education

ART OF MEDICINE

A delicate gesture

A bunch of flowers is brought to my office, paraded through the waiting room of patients, a glorious ensemble of tulips overflowing with thanks. Pride swells as I receive them.

I am new to the practice and have stumbled my way through my first few months. My trainer is always encouraging, but this is a tangible boost—a present from a real patient—evidence that I actually helped someone.

I read the accompanying card and my soaring pride plummets. A tentative invitation from a young male patient, would I go for a drink? No miraculous cure, no relief, no note for my portfolio.

As I shuffle the flowers to the windowsill, I know that no relationship could exist. The General Medical Council guides my path fairly to consider the imbalance of power, the patient's potential vulnerability. So I say a simple thank you, write a note about the potential conflict of interest on his records, and arrange transfer of care to another doctor.

I move on to the next patient—a child with conjunctivitis—pausing briefly to reflect in the mirror. Can patients see past me being a young woman to see the doctor I am? Will they ever see past the doctor I am to the person underneath? And should they be able to?

A gesture of kindness and concern for my next patient comes with the delicate edge of restraint. As the tulips lighten my room I consider the merits of a script for chloramphenicol in this strange and subtle art of general practice.

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We welcome contributions to this column. Please email samuel.parker@bmj.com.

CLINICAL UPDATES

Better transition between health and social care

Lack of integration between services and support for adults with social care needs can lead to delayed transfers of care, readmissions, poor care, and avoidable admissions to residential or nursing care, says the National Institute for Health and Care Excellence. New guidance considers how person centred care and support should be planned and delivered during admission to, and discharge from, hospital. It discusses how services should work together and with the person, the family, and carers to ensure that transitions are timely, appropriate, and safe.

www.nice.org.uk/guidance/ng27

RCGP launches new resource on GP federations

The Online Learning Network commissioned by NHS England seeks to support general practitioners, practice staff, and organisations, including clinical commissioning groups, who are interested in scaling up into larger general practice organisations. This new resource includes bespoke tools and case studies, provided by the Nuffield Trust. Mike Holmes, clinical lead for the supporting federations programme, said: "There is no doubt that the challenges we are currently facing in general practice are contributing to the decision to work at scale. We hope that through our Online Learning Network we can encourage federations to share their experiences for the benefit of our patients."

https://federations.rcgp.org.uk

Improving care in cosmetic surgery

The Royal College of Surgeons is developing new services and tools to improve care for patients undergoing cosmetic surgery. The aim is to help patients make more informed decisions and to introduce a new certification system. These changes should come into practice by the middle of next year.

www.rcseng.ac.uk/surgeons/surgical-standards/working-practices/cosmetic-surgery/cosmetic-surgery

EDUCATION INTO PRACTICE

Ideas to make an impact on patient care and quality improvement

BMJ Quality

Palliative care in COPD

Although chronic obstructive pulmonary disease (COPD) is common and a leading cause of death, many patients do not benefit from palliative care. Consider these four criteria when thinking about referral:

1. Would you be surprised if this patient were alive in 6-12 months?
2. Is the patient's FEV_1 (forced expiratory volume in one second) ‹30%?
3. Is the patient on long term oxygen therapy?
4. Is the patient's Medical Research Council dyspnoea score ›3 or COPD assessment test score ›20?

How could you ensure that patients you see with COPD are not missing out on palliative care?

To develop this idea further as a quality improvement project, visit BMJ Quality at http://quality.bmj.com.

CPD/CME

You can gain CPD points from your reading by recording what you have read in your appraisal folder. You should try to link your reading back to a learning need and also consider how you plan to improve your practice as a result of your learning. http://learning.bmj.com

FIG 2 | **Red flags for clinical dehydration**[4]

No clinically detectable dehydration	Clinical dehydration	Hypovolaemic shock
Alert and responsive	Altered responsiveness (for example, irritable, lethargic)	Decreased level of consciousness
Appears well	Appears to be unwell or deteriorating	–
Eyes not sunken	Sunken eyes	–
Moist mucous membranes (except after a drink)	Dry mucous membranes (except for "mouth breather")	–
Normal blood pressure	Normal blood pressure	Hypotension (decompensated shock)
Normal breathing pattern	Tachypnoea	Tachypnoea
Normal capillary refill time	Normal capillary refill time	Prolonged capillary refill time
Normal heart rate	Tachycardia	Tachycardia
Normal peripheral pulses	Normal peripheral pulses	Weak peripheral pulses
Normal skin turgor	Reduced skin turgor	–
Normal urine output	Decreased urine output	–
Skin colour unchanged	Skin colour unchanged	Pale or mottled skin
Warm extremities	Warm extremities	Cold extremities

Within the category of "clinical dehydration" there is a spectrum of severity indicated by increasingly numerous and more pronounced clinical features. For hypovolaemic shock, one or more of the clinical features listed would be expected to be present. Dashes (–) indicate that these features do not specifically indicate hypovolaemic shock. This figure has been adapted from the assessing dehydration and shock section in 'Diarrhoea and vomiting in children' (NICE guideline CG84)[4]

WHAT YOU NEED TO KNOW

- Careful assessment and monitoring of body weight, fluid balance, and fluid status are essential during intravenous fluid therapy in children, as is the correct choice of fluid, to avoid serious complications including death and neurological injury
- To reduce anxiety and improve compliance with blood tests, explain their importance to children who are old enough to understand and to their carers; consider distraction techniques and comfort measures in younger children and use topical local anaesthetics before taking blood
- Isotonic crystalloids with a sodium content of 131-154 mmol/L are appropriate for initial maintenance requirements
- In children receiving intravenous fluids, symptoms such as nausea and vomiting, lethargy, confusion, and irritability may indicate hyponatraemia. This is a medical emergency requiring immediate expert advice and treatment

Inappropriate use of intravenous fluids in children may have serious consequences. These include death or permanent neurological injury from hyponatraemia,[1] hypovolaemia, and poor organ perfusion, as well as the risks of hypervolaemia, oedema, and heart failure. Children have different fluid requirements from adults, for whom specific guidance exists.[2] This article summarises the most recent recommendations from the National Institute for Health and Care Excellence (NICE).[3]

Take into account pre-existing conditions (such as cardiac disease or renal disease), as these may require smaller fluid volumes.

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

The guideline committee included lay members who contributed to the formulation of the recommendations summarised here.

WHAT'S NEW IN THIS GUIDANCE

- Intravenous fluids are potentially dangerous; they should be used only when clinically indicated and with close observation and assessment
- Recognise that children are at greater risk than adults of permanent neurological complications and death due to hyponatraemia from inappropriate use of intravenous fluids

Fluid resuscitation

- If children and young people need intravenous fluid resuscitation, use glucose-free crystalloids that contain 131-154 mmol/L sodium, with a bolus of 20 mL/kg over less than 10 minutes. Take into account pre-existing conditions (such as cardiac disease or renal disease), as these may require smaller fluid volumes.
- If term neonates need intravenous fluid resuscitation, use glucose-free crystalloids that contain 131-154 mmol/L sodium, with a bolus of 10-20 mL/kg over less than 10 minutes.

Replacement and redistribution

For term neonates, children, and young people:

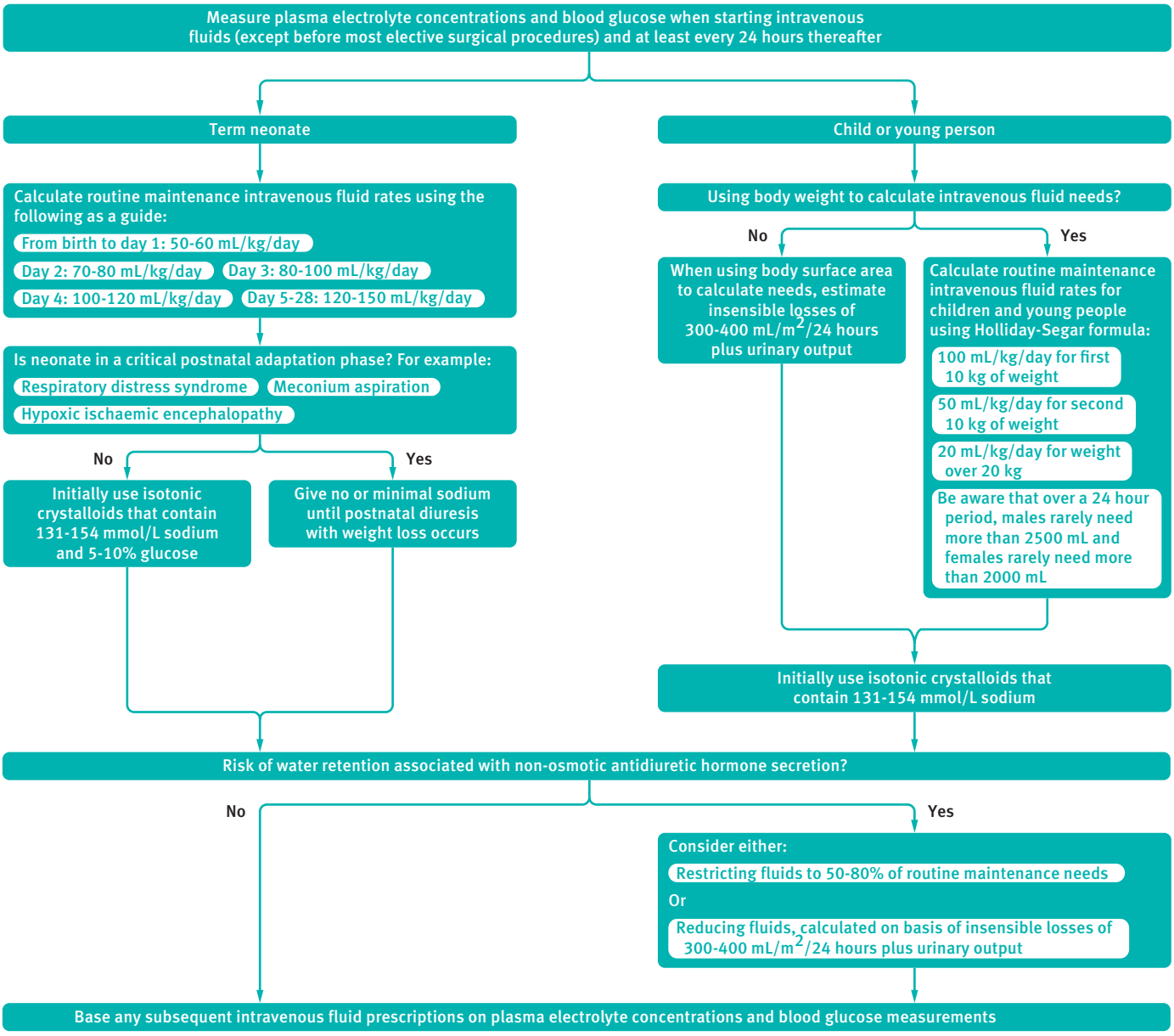
- Adjust the intravenous fluid prescription (in addition to maintenance needs) to account for existing fluid and/or electrolyte deficits or excesses, ongoing losses (see fig 4), or abnormal distribution (for example, tissue oedema in sepsis).
- Consider isotonic crystalloids that contain 131-154 mmol/L sodium for redistribution.
- Use 0.9% sodium chloride solution containing potassium to replace ongoing losses.
- Base any subsequent fluid prescriptions on plasma electrolyte concentrations and blood glucose measurements.

Hypernatraemia that develops during intravenous fluid therapy

Review the fluid status and take action as follows:

- If there is no evidence of dehydration and an isotonic fluid is being used, consider changing to a hypotonic fluid (such as 0.45% sodium chloride with glucose).

FIG 3 | **Algorithm for routine maintenance**



- If dehydration is diagnosed, calculate the water deficit and replace it over 48 hours, initially with 0.9% sodium chloride.
- If the fluid status is uncertain, measure urine sodium and osmolality.
- If hypernatraemia worsens or is unchanged after replacing the deficit, review the fluid type and consider changing to a hypotonic solution (such as 0.45% sodium chloride with glucose).
- When correcting hypernatraemia, ensure that plasma sodium does not fall by more than 12 mmol/L in a 24 hour period.
- Measure plasma electrolyte concentrations every 4-6 hours for the first 24 hours; after this base the frequency of further plasma electrolyte measurements on the treatment response.

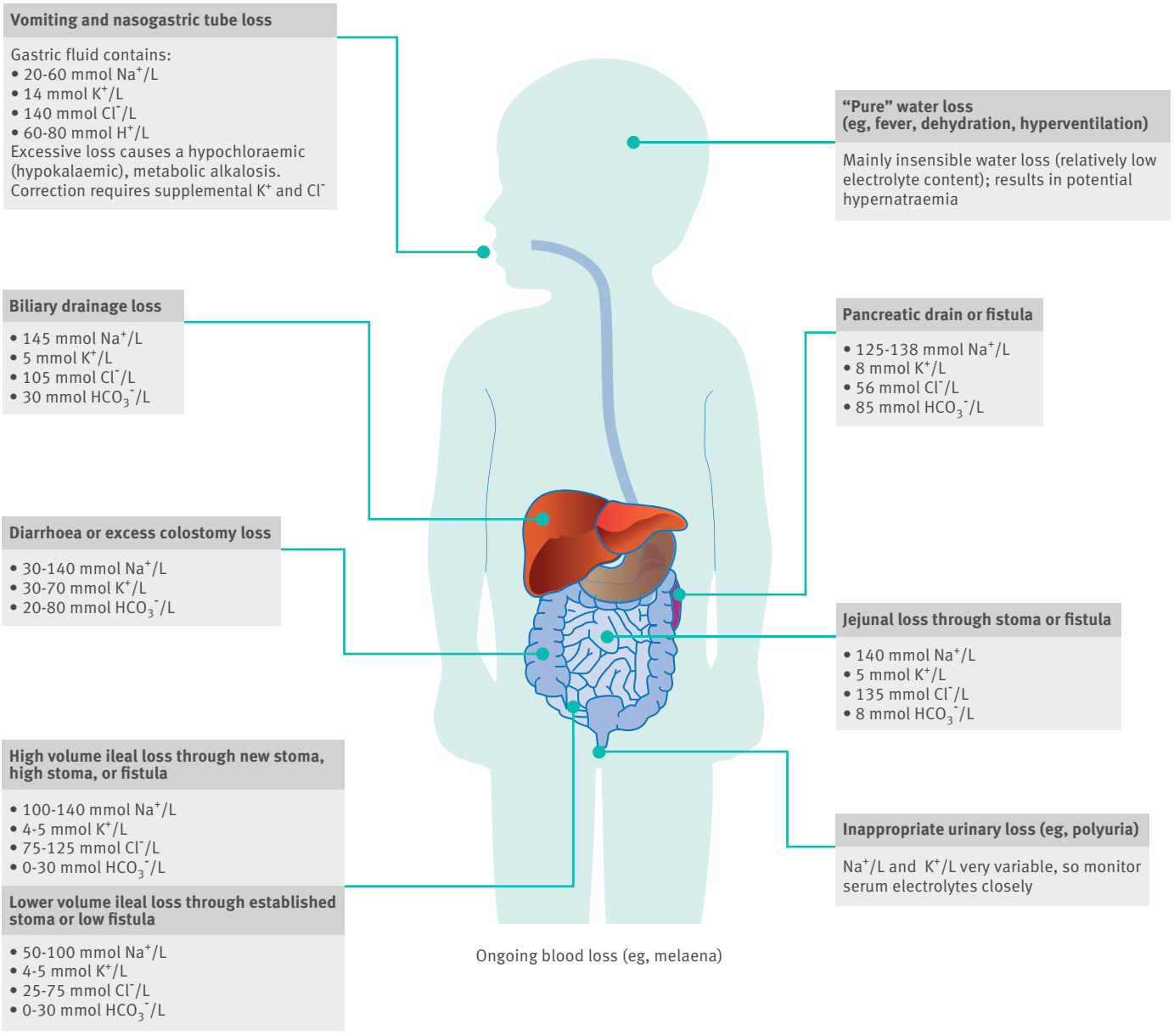
If hypernatraemia worsens or is unchanged after replacing the deficit, review the fluid type

Asymptomatic hyponatraemia that develops during intravenous fluid therapy

This is potentially dangerous and associated with permanent neurological damage and death in children.

- If hyponatraemia develops in term neonates, children, and young people, review the fluid status, and if the child is being prescribed a hypotonic fluid change to an isotonic fluid (such as 0.9% sodium chloride).
- In children and young people who are hypervolaemic or at risk of hypervolaemia (for example, if increased antidiuretic hormone secretion is possible), either:
  - Restrict maintenance fluids to 50-80% of routine maintenance needs, or
  - Reduce maintenance fluids, calculated on the basis of insensible losses within the range 300-400 mL/m^2/24 h plus urinary output.

FIG 4 | **Ongoing fluid and electrolyte losses in children. Reproduced with permission from the National Clinical Guideline Centre**



Acute symptomatic hyponatraemia that develops during intravenous fluid therapy

This is associated with the following symptoms:

- Headache
- Nausea and vomiting
- Confusion and disorientation
- Irritability
- Lethargy
- Reduced consciousness
- Convulsions
- Coma
- Apnoea

In those who develop acute symptomatic hyponatraemia, do not manage acute hyponatraemic encephalopathy using fluid restriction alone. Instead, review the fluid status, seek immediate expert advice (for example, from the paediatric intensive care team), consider taking action as follows:

- Use a bolus of 2 mL/kg (maximum 100 mL) of 2.7% sodium chloride over 10-15 minutes.
- Use a further bolus of 2 mL/kg (maximum 100 mL) of 2.7% sodium chloride over the next 10-15 minutes if symptoms are still present after the initial bolus.
- If symptoms are still present after the second bolus, check the plasma sodium level and consider a third bolus of 2 mL/kg (maximum 100 mL) of 2.7% sodium chloride over 10-15 minutes.
- Measure the plasma sodium concentration at least hourly.
- As symptoms resolve, decrease the frequency of plasma sodium measurement on the basis of the response to treatment.
- After hyponatraemia symptoms have resolved, ensure that plasma sodium does not increase by more than 12 mmol/L in a 24 hour period.

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Biomarkers in rheumatic diseases

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This is an edited version of the state of the art review. The full version is on thebmj.com under the title "Biomarkers in rheumatic diseases: how can they facilitate diagnosis and assessment of disease activity?"

Rheumatoid arthritis

Evidence shows that early diagnosis combined with the timely use of disease modifying anti-rheumatic drugs (DMARDs) improves the disease course of rheumatoid arthritis.[5-7] The challenge is therefore to identify the best markers to diagnose and monitor the disease. Leading laboratory markers that have been clinically useful include autoantibodies, acute phase reactants, bone and cartilage markers, and various cytokines.

The American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) proposed new classification criteria for rheumatoid arthritis in 2010 (fig 1).

Autoantibodies

Rheumatoid factor

Rheumatoid factors are antibodies that are directed against IgG isotype antibodies. They are detected in the serum of 70-80% of patients with rheumatoid arthritis but are not specific for rheumatoid arthritis. They can be detected in other systemic diseases, such as Sjögren's syndrome and systemic infections, and in about 10% of healthy people.

As well as having a role in diagnostics, rheumatoid factor can also be used in prognosis. Several studies have shown that higher levels of rheumatoid factor are associated with more severe disease marked by disease progression, rheumatoid nodules, and various extra-articular manifestations.[6 7] Rheumatoid factor may also reflect the response to treatment, as exemplified in treatment studies using gold salts and DMARDs.[9 10]

WHAT YOU NEED TO KNOW

- Rheumatoid factors are detected in the serum of 70-80% of patients with rheumatoid arthritis and in about 10% of healthy people.
- Anti-cyclic citrullinated protein (CCP) antibodies are the most specific for rheumatoid arthritis, with 67% sensitivity (95% confidence interval 62% to 72%) and specificity of 95% (94% to 97%).
- Anti-phospholipid antibodies (including anti-cardiolipin antibodies and the lupus anticoagulant) are present in a third of patients with SLE.
- Although 85-95% of white patients with ankylosing spondylitis have HLA-B27, only 6% of HLA-B27 carriers in the general population develop the condition.
- In patients with ankylosing spondylitis, HLA-B27 positivity is associated with younger age at disease onset.

Anti-cyclic citrullinated protein antibodies

Anti-cyclic citrullinated protein (CCP) antibodies are autoantibodies that react with a wide array of citrullinated peptides and proteins, including anti-perinuclear factor (APF), keratin, the Sa antigen, fibrin, fibrinogen, α enolase, eukaryotic translation initiation factor 4G1, vimentin, collagens, and synthetic CCPs.

Among these, anti-CCP antibodies are the most specific for rheumatoid arthritis, with 67% (95% confidence interval 62% to 72%) sensitivity and specificity of 95% (94% to 97%).[8] It has been described as a complementary marker, because it is useful in rheumatoid factor negative patients with rheumatoid arthritis in the early phase of the disease.[11 12]

Anti-CCP antibodies emerge as early as 10-14 years before the onset of symptoms.[15 16] Evidence about whether anti-CCP antibodies can be used to monitor the response to treatment is conflicting.

Acute phase reactants

Erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) have been widely studied in rheumatoid arthritis. An increases in these acute phase reactants correlates with disease outcomes such as joint erosion, Health Assessment Questionnaire Disability Index (HAQ-DI) scores, radiographic progression, and functional outcomes.[18-20] Inclusion of these acute phase reactants in composite disease activity indices such as the disease activity score 28 (DAS28) reliably predicts disease course (fig 1).[21 22]

Bone and cartilage turnover markers

Although synovial, cartilage, and bone derived markers have limited value in diagnosing rheumatoid arthritis they may be useful in predicting and monitoring disease progression and response to treatment.[25]

Cytokines

Research groups have identified about 20 cytokines and related mediators that are raised in rheumatoid arthritis.[37-40]

An offshoot of these types of studies is the use of a novel commercially available protein biomarker panel to monitor disease activity in rheumatoid arthritis. This panel incorporates various cytokines, chemokines, adhesion molecules, adipokines, and synovial or skeletal markers and has been shown to track disease activity in rheumatoid arthritis.[41-43]

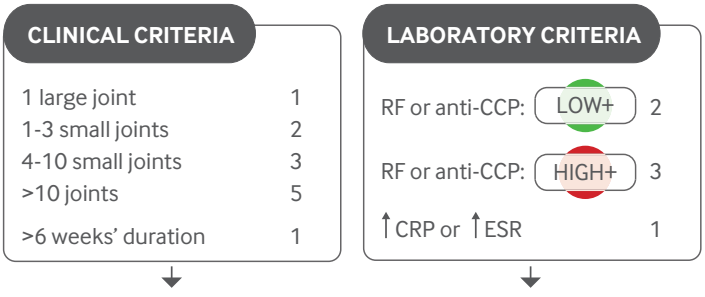


Fig 1| American College of Rheumatology and European League Against Rheumatism (ACR/EULAR) 2010 classification criteria for rheumatoid arthritis

Systemic lupus erythematosus (SLE)

SLE is currently diagnosed by the presence of at least four of 11 criteria outlined by the ACR.[47 48] The Systemic Lupus Erythematosus Collaborating Clinics (SLICC) group revised the classification criteria in 2012 (fig 2).[3]

Autoantibodies

Anti-nuclear antibodies

The ANA test is a broad screening test with sensitivities exceeding 98% for the diagnosis of SLE and lupus nephritis.[53 54] However, the test lacks specificity because ANA can be positive in other autoimmune diseases, thyroid diseases, hepatic diseases, cancers, chronic infections, and elderly people.[53 54] The dsDNA reactive anti-DNA antibodies are more specific for SLE.[53]

Association with disease activity

Antibody titres vary with disease activity and flares, and they are also associated with the presence of lupus nephritis.[56-59] The utility of anti-dsDNA in predicting and monitoring treatment response has been re-validated in more recent clinical trials aimed at depleting B cells using rituximab or blocking the B cell activating factor (BAFF) pathway.[64-67]

Anti-Sm and anti-U1 RNP

Anti-Sm is also useful for the diagnosis of SLE (fig 2). It is more specific than anti-dsDNA antibodies because it has not been described in other rheumatic diseases, but it is less sensitive, being present in only 20-30% of patients.

Anti-U1 RNP is strongly associated with a related overlap disease termed mixed connective tissue disease, marked by the presence of other connective tissue diseases such as inflammatory myositis or systemic sclerosis. The specificity of anti-U1 RNP for mixed connective tissue disease ranges from 85% to greater than 99%.[68]

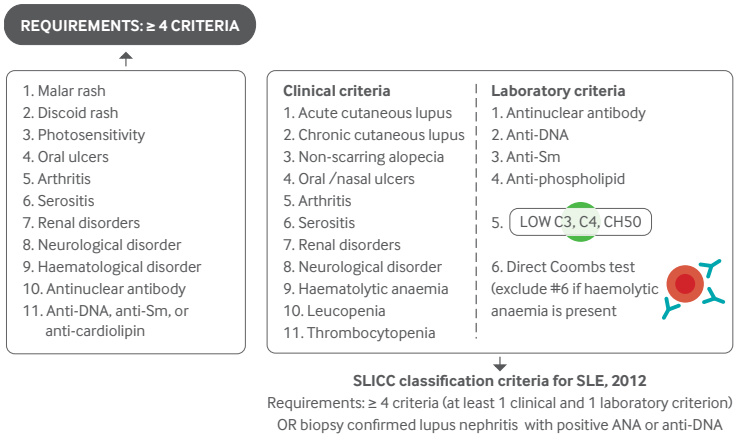


Fig 2| Comparison of the American College of Rheumatology revised classification criteria for systemic lupus erythematosus (1997) and the Systemic Lupus Erythematosus Collaborating Clinics classification criteria (2012). ANA=anti-nuclear antibodies

Anti-SSA

Another anti-nuclear antibody of clinical interest in SLE is anti-SSA/Ro. Although this antibody is highly associated with Sjögren's syndrome and its associated symptoms (dry mouth and dry eyes), it is also present in 30% of patients with SLE and 70-90% of those with subacute cutaneous lupus erythematosus.

Anti-SSB/La

A related antibody, anti-SSB/La (Sjögren's syndrome type B antigen, also known as La (Lupus)), is present in 10% of patients with SLE, 30% of those with subacute cutaneous lupus erythematosus, and 90% of patients with neonatal lupus and congenital heart block.

Anti-phospholipid antibodies

Anti-phospholipid antibodies (including anti-cardiolipin antibodies and the lupus anticoagulant) are present in a third of patients with SLE. Of these, one third go on to develop livedo reticularis and skin ulcers as well as clinical features of anti-phospholipid syndrome, including venous thrombosis, arterial thrombosis, recurrent pregnancy loss, thrombocytopenia, and haemolytic anaemia.[70] These antibodies are also associated with cerebral vascular disease and focal damage in neuropsychiatric SLE.

Reduced complement

Several early studies showed that reduced complement C3 and C4 levels are associated with more severe disease in SLE, including renal flares.[74-76] In recent longitudinal studies, baseline reductions in complement C3 or C4 were shown to predict disease flares two months or even a year before they occurred.[67 77]

CRP and ESR

Although CRP is often raised in SLE, these increases are inconsistently related to disease activity or flares.

ESR has also been evaluated for its potential as a biomarker. In a longitudinal study, raised ESR was associated with disease activity or damage accrual in SLE,[84] independent of the presence of anti-dsDNA antibodies. A subsequent analysis showed that increased ESR was an independent predictor of renal flares two months later and a marker of concurrent renal flare.[77]

Systemic sclerosis

Systemic sclerosis related autoantibodies

Three antibodies—anti-centromere, anti-topoisomerase I, and anti-RNA polymerase III antibodies—are included in the 2013 ACR/EULAR classification criteria for this disease.[2 3 96]

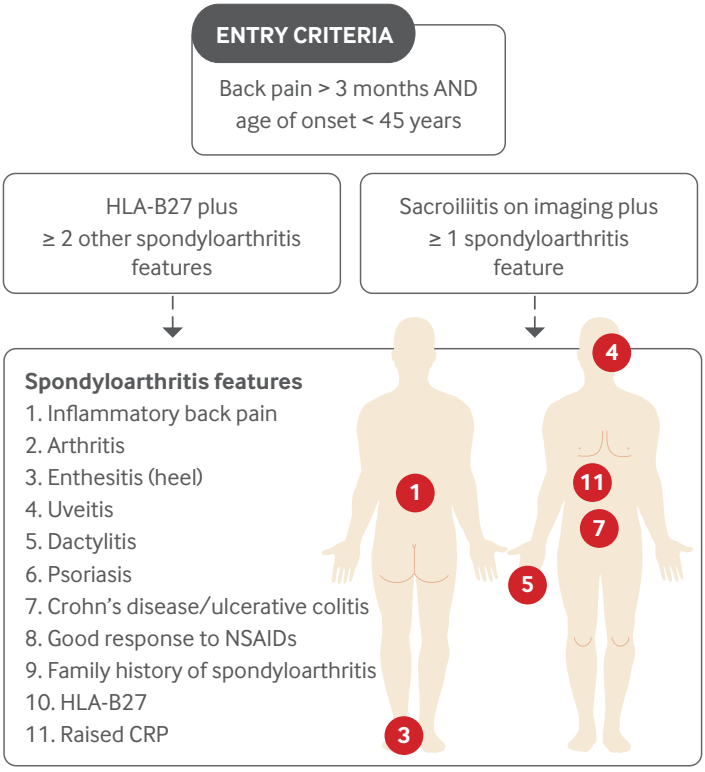


Fig 4 | ASAS classification criteria for axial spondyloarthritis

Systemic sclerosis is classified clinically on the basis of the extent of skin involvement into limited and diffuse subtypes—patients with skin involvement proximal to the elbows and knees are categorised as having diffuse cutaneous involvement.[97] ANAs are present in 96% of patients with systemic sclerosis.[98]

Anti-centromere antibodies are associated with limited cutaneous involvement, absence of clinically significant ILD, and better survival.[95 100 101 103] Anti-RNA polymerase III is also associated with diffuse cutaneous involvement and is strongly linked to the occurrence of scleroderma renal crisis.[104-106] Therefore, patients with systemic sclerosis and anti-RNA polymerase III antibodies should be monitored closely for new onset hypertension. In addition, oral glucocorticoids should be used with caution in patients with systemic sclerosis, especially those with anti-RNA polymerase III antibodies, because these drugs have been linked to the development of scleroderma renal crisis.[107 108]

CRP

In two recent studies, raised CRP was associated with the diffuse cutaneous disease type and more severe skin and lung involvement. More importantly, higher CRP was associated with progressive ILD and shorter survival.[118 119]

Idiopathic inflammatory myositis

Idiopathic inflammatory myositis (IIM) comprises a group of diseases that cause bilateral muscle weakness as a result of muscle inflammation. Polymyositis and dermatomyositis are two typical IIMs. Although several classification criteria have been proposed for these two diseases, none has been validated.[127 128]

Antibodies associated with IIM are subdivided into myositis related antibodies and myositis specific antibodies. Table 2 (see thebmj.com) lists the commercially available myositis antibodies, as well as their prevalence and clinical correlates.

Creatine kinase, aldolase, lactate dehydrogenase, aspartate aminotransferase, and alanine aminotransferase can be raised in IIM. In a case series study from a single centre, all patients with IIM had an elevation of at least one muscle enzyme. Creatine kinase and aldolase were raised in 96% and 86% of patients, respectively.[143]

Axial spondyloarthritis

Axial spondyloarthritis is a chronic inflammatory condition that affects the axial skeleton. Ankylosing spondylitis is the prototype of axial spondyloarthritis. Signs of sacroiliitis on plain radiographs are needed for the diagnosis of ankylosing spondylitis.[145] However, inflammation and pain precede the development of radiographic features of sacroilliitis by several years.[146] Therefore, a second category of non-radiographic axial spondyloarthritis has been defined, the diagnosis of which is based on evidence of active inflammation in the sacroiliac joints on magnetic resonance imaging (MRI) or a combination of other findings. New criteria have been proposed to facilitate early diagnosis of axial spondyloarthritis (fig 4).[147] (Please note fig 3 is on thebmj.com.)

HLA-B27

Although HLA-B27 was not included in the classification criteria for ankylosing spondylitis,[145] it is an integral part of the new ASAS criteria for axial spondyloarthritis.[147] The prevalence of HLA-B27 varies among different ethnic groups. Although 85-95% of white patients with ankylosing spondylitis have HLA-B27,[149] only 6% of HLA-B27 carriers in the general population develop the condition.[150]

In patients with ankylosing spondylitis, HLA-B27 positivity is associated with younger age at disease onset, development of anterior uveitis, and positive family history of spondyloarthritis, but it is not associated with increased structural damage on radiography.[151-153]

HLA-B27 is particularly useful for diagnosing non-radiographic spondyloarthritis.

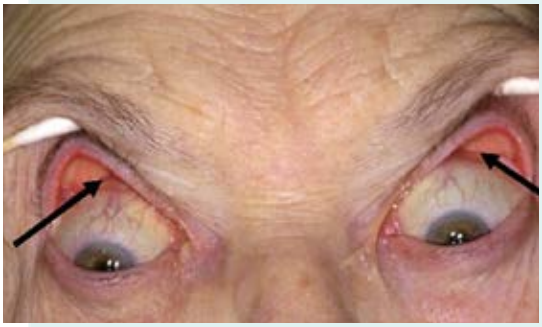
CRP and ESR

Raised CRP is included in the ASAS classification criteria for axial spondyloarthritis.[147] CRP or ESR is raised in 40-50% of patients with ankylosing spondylitis,[153] so a normal ESR or CRP does not rule out this condition. Levels of both of these acute phase reactants are higher in patients with ankylosing spondylitis than in those with non-radiographic axial spondyloarthritis.[155]

Raised CRP is also associated with increased radiographic changes on spinal radiographs[153] and signs of inflammation on sacroiliac MRI.[156] Furthermore, raised CRP and ESR predict future radiographic progression in sacroiliac joints and the spine in patients with ankylosing spondylitis.[157-159]

Cite this as: *BMJ* 2015;351:h5079

Giant fornix syndrome



A 97 year old woman presented to eye casualty with reduced vision and chronic yellow discharge. She had been treated with multiple topical antibiotics without benefit. Examination showed copious purulent discharge from both eyes; her eyelids were floppy with deep upper fornices (arrows) and corneal toxic epitheliopathy. These findings led to the diagnosis of giant fornix syndrome. A conjunctival swab grew *Staphylococcus aureus*. She was treated with systemic and topical antibiotics and referred for conjunctival sac reconstruction surgery. This newly described syndrome is rare. It should be considered in older patients with recurrent chronic purulent conjunctivitis and corneal epitheliopathy. Surgical treatment ensures obliteration of deep pockets of bacterial colonisation.

Shampa Gupta, trust doctor in ophthalmology, **T S Balaji Prasad**, associate specialist in ophthalmology, Department of Ophthalmology, University Hospitals Coventry and Warwickshire NHS Trust, Coventry

Patient consent obtained.

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Doing this and that for stroke

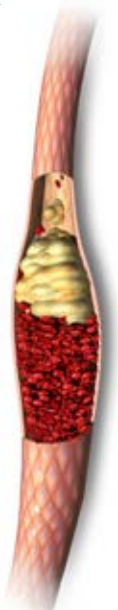
Systematic reviews are meant to help clinicians and health services decide on the best kinds of care to provide patients—for example, after stroke. But random sampling of 60 reviews of non-pharmacological interventions for stroke found that most lacked information on interventions for items sought (*BMJ Open* 2015;5:e009051, doi:10.1136/bmjopen-2015-009051). The most incompletely described items were modifications, fidelity, materials, procedure, and tailoring (missing from all interventions in 97%, 90%, 88%, 83%, and 83% of reviews, respectively).

Could you be an eye surgeon?

Most medical students do not want to be eye surgeons, but a brave few feel drawn to this vocation. Armed with a surgical simulator, ophthalmologists at University College London tested whether such students showed a superior aptitude for ocular procedures (*Br J Ophthalmol* 2015, doi:10.1136/bjophthalmol-2015-307127). They were not significantly different from other students.

Old hardened arteries

In the works of PG Wodehouse, a snorting curmudgeon called Uncle Percy is referred to as "old hardened arteries." This was in 1946, just before the Framingham Heart Study was set up. Since then, cardiovascular cohorts have multiplied across the world, and one from the US has been used to track how cognitive decline correlates with annual recordings of blood pressure as compared with arterial stiffness (*Hypertension* 2015, doi:10.1161/HYPERTENSIONAHA.115.06277). Hardening of the arteries is more predictive.



HENNING DALHOFF/SPL

Struck down with SAH

Sometimes it is good to confirm clinical experience with large cohort studies. In a retrospective analysis, 40% of 1460 patients with subarachnoid haemorrhage (SAH) presented with unconsciousness (*JAMA Neurol* 2015, doi:10.1001/jamaneurol.2015.3188). This was associated with poor clinical grade, more subarachnoid and intraventricular blood seen on admission computed tomography, and a higher frequency of global cerebral oedema. It was also associated with a higher risk of death and severe disability.

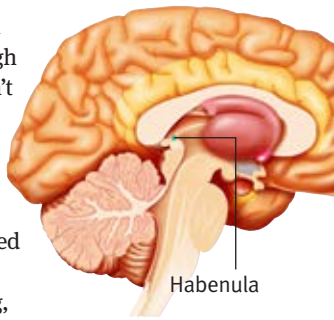
Autistic spectrum and mortality

The diagnosis of autistic spectrum disorder is based on personality traits that seem to have little to do with physical health. But Swedish investigators found that the risk of death in people given this label between 1987 and 2009 was 2.56 times that of the general population (*Br J Psychiatry* 2015, doi:10.1192/bjp.bp.114.160192). Causes of death were spread across almost the whole diagnostic range.

Adenovirus urethritis

It is the season of viral rhinoconjunctivitis, which is often caused by adenoviruses. Some of the 68 serotypes also have an affinity for the male urethral meatus, where they cause soreness for about a week, leading some men to attend sexually transmitted disease clinics (*Sex Transm Infect* 2015, doi:10.1136/sextrans-2015-052243). A review of cases from a Melbourne clinic describes its clinical features, usually a combination of conjunctivitis and meatitis with varying numbers of pus cells in the exudate. It's unclear whether the condition is sexually transmitted or whether it can simply be caused by wiping an eye before going to the lavatory. Minerva's male friends assure her this is a common occurrence.

Have you a habenula?



BSIP SA/ALAMY

Yes, you do have a habenula, although you probably didn't know this. It is found medial to the posterior thalamus and is uniquely positioned to participate in reward processing, acting as a convergence point for the limbic system and basal ganglia circuits. Neuroimagers from Queen Square Hospital London argue that although it is a long way from the frontotemporal cortex, shrinkage of the habenula plays a role in frontotemporal dementia (*J Neurol Neurosurg Psychiatry* 2015, doi:10.1136/jnnp-2015-312067).

If only we'd started sooner

A study using national data from the British Thoracic Society community acquired pneumonia audit finds that time to administration of the first dose of antibiotic (TFA) was less than four hours in 63% of patients. After using propensity matching to reduce bias, investigators found a slightly higher risk of mortality in those who got their antibiotics later (*Thorax* 2015, doi:10.1136/thoraxjnl-2015-207513). But they wisely state that "we cannot say whether this is causal or whether TFA might just be a quality measure for overall or other processes of care."

Cite this as: *BMJ* 2015;351:h6572

CASE REVIEW

A premenopausal woman with abdominal discomfort and iron deficiency anaemia

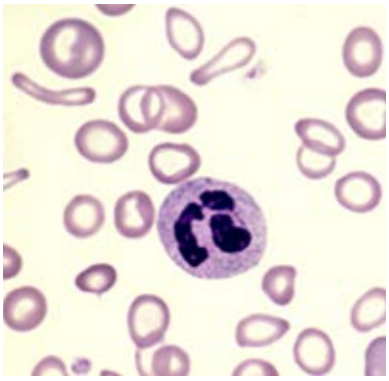
A 48 year old premenopausal woman presented to our clinic with a 12 month history of intermittent abdominal discomfort associated with bloating, constipation, and weight loss of 6.3 kg. Her medical history included chronic iron deficiency anaemia, vitamin D deficiency, and episodes of fresh rectal bleeding caused by haemorrhoids, which required sclerotherapy. She had undergone a diagnostic laparoscopy for abdominal pains, which did not detect any abnormalities. There was no family history of colorectal cancer. On examination she looked well, weighed 50 kg, with a body mass index of 20. Her abdomen was soft, non-tender, and without palpable masses. The results of a digital rectal examination were normal. Routine blood tests showed iron deficiency anaemia, with haemoglobin 109 g/L (reference range 117-155), mean corpuscular volume 80.6 fL (80-100), mean corpuscular haemoglobin 27.3 pg/cell (27-33), and ferritin 4 ng/mL (10-232).

1 What are the most important common causes of iron deficiency anaemia in Western countries?
2 What is the most likely diagnosis in this patient and how would you confirm it?
3 What are the complications of this condition?
4 How would you follow up the patient long term?

Submitted by Abdulkani Yusuf, Bhamini Vadhwana, Ujjwala Mohite, and Carole Collins

Patient consent obtained.

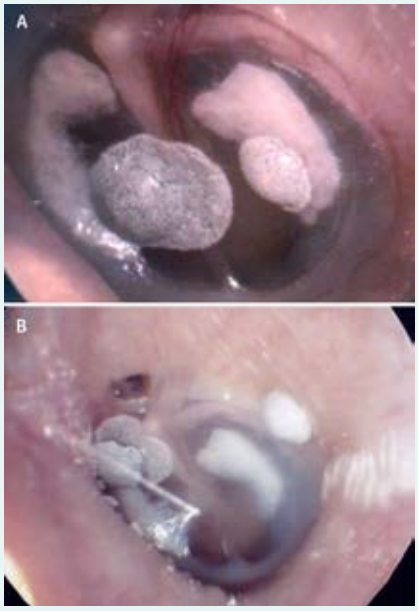
Cite this as: *BMJ* 2015;351:h4664



DR GLADDEN WILLIS/VISUALS UNLIMITED/CORBIS

Severe iron deficiency anaemia showing hypochromic pale haemoglobin deficient microcytic small and misshappen red blood cells in human peripheral blood smear

We welcome contributions that would help doctors with postgraduate examinations. We also welcome submissions relevant to primary care.
See thebmj.com/endgames



Appearance of the tympanic membrane at presentation (A) and four weeks later (B)

CASE SCENARIO

A case of cement in the ear

A 23 year old bricklayer attended the emergency department when cement mortar splashed into his left ear. He irrigated the ear with water at the scene, but unfortunately the cement had set on his tympanic membrane. In the absence of an alkaline burn, and with a mild conductive hearing deficit, he was managed conservatively. Upon review four weeks later, deposits of cement could be seen migrating outwards on to the external auditory canal (figure). His hearing continued to improve as migration occurred.

Learning points:

- This case demonstrates the natural migration of tympanic membrane epithelium
- It supports an ear, nose, and throat led, conservative approach to adherent foreign bodies, relying on natural migration rather than risking damage to the tympanic membrane as a result of surgical removal.

Submitted by Victoria Wilmot, Kim To, Megan Anderson, and Alex Bennett

Patient consent obtained.

Cite this as: *BMJ* 2015;351:h5383

answers

CASE REVIEW

A premenopausal woman with abdominal discomfort and iron deficiency anaemia

1 The most important common causes of iron deficiency anaemia in men and postmenopausal women in Western countries are colorectal cancer and malabsorptive disorders. In premenopausal women menstrual blood loss is the primary cause.
2 The combination of abdominal symptoms, such as bloating and weight loss, iron deficiency anaemia, and vitamin D deficiency suggests an underlying malabsorptive state. The most likely diagnosis is coeliac disease. The diagnosis is suggested by raised tissue transglutaminase antibody (tTGA) and is confirmed by the presence of villous atrophy on duodenal biopsy.
3 Non-compliance with a gluten-free diet is the most important factor predisposing patients to osteoporosis and splenic atrophy. Rare complications include small bowel T cell lymphoma and other gastrointestinal cancers.
4 Patients with coeliac disease require regular follow-up to ensure and encourage compliance with a gluten-free diet and to monitor them for complications and associated autoimmune diseases.