

SELF ASSESSMENT QUESTIONS

Pulmonary nodules and splinter haemorrhages

A Schattner, N Kozak, J Friedman

Answers on p 792.

A 42 year old man developed upper respiratory symptoms that were accompanied by a marked decrease in appetite, severe fatigue, and the later appearance of arthralgia of the ankles and of drenching night sweats. He was admitted in January 2000, after he had been ill for one month, showed no response to cefuroxime, and had a chest x ray which revealed multiple pulmonary nodules (fig 1). The patient was a gardener who smoked heavily and had a distant history of drug abuse but was otherwise healthy. On examination low grade fever (37.7°C), sinus tachycardia (104 beats/min), enlarged red tonsils, right conjunctivitis, and tenderness over one knee, were the only notable findings. The erythrocyte sedimentation rate was 109 mm/hour, haemoglobin concentration 119 g/l (mean corpuscular volume 90 fl), leucocytes $12 \times 10^9/l$ with 75% neutrophils and 6% eosinophils, and platelets $461 \times 10^9/l$. The urinary sediment, renal function tests, and electrolytes were normal. Serum albumin was 29 g/l, globulins 41g/l (polyclonal), aspartate aminotransferase 87 U/l, alanine aminotransferase 240 U/l, lactate dehydrogenase 460 U/l. Serum alkaline phosphatase, γ -glutamyltransferase, amylase, creatine phosphokinase, as well as antinuclear antibodies, rheumatoid factor, and complement were normal. Blood cultures, viral serology (including hepatitis and HIV), and the tuberculin test were negative. Computed tomography of the chest and abdomen did not show any additional findings to the pulmonary nodules except for a 2.5 cm hypodense round mass in the left adrenal. Endocrine studies were normal as was a transthoracic echocardiography. On the fourth hospital day, multiple new splinter haemorrhages were found under the fingernails (fig 2), and a few hours later, the patient appeared seriously ill and had palpable purpuric lesions on the distal part of both legs.



Figure 1 Radiograph showing multiple pulmonary nodules.



Figure 2 Splinter haemorrhages under the fingernails.

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Submitted 12 April 2000
Accepted 22 June 2000

Questions

- (1) What is the main differential diagnosis of this patient's pulmonary nodules?
- (2) How does the appearance of the skin signs affect the diagnosis?
- (3) How would you proceed with the diagnosis?

An unusual cause of trash feet

A M Khan, S Jacobs

Answers on p 794.

A 65 year old man presented to the accident and emergency (A&E) department with complaint of patchy mottling of both his feet. The following day this progressed to painful blisters and then to ulcers and gangrene (see fig 1).

Three weeks before this event, he had presented to the A&E department with bilateral painful thighs after routine and uneventful cardiac angiography. He was admitted on that occasion and treated with anticoagulant therapy. He was discharged home on the 10th day with warfarin.

His was known to have unstable angina, chronic obstructive airway disease, aortic aneurysm, and borderline hypercholesterolaemia, and he was a known ex-smoker. He was not known to be diabetic nor was his family history significant.

Physical examination revealed tender distal toes with intact distal pulses. Both his feet were

warm and well perfused other than the gangrenous patches. Other neurovascular examinations of the lower limb yielded no abnormalities.

Laboratory investigation on admission revealed an eosinophilic count of $1.03 \times 10^9/l$, erythrocyte sedimentation rate 34 mm/hour, urea 8.4 mmol/l, and creatinine 138 $\mu\text{mol/l}$. A dipstick test of his urine showed it to be positive for red blood cells. His creatine kinase was 1363 U/l. His dorsalis pedis resting pressure indices were 0.99 and 0.97 for the left and right foot respectively.

Questions

- (1) What is the clinical diagnosis? What could be the cause of the ulceration and gangrene in the feet?
- (2) How can we confirm the diagnosis?
- (3) What is the treatment and prognosis?



Figure 1 Patient's feet showing the progression from blisters to ulcers and gangrene (A-F) (reproduced with patient's permission).

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Submitted 29 August 2000
Accepted 17 October 2000

Breathlessness after percutaneous biliary drainage

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Answers on p 795.

A 77 year old woman presented with painless progressive jaundice and weight loss. She also had generalised pruritus and was passing dark urine and pale stools. On examination there was a palpable right upper quadrant abdominal mass. Plasma bilirubin level was 56 $\mu\text{mol/l}$ and alkaline phosphatase was 350 U/l. Serum amylase was within normal limits. Abdominal ultrasound scan showed a dilated biliary tree



Figure 1 Abdominal computed tomogram.



Figure 2 Thoracic radiograph.



Figure 3 Pleural aspirate.

and a distended gallbladder but there was no evidence of gallstones. Abdominal computed tomography was performed (fig 1). The patient underwent an endoscopic retrograde cholangiopancreatogram (ERCP) and insertion of a biliary stent, with resolution of the jaundice.

Ten days later the patient became jaundiced again and an abdominal ultrasound suggested stent occlusion with recurrent biliary tree dilatation. Percutaneous transhepatic cholangiography (PTC) with external biliary drainage was performed. The patient gradually became short of breath with right sided pleuritic pain and a thoracic radiograph was obtained (fig 2). Thoracocentesis was performed and the pleural aspirate is shown (fig 3).

Questions

- (1) What is the most likely cause of her jaundice and why was this suspected clinically?
- (2) What complication of PTC has occurred?
- (3) What is the treatment of this complication?

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Submitted 14 August 2000
Accepted 17 October 2000

Episodic weakness in a young woman

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Answers on p 795.

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Submitted 22 March 2000
Accepted 6 November 2000

A 37 year old woman presented with a 10 year history of episodes of weakness and palpitations. On one or two occasions each year she was admitted to her local hospital where her serum potassium concentration was always below the reference range with the lowest concentration reported at 2.2 mmol/l (3.5–5.0 mmol/l). The episodes were terminated with intravenous potassium. At other times she had mild weakness on exertion and occasional palpitations. These symptoms disappeared with oral potassium supplements. She was always normotensive. She had no diarrhoea or vomiting, her drug history consisted of oral amiloride

15 mg/day and a variable amount of effervescent potassium chloride supplements. She had a sister who had one minor episode of weakness but had not been investigated. She was referred to a renal unit for further investigations and to obtain a diagnosis.

On examination she looked well, with a blood pressure of 115/70 mm Hg, she was clinically euvoelaemic, she had no signs of self induced vomiting, and she was not cushingoid.

Her initial investigations revealed the following serum electrolyte concentrations: sodium 137 mmol/l (135–145), potassium 3.0 mmol/l (3.5–5.0), chloride 100 mmol/l (97–107),

bicarbonate 30 mmol/l (23–30), urea 4.9 mmol/l (2.5–8.0), creatinine 90 μ mol/l (40–130), and calcium 2.6 mmol/l (2.2–2.6). Arterial blood gases: arterial oxygen tension 12 kPa (12–15), arterial carbon dioxide tension 4.8 kPa (4.4–5.6), and hydrogen ion 32 nmol/l (35–45). Her 24 hour urine sample performed when her serum potassium was 3.0 mmol/l revealed a urinary potassium of 63 mmol/24 hours. Her plasma cortisol and thyroid function tests were normal. A supine plasma renin activity of 32.8 ng/ml/hour (0.5–2.4) and

aldosterone of 990 pmol/l (100–400) and ambulatory values of 40.4 ng/ml/hour (0.98–4.18) and 2500 pmol/l (400–800) were measured.

Questions

- (1) What is the metabolic abnormality?
- (2) What are the differential diagnoses?
- (3) What investigations will help distinguish between the possible diagnoses?
- (4) What is the likely diagnosis?

From cutaneous ulceration to chronic diarrhoea

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Answers on p 797.

A 48 year old woman presented with a 10 day history of painful red nodules over her face, trunk, and limbs which rapidly enlarged before developing central ulceration. She was otherwise well with no complaints. She had received four days of oral flucloxacillin but otherwise was on no medication. There was no past medical history of note. Examination revealed deep cutaneous ulcers with purplish undermined edges and slough at the ulcer bases (fig 1). Ulcer swabs and blood cultures failed to grow any organisms. Chest radiography, full blood count, and biochemical profile were normal, but the C reactive protein was markedly raised at 380 mg/l. Complement C3 and C4 levels and IgG, IgA, and IgM titres were normal. Antinuclear antibodies, DNA antibodies (single and double strand), and neutrophil cytoplasmic antibodies were negative. Neutrophil function tests were normal (as assessed by respiratory burst chemiluminescence). A skin biopsy showed a predominantly neutrophilic infiltrate of the dermis with a leucocytoclastic vasculitis. She was treated with 14 days of flucloxacillin and the ulcers healed over the next month.

Two years later she developed diarrhoea after a chicken meal. After one week she again developed cutaneous ulcers over her face, trunk, and limbs. She presented six weeks later with weight loss, abdominal pains, multiple



Figure 1 Typical cutaneous lesion (reproduced with permission).

skin ulcers, and passing faeces through her vagina. On examination the skin lesions were as before, there was tenderness in the left iliac fossa, and a rectovaginal fistula was present. Inflammatory markers were raised but ulcer, stool, and blood cultures were sterile.

Questions

- (1) What is the skin lesion shown in fig 1?
- (2) What is the gastrointestinal diagnosis?
- (3) What other skin lesions are associated with this disease?

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Submitted 10 January 2001
Accepted 22 February 2001

Multiple small opacities of metallic density in the lung

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Answers on p 798.

Questions

- (1) What is the likely diagnosis?
- (2) What is the differential diagnosis?

A 24 year old young man presented with sudden onset of chest pain, dry cough, and shortness of breath of five days' duration. He denied trauma, fever, chills, or haemoptysis. A physical examination was unremarkable. Chest auscultation revealed decreased breath sounds with pleural rub in the right infra-axillary region. His complete blood count, urine analysis, and liver and renal function tests were normal. Chest radiography showed bilateral, multiple point opacities with a metallic density confined primarily to lower and middle zones

as well as in the right ventricular apex (figs 1 and 2). High resolution computed tomography (HRCT) of the thorax revealed bilateral diffusely distributed multiple, tiny dense spherules, sometimes appearing as beaded chains filling pulmonary arterioles. Metallic density was also noted at the apex of the right ventricle with streak artefacts (figs 3 and 4). A radiograph of the abdomen showed numerous metallic deposits in the distribution of the paravertebral veins and kidney. Ultrasound showed multiple metallic deposits in the right kidney, however, they were absent in the left kidney; the liver, gall bladder, and spleen were normal. Pulmonary function tests showed restrictive ventilation defect, with reduction in vital capacity. The single breath carbon monoxide (CO) diffusing capacity was at the lower limit of normal (33.04 ml CO/min/mm Hg) and the ratio of diffusing capacity to single breath helium dilution total lung capacity was low (3.90 ml CO/min/mm Hg/l, predicted value 5.76).



Figure 1 Chest radiograph (posteroanterior view) showing multiple small opacities of metallic density. A large metallic opacity is also visible in right ventricle (arrow).

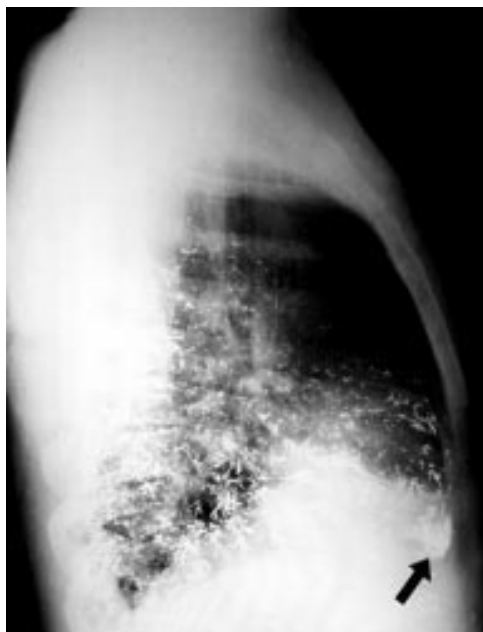


Figure 2 Chest radiograph (lateral view) showing multiple small opacities of metallic density. A large metallic opacity is also visible in right ventricle apex (arrow).

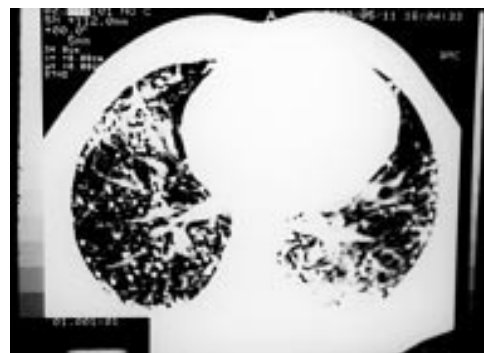


Figure 3 HRCT of chest showing spherules of metallic density.



Figure 4 HRCT of chest showing multiple opacities of metallic density conglomerating at some places. Opacities of similar density with multiple streak artifacts are also seen in the heart.

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Submitted 3 July 2000
Accepted 15 August 2000

Unusual cause of an abduction deficit in a 14 year old girl

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Answers on p 799.

A 14 year old girl presented with sudden onset of diplopia and slurred speech with no fever. A week previously, she had a headache, cough, and rhinorrhoea. However her symptoms resolved a few days before admission. Eye examination confirmed an abduction paresis of the left eye and abduction nystagmus of the right eye. Esotropia of the left eye was absent when the eyes were fixated in the primary position. There was full vertical eye movement, insufficient vestibulo-ocular reflex, and slow saccades of the affected eye. Neurological evaluation revealed slurred speech and ataxic gait.

Cerebrospinal fluid (CSF) analysis showed 40 white cells (all lymphocytes), protein of 0.4 g/l, and normal glucose content. Viral serology and bacterial cultures of blood, urine, CSF, stools, and throat swab were negative. Polymerase chain reaction analysis was negative for herpes simplex genome in the CSF. Serological tests for toxoplasmosis and *Mycoplasma pneumoniae* were negative. An autoimmune screen and a metabolic profile were normal. An electroencephalogram showed bilateral delta waves, more posteriorly, with no epileptiform discharges.

Magnetic resonance imaging (MRI) of the brain using T2 weighted axial images (fig 1) showed an increased signal in the cerebellar white matter and the dorsal aspect of the lower pons. There was an increased signal in the region of the abducens nucleus and the paramedian pontine reticular formation.

Treatment with oral erythromycin, intravenous acyclovir, and cefotaxime was started immediately and a degree of clinical improvement was seen within 48 hours. After three weeks of treatment, ocular movement, speech, and gait returned to normal and a repeat CSF analysis showed no abnormality. Subsequent MRI of the brain showed a marked improvement in the abnormal areas identified previously (fig 2).

Questions

- (1) What is the most likely diagnosis?
- (2) Give possible differential diagnoses of an abduction deficit.
- (3) How would you manage a patient with an abduction deficit?



Figure 1 T2 weighted axial image showing increased signal in the cerebellar white matter. There is increased signal at the dorsal aspect of the lower pons, and lateral to this on the left at the level of paramedian pontine reticular formation and abducens nucleus.



Figure 2 T2 weighted axial image showing a marked improvement in the previously identified abnormal areas of high signal.

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Submitted 21 March 2000
Accepted 10 January 2001

An unusual cause of acute bacterial meningitis

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Answers on p 800.

A 31 year old man was admitted after a 24 hour febrile illness with headache and confusion. On examination he was unwell, pyrexial at 39.4°C, tachycardic, and uncooperative. He became increasingly confused and agitated and exhibited signs of meningism. There was no rash or focal localising neurological signs, and visual fields to confrontation were normal.

He required intubation and sedation to control his agitation and was admitted to the intensive care unit. Lumbar puncture revealed turbid cerebrospinal fluid (CSF) containing $8300 \times 10^6/l$ white blood cells (95% polymorphs), protein 4.6 g/l, glucose 1.4 mmol/l

(blood glucose 5.9 mmol/l), with negative Gram stain and culture. Blood and throat cultures were sterile. Nasal swabs cultured methicillin resistant *Staphylococcus aureus*. Computed tomography of his head with contrast showed expansion of the sella and generalised cerebral oedema. Further imaging with magnetic resonance and coronal computed tomography of the pituitary fossa was obtained (fig 1). Thyroid function, random cortisol, follicle stimulating hormone, and luteinising hormone were normal but raised values of prolactin (15400 IU/l) and decreased testosterone concentrations (9.0 nmol/l) were found.

He received two weeks of intravenous ceftriaxone, vancomycin, and metronidazole and made an uncomplicated recovery. CSF rhinorrhoea was noted by the patient on bending forwards. Subsequent CSF cultures, obtained from the rhinorrhoea, was sterile.

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Submitted 8 September 2000

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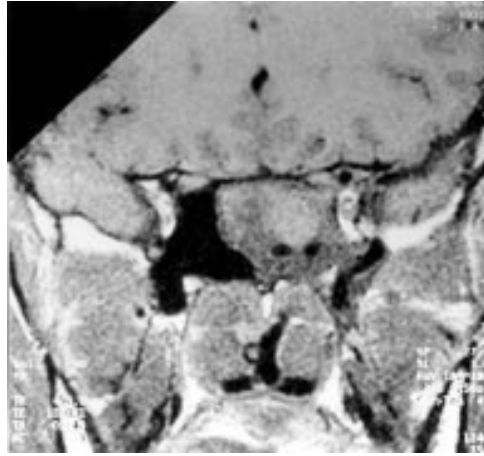


Figure 1 Coronal magnetic resonance image of the brain.

Questions

- (1) What is the lesion shown on the magnetic resonance imaging scan?
- (2) How does this usually present?
- (3) How may this condition present with meningism?
- (4) What is the treatment of the underlying lesion?
- (5) What are the causes of hyperprolactinaemia?