal and supraclavicular sites. When considering needle depth in acupuncture, different needle lengths are favoured depending on the target area for stimulation of the underlying muscle, nerve, or flow of energy (known as qi). Needle lengths of approximately 3 mm, 10 mm, and 50 mm are typically used to treat the scalp, chest wall, and back or hip, respectively. There are no clearly defined safe needle insertion depths, and the risk of complication is dependent on many variables (eg, body mass index and muscle mass).

Although iatrogenic pneumothorax is a separate entity from spontaneous pneumothorax, expert opinion often recommends a similar management approach to that of spontaneous pneumothorax, where intervention is generally considered unnecessary. Updated British Thoracic Society guidelines were published in 2023 with a shift in focus from previous iterations that were based on thresholds to the adoption of an individual, personalised approach. Conservative management (observation) of patients with small primary spontaneous pneumothoraces with minimal symptoms, or those who are asymptomatic, independent of the size of the pneumothorax, is advocated. The size of the pneumothorax is no longer used as a criterion to determine management.

If intervention is required (eg, patient preference or symptoms), options include needle aspiration, chest drain placement (requiring admission), or siting of an ambulatory chest drain (when expertise and facility exist).

LEARNING POINTS

- Pneumothorax is a rare but serious adverse event of acupuncture performed in the chest area.
- Management of pneumothorax is determined by the severity of symptoms and patient preference. Intervention is seldom necessary for patients with a minimally symptomatic iatrogenic pneumothorax.
- The size of the pneumothorax should not influence the doctor's decision to intervene.

PATIENT OUTCOME

Our patient was managed conservatively with observation in the emergency department and follow-up was arranged four days later. A repeat x ray image of the chest showed resolution of the pneumothorax, and she was discharged.

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 learning.bmj.com.