Hepatic abscess in a diabetic patient

J Valabhji, S Robinson, R S Elkeles

A 66 year old Trinidadian Asian woman was admitted with a two day history of fever, nausea, and vomiting. Type 2 diabetes had been diagnosed 17 years before and she had extensive microvascular and macrovascular complications. Glycaemic control had been consistently poor (HbA1 readings 13–16%), and she had needed insulin treatment over the preceding two years. She was hypertensive and had a typical diabetic dyslipidaemia. She had retinopathy for which she had received bilateral photocoagulation treatment. She had presumed diabetic nephropathy with proteinuria (previously quantified at 3 g/day) and impaired renal function (serum creatinine 140–160 µmol/l). She had a peripheral neuropathy. She had cerebrovascular disease, having previously suffered a transient ischaemic attack, and had been shown to have bilateral carotid stenoses. She had absent foot pulses, so she also had peripheral vascular disease.

On examination she was febrile but had no abdominal signs. Initial chest x ray was normal. White blood cell count was 12.6 x 10^9/l, alanine transaminase 172 IU/l, bilirubin 38 µmol/l, alkaline phosphatase 257 IU/l, and albumin 29 g/l. Clotting profile and platelet count were normal. Liver imaging showed a solitary lesion (fig 1) from which pus was aspirated, and also gallstones. Aspiration of the hepatic lesion was complicated by haemorrhage, requiring resuscitation and laparotomy to achieve haemostasis. The period of hypotension resulted in acute oliguric renal failure for which the patient required haemodialysis before spontaneous recovery of renal function. Culture of both liver aspirate and blood grew *Klebsiella pneumoniae*, and intravenous antibiotics were given according to sensitivities.

The patient developed progressive dyspnoea, persistent fever, and bilateral pleural effusions on chest x ray. Computed tomography of the chest showed numerous small areas of consolidation throughout both lung fields (fig 2). Despite continued treatment with appropriate antibiotics, she suffered several respiratory arrests, from which she was successfully resuscitated. Fluid balance was complicated by limited cardiac reserve (demonstrated by echocardiography) as well as by impaired renal function.

Answers on p 802.
With continued antibiotic treatment, she made a slow recovery. Despite clinical improvement, she developed an acute left sided pyramidal weakness. Computed tomography of the brain showed an area of low attenuation in the right parietal region (fig 3). Her neurological signs improved over the ensuing few weeks. Her respiratory function improved, and repeated imaging showed resolution of both hepatic and lung lesions. She subsequently left hospital, fully mobile and independent, and has remained well since discharge.

**Questions**

1. How does this woman’s diabetes relate to her current infective illness?
2. What was the probable source of her *Klebsiella pneumoniae* sepsicaemia?
3. What was the cause of her left pyramidal deficit?

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**Broad complex tachycardia: a diagnostic dilemma**

V Adhiyaman, S Froese, A Vaishnavi, R Cowell

A 56 year old woman was admitted to hospital with an hour long history of palpitations. She had suffered palpitations on three occasions over the preceding 12 months, each lasting for approximately half an hour. During these episodes she felt light headed but had not lost consciousness. Past medical history was unremarkable and she was taking no drugs.

On admission to hospital her symptoms had settled spontaneously and clinical examination was normal. A 12 lead ECG showed sinus rhythm and no other abnormality. Baseline blood tests, cardiac enzymes, and thyroid function tests were normal. She remained in sinus rhythm over the subsequent 24 hours and Holter monitoring was arranged as an outpatient. However, just before her discharge her symptoms returned and the heart rate was 160 beats/min and blood pressure 90/70. A 12 lead ECG was recorded which is shown in fig 1.

**Questions**

1. What is the diagnosis?
2. What is the most important differential diagnosis?
3. How would you treat this condition?
Bilateral fractures of the second metatarsals

S Shyamsundar, A L Pimpalnerkar

Case history
A 45 year old housewife presented with bilateral painful feet. The pain was located over the metatarsals and was increased by walking and decreased on rest. The initial x-ray was normal and the patient was sent home with advice to rest. A provisional diagnosis of metatarsalgia was made. The patient was reviewed after two months with persistent symptoms and a severe antalgic gait; x-rays (fig 1) of both feet revealed callus around both second metatarsals, indicating healing fractures.

Questions
(1) What type of fractures are these and what is their aetiology?
(2) Where are the other sites in the foot that stress fractures can occur?
(3) What would be the best management at this stage?

Figure 1  x Ray of feet showing healing metatarsal fractures.

Thyrotoxicosis of a rare aetiology

M Ismail, R V Bhat

A 28 year old woman was admitted with an eight month history of menstrual irregularities and a four month history of weight loss, haemoptysis, and dyspnoea. She had had menorrhagia for six months, for which a dilatation and curettage was done. Following this, she developed amenorrhoea. Her obstetric history was unremarkable. She was a mother of two (the last childbirth was two years previously). Physical examination revealed bilateral exopthalmos, lid lag, fine tremors in the outstretched fingers, warm and moist skin, and pallor. She had resting tachycardia and tachypnoea. Temperature and blood pressure were normal. There was no goitre. Fine crepitations were heard throughout inspiration and expiration all over the thorax. Haematological studies showed a haemoglobin concentration of 89 g/l, a leucocyte count of $10.9 \times 10^9/l$, and erythrocyte sedimentation rate of 70 mm/h. Thyroid function tests confirmed the diagnosis of thyrotoxicosis: thyroid stimulating hormone 0.01 mIU/l (reference range 0.35–5.5 mIU/l), T$_4$ 22.4 µg/dl (3.2–12.6), and T$_3$ 2.54 ng/ml (0.6–1.81). The serum biochemical profile was in the normal range. Chest x-ray showed multiple cannon ball opacities. An ultrasound study of the abdomen was normal. Further studies were performed to establish the aetiology.

Questions
(1) What is the diagnosis?
(2) What diagnostic procedure supports it?
(3) What is the cause of the thyrotoxicosis?
Iatrogenic recurrent severe hypercalcaemia and renal impairment

T Sulkin, A J Krentz

Answers on p 807.

Case 1
A 77 year old woman presented to her general practitioner with a three week history of vague and non-specific symptoms including anorexia, sore throat, and weakness of her legs. Her past medical history included hypertension and osteoarthritis, for which she was receiving bendrofluazide 2.5 mg daily together with ibuprofen (modified release) 1600 mg daily and co-proxamol (dextropropoxyphene and paracetamol) as required. Physical examination revealed an unwell patient with no specific abnormalities. Emergency laboratory investigations showed significant renal impairment with a plasma urea of 24 mmol/l (normal 3.0–6.5) and plasma creatinine concentration of 250 µmol/l (normal 60–125) together with hyperkalaemia (plasma potassium concentration 5.6 mmol/l (normal 3.5–5.0)). Plasma calcium concentration, corrected for the prevailing plasma albumin concentration,1 was 3.63 mmol/l (normal 2.15–2.55) with a marginally low plasma phosphate of 0.56 mmol/l (normal 0.7–1.5). Plasma alkaline phosphatase was normal. The patient’s erythrocyte sedimentation rate (ESR) was markedly raised at 100 mm/h, and a urinary tract infection was confirmed on microscopy and culture of her urine. Intravenous rehydration, infusion of a single dose of a bisphosphonate (pamidronate), and appropriate antibiotics led to a rapid clinical and biochemical improvement.

Potential causes of hypercalcaemia including multiple myeloma, thyrotoxicosis, and primary hyperparathyroidism were excluded by appropriate laboratory tests; serum parathyroid hormone (PTH) concentration was suppressed at 2.0 pmol/l (normal < 7.3) in the presence of hypercalcaemia. There were no features suggestive of malignant disease. Renal function and plasma calcium had returned to normal and ESR had fallen to 34 mm/h by the time of discharge from hospital. In the absence of a clear cause for the hypercalcaemia and acute renal impairment the patient’s thiazide diuretic was discontinued.1

Later the same year, following the prescription of a loop diuretic (frusemide (furosemide) 40 mg with amiloride 5 mg) for peripheral oedema, she was readmitted with symptoms of acute confusion, anorexia, and dyspepsia. She was clinically dehydrated with a corrected plasma calcium concentration of 3.24 mmol/l and plasma creatinine 166 µmol/l. Once again, rehydration with intravenous fluids resulted in rapid resolution of the abnormal biochemistry, and a calcium antagonist (amlodipine) was substituted for the diuretic.

The following year she was admitted a third time with similar symptomatology and biochemistry, (plasma calcium 3.57 mmol/l). Arterial blood gases confirmed a metabolic alkalosis (pH 7.48 (normal 7.38–7.44), standard bicarbonate 30.3 mmol/l (normal 21–28)). Inorganic phosphate and alkaline phosphatase were normal, as was her ESR; serum PTH was again suppressed at 1.1 pmol/l. On this occasion, direct questioning revealed a long history of symptoms suggestive of reflux oesophagitis for which she had been self medicating with a proprietary medicine. At endoscopy, a large hiatus hernia with oesophagitis was found. The ibuprofen was discontinued and a proton pump inhibitor (omeprazole) begun, with effective symptomatic relief. Following appropriate advice no recurrence of hypercalcaemia has been observed for almost two years.

Case 2
A 64 year old woman was admitted as an emergency with acute confusion. Physical examination revealed bilateral basal crackles. There was a neutrophilia, and a chest infection was diagnosed. Plasma creatinine concentration was 591 µmol/l with a plasma urea of 28.7 mmol/l. Plasma calcium concentration was 4.29 mmol/l with a low albumin of 26 g/l. The ESR was raised at 74 mm/h. Thyroid function tests were normal. No focus of infection was confirmed. Plasma electrophoresis was normal and urine was negative for Bence-Jones protein. Plasma parathyroid hormone was suppressed at 0.6 pmol/l. Computed tomography of the abdomen (performed because of transiently deranged liver function tests) showed no significant abnormality.

The patient was hydrated with intravenous saline; this led to an improvement in the hypercalcaemia to 3.7 mmol/l within two days. Intravenous pamidronate and continued hydration led to a further fall in plasma calcium concentration during the following days, and by discharge on day 12 renal function was normal. A history of chronic dyspepsia prompted upper gastrointestinal gastroscopy which revealed a large benign ulcer at 32–35 cm. Antral biopsy was positive for Helicobacter pylori. A proton pump inhibitor together with H pylori eradication treatment led to resolution of symptoms with no recurrence of hypercalcaemia (plasma calcium 2.48 mmol/l most recently).


Questions

(1) What was the cause of the recurrent hypercalcaemia and renal impairment in these patients?

(2) What is the appropriate management of this syndrome?

(3) What are the potential long term sequelae of the untreated syndrome?

(4) What, if any, was the contribution of the diuretics to the disturbance of calcium metabolism in the first patient?
Two cases of acute chest pain

J Tharakan, E Seth, A Radwan

Case 1
A 53 year old man presented with a four hour history of epigastric and lower chest pain radiating to the back and associated with vomiting, sweating, and breathlessness. He smoked six cigarettes a day. Examination showed him to be distressed. There was a significant discrepancy in the blood pressure between the two arms. The left carotid, brachial, and radial pulses were not palpable. The abdomen was soft and non-tender. An ECG was unremarkable, as was the chest x-ray. Computed tomography of the chest is shown (fig 1). Emergency surgery was carried out, from which he made a good recovery and was discharged home 10 days later.

Case 2
A 66 year old man was admitted with a three hour history of severe chest pain radiating to the back. He had had an abdominal aneurysm repaired two years ago. He smoked 10 cigarettes a day. Clinically he was found to be cold and clammy, with a high blood pressure in both arms. He was in sinus rhythm. There was a soft systolic murmur heard over the precordium, but the lungs were clear. ECG showed a left bundle branch block. Chest x-ray showed a large heart, with unfolding of the thoracic aorta. In view of these findings and bearing in mind his past history, computed tomography of the chest was undertaken (fig 2). He was transferred to intensive care for further management and was discharged home two weeks later.

Questions
(1) What is your diagnosis in both the cases?
(2) What investigations would you order to obtain a correct diagnosis?
(3) How are the two cases managed in clinical practice?
Hepatic abscess in a diabetic patient

Q1: How does this woman’s diabetes relate to her current infective illness?

Although it is recognised that diabetes can be associated with an increased susceptibility to infections, only a few bacterial infections have been proven to have a higher incidence in diabetic patients. These are: staphylococcal infections; Corynebacterium minutissimum causing erythrasma; Pseudomonas aeruginosa causing malignant external otitis; bacteroides and anaerobic streptococci causing non-clostridial gas gangrene; and Klebsiella pneumoniae causing hepatic abscess. A review of reported cases showed that diabetes was an underlying condition in 63% of hepatic abscesses caused by Klebsiella pneumoniae. There is marked geographical variation. In Taiwan, hepatic abscess caused by Klebsiella pneumoniae has been considered endemic for at least 15 years, presenting as an infectious complication in diabetic patients. Interestingly five cases, all involving diabetic patients, have been reported from Trinidad, which is our patient’s country of origin.1

Q2: What was the probable source of her Klebsiella pneumoniae septicaemia?

Klebsiella pneumoniae hepatic abscess is a rare condition associated with significant mortality. The infection often originates from the biliary tract or the gut. Klebsiella pneumoniae hepatic abscess has been associated with cholelithiasis and acute cholecystitis, carcinoma of the pancreas and the biliary tract, diverticular disease, and carcinoma of the colon. Treatment involves drainage and appropriate antibiotics, as well as screening for and eradicating any potential source of the bacteraemia.4

To identify a source of the Klebsiella pneumoniae septicaemia, an endoscopic retrograde cholangiopancreatogram was performed electrovally and showed only gallstones. A barium enema was normal. The source was therefore assumed to be cholelithiasis. However, in view of the patient’s intercurrent medical conditions, it was felt that the risks of elective cholecystectomy outweighed the risk of recurrent Klebsiella pneumoniae septicaemia.

Q3: What was the cause of her left pyramidal deficit?

Klebsiella pneumoniae hepatic abscess is associated with spread of infection to other organs, including the lung and brain.2 The development of respiratory symptoms and signs was associated with worsening signs of sepsis and so was assumed to reflect spread of infection to the lungs. However, the development of the pyramidal deficit occurred in the context of clinical improvement, and the appearances on computed tomography were more consistent with an area of thrombosis in the right middle cerebral artery territory than with a brain abscess. The pyramidal deficit was therefore assumed to be an incidental thromboembolic event.

Final diagnosis

Klebsiella pneumoniae hepatic abscess with spread of infection to the lungs.

Learning points

- Klebsiella pneumoniae hepatic abscess is one of the few bacterial infections proven to be more prevalent in diabetic patients
- Diabetic subjects with extensive microvascular and macrovascular complications have significant morbidity associated with septicaemia

References

Broad complex tachycardia: a diagnostic dilemma

Q1: What is the diagnosis?
The ECG shows a broad complex tachycardia, atrioventricular (AV) dissociation, fusion beats, and capture beats. P waves are seen throughout the tracings, but have no consistent relation to the QRS complexes and are occasionally followed by fusion beats. Capture beats are recognised by narrow QRS complexes followed by upright T waves. Fusion beats are slightly wider complexes followed by inverted T waves, which are of lesser magnitude compared with the deeply inverted T waves following ventricular complexes. In the rhythm strip on the lower trace, complexes suggesting capture beats and fusion beats are labelled C and F, respectively. These findings are consistent with ventricular tachycardia.

Q2: What is the most important differential diagnosis?
The differential diagnosis is supraventricular tachycardia with aberrant conduction. These may be difficult to distinguish on a surface ECG. However, the presence of fusion beats and capture beats provides support for the diagnosis of ventricular tachycardia. The presence of ischaemic heart disease, increased age, and very wide QRS complexes makes the diagnosis of ventricular tachycardia more likely.

Q3: How would you treat this condition?
Ventricular tachycardia or supraventricular tachycardia associated with haemodynamic decompensation should be treated with electrical cardioversion. If the patient is otherwise well ventricular tachycardia can be treated medically by various antiarrhythmic drugs, including intravenous lignocaine (lidocaine), amiodarone, disopyramide, and flecaïnome. Bretylium can be used in life threatening situations when other antiarrhythmic agents have failed. Overdrive pacing may be useful in patients with recurrent ventricular tachycardia not responding to drug treatment.

Discussion
It is important to differentiate between supraventricular and ventricular tachycardia, as the optimal treatment is different. The haemodynamic state should not be used as a guide for making the diagnosis, as this depends on the ventricular rate and left ventricular function; supraventricular tachycardia is often assumed to be the diagnosis in patients who are clinically well. At times it may be impossible to differentiate between these two based on the surface ECG, but the following features aid in the differential diagnosis:

- Wide and bizarre QRS complexes, AV dissociation, and the presence of fusion beats and capture beats support the diagnosis of ventricular tachycardia. Ventricular complexes with bizarre or prolonged configuration indicate only that conduction through the ventricle is abnormal, and such complexes can occur in supraventricular tachycardias because of pre-existing bundle branch, aberrant conduction, or conduction over accessory pathways. AV dissociation has long been considered a hallmark of ventricular tachycardia, but at times it is difficult to determine whether the P wave is conducted anterogradely (supraventricular tachycardia) or retrogradely (ventricular tachycardia). As a general rule, the presence of AV dissociation during a wide QRS tachycardia is strong presumptive evidence that it is of ventricular origin.

- Fusion beats indicate the activation of the ventricle from two foci, implying that one is of ventricular origin. When two impulses invade the ventricle simultaneously, each impulse activates the part of the ventricle, and the resulting QRS complex has a configuration that is between that of a QRS complex of the ectopic beat and the QRS complex of a sinus beat. Also the modification of the QRS complex by fusion will depend upon the relative contribution of the supraventricular and ventricular impulse to ventricular activation.

- A capture beat is the momentary activation of the ventricles by the sinus impulse during AV dissociation. During ventricular tachycardia, the slower sinus impulse cannot be conducted anterogradely to the ventricles, as a result of the lower AV nodal refractoriness reflecting partial retrograde penetration of the ventricular impulse. However, as the two pacemakers discharge asynchronously, the slower sinus discharge occurs progressively later in relation to ventricular discharge. The sinus impulse eventually reaches the AV node when it is no longer refractory, and is able to penetrate and capture the ventricles, resulting in a capture beat. A capture beat resembles the QRS complex of a normal sinus impulse and is preceded by a P wave (which is clearly shown in lead I). The presence of fusion beat and capture beats provides the maximum support for the diagnosis of ventricular tachycardia, although they are not very common.

- Vagal manoeuvres or intravenous adenosine may terminate or slow down supraventricular tachycardia. Adenosine has an important role as a diagnostic and therapeutic agent in the emergency management of broad complex tachycardia. The ability to block AV conduction allows diagnosis and treatment for most supraventricular tachycardias, and its short half life and absence of negative inotropic effects makes it safe even given during ventricular tachycardia. Verapamil should only be used if the diagnosis of supraventricular tachycardia is established as it may cause haemodynamic collapse in some patients with ventricular tachycardia.

After the immediate management, further treatment depends on the underlying diagnosis, and a specialist opinion may be needed to choose the correct option to prevent any further attacks.

Final diagnosis
Ventricular tachycardia.
Bilateral fractures of the second metatarsals

Q1: What type of fractures are these and what is their aetiology?
These are known as stress fractures. They appear as a result of increased load because of altered mechanics of the foot, occurring most often in athletes, but also in patients with diminished function of the first metatarsophalangeal joint, neuropathy, metabolic disorders, and hindfoot malalignment.\(^1\) Metatarsal fractures are particularly seen in military service in young adults and are also called “march fractures.” In that case the aetiology is thought to be repeated long term rhythmic loading.\(^2\)

Stress fractures are also mentioned in connection with some diseases or special clinical deformations such as rheumatoid arthritis, chronic bronchitis, deformities, and so on. Thus they do not appear only in a healthy bone as was originally thought.

Q2: Where are the other sites in the foot that stress fractures can occur?
Fractures occur most commonly in the distal second and third metatarsal shafts and in the navicular and calcaneum, but can occur in almost any bone of the foot.\(^3\)

Q3: What would be the best management at this stage?
The fracture is healing well, with callus formation. As the patient still has pain, all she needs is some protection from weight bearing until the pain resolves, after which normal walking is allowed. This patient responded to our conservative line of treatment.

Discussion
While stress fractures are common in military practice, they are much less so in civilian life. Bilateral ones are very unusual. The diagnosis depends on a careful history and physical examination, with selected use of imaging techniques. Any suspicious history associated with swelling and point tenderness warrants further investigation with a bone scan or magnetic resonance imaging. These are, however, not always readily available; but a repeat x ray after two to three weeks usually reveals some callus formation, even if a fracture cannot be visualised. In terms of management, non-operative treatment is usually successful, but surgery is recommended in athletes with fifth metatarsal stress fractures.\(^1\)

Final diagnosis
Bilateral stress fractures of the second metatarsal bones.

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Thyrotoxicosis of a rare aetiology

Q1: What is the diagnosis?
The most likely explanation for the association of haemoptysis and menstrual irregularities with multiple pulmonary nodules in a young woman with recent childbirth is choriocarcinoma.

Q2: What diagnostic procedure supports it?
Serum β human chorionic gonadotrophin (hCG) level is the most specific and sensitive marker for trophoblastic tumours. The value in our patient was 125 000 IU/l. Normal is less than 10 IU/l in the non-pregnant woman. Free β subunit is present in normal pregnancy and averages less than 4% of total hCG up to the time of hCG peak (peak level of total hCG, 100 000 IU/l after 60–80 days).

Q3: What is the cause of thyrotoxicosis?
Occasionally, thyrotoxicosis is present in patients with a very high concentration of hCG because of cross reaction between α subunits of hCG and thyroid stimulating hormone.

Discussion
Primary tumours of the breast, skeleton, and urogenital system account for approximately 80% of pulmonary metastases. Our patient with thyrotoxicosis and initial menorrhagia had a choriocarcinoma. The association of amenorrhoea with choriocarcinoma in this case is of interest and could have resulted from the thyrotoxicosis or from intrauterine adhesions following the curettage. Choriocarcinoma is most often preceded by a hydatidiform mole. Abortion or ectopic pregnancy are the next most common antecedents, followed by live births. The incidence following normal term delivery is 1 in 50 000, presentation usually being within the first year. Vaginal bleeding is the most common presentation. In approximately one third of the cases, symptoms arise not from the primary but from metastases. The long term survival in patients treated with chemotherapy ranges from 93% in high risk groups to 100% in low and medium risk groups.

Final diagnosis
Choriocarcinoma complicated by pulmonary secondaries and thyrotoxicosis.

Learning points
- Choriocarcinoma should be considered in a young woman with recent childbirth, menorrhagia, thyrotoxicosis, and pulmonary secondaries.
- Long term survival following chemotherapy ranges from 93% in high risk groups to 100% in low and medium risk groups.
- Most metastatic pulmonary nodules have their primaries in the breast, skeleton, or urogenital system.

Iatrogenic recurrent severe hypercalcaemia and renal impairment

Q1: What was the cause of the recurrent hypercalcaemia and renal impairment in these patients?
The clinical and biochemical features are typical of the nowadays rather inappropriately named, “milk-alkali” syndrome.

Q2: What is the appropriate management of this syndrome?
Treatment involves strict avoidance of the offending calcium and antacid containing preparations. Accurate diagnosis and, where possible, definitive treatment for the underlying condition for which the antacids are being taken are indicated.

Q3: What are the potential long term sequelae of the untreated syndrome?
Burnett's syndrome: this is a chronic state characterised by irreversible soft tissue calcification and renal impairment.

Q4: What, if any, was the contribution of the diuretics to the disturbance of calcium metabolism in the first patient?
Thiazide diuretics may exacerbate hypercalcaemia by reducing renal calcium excretion; loop diuretics, by contrast, have a calcicuric effect.

Discussion
In patients with otherwise unexplained hypercalcaemia, renal impairment in conjunction with suppressed plasma PTH levels should raise the possibility of self medication with antacid containing preparations containing calcium (particularly as the carbonate salt) and alkali. Patients with dyspeptic symptoms in whom the commonest causes of hypercalcaemia—primary hyperparathyroidism and malignancy—have been excluded, should be questioned about calcium containing antacid drugs. This inquiry immediately provided the diagnosis in our first patient. Direct questioning about dyspeptic symptoms prompted the production of a bottle of Bisodol tablets from the patient’s handbag. Each tablet contains sodium bicarbonate together with 522 mg of calcium carbonate. She had been consuming six tablets a day for chronic dyspepsia, giving a daily calcium intake in excess of 3 g. With strict avoidance of Bisodol there has been no recurrence of hypercalcaemia (latest plasma calcium 2.47 mmol/L). Similarly, close questioning of our second patient confirmed chronic ingestion of large quantities of a proprietary calcium carbonate containing preparation (Rennie).

The cardinal features of the milk-alkali syndrome include hypercalcaemia, a metabolic alkalosis, and renal impairment. This syndrome, originally described in patients treated with peptic ulcer regimens in the 1920s, is most often attributable to self medication with antacid preparations. Excessive milk ingestion is not a prerequisite for the development of the syndrome; the term milk-alkali syndrome has therefore become somewhat anachronistic. Despite therapeutic advances in the treatment of peptic ulcer disease the syndrome of calcium and alkali induced hypercalcaemia remains an important entity in the differential diagnosis of patients with hypercalcaemia. A chronic irreversible state characterised by soft tissue calcification and renal impairment (Burnett’s syndrome) is recognised. Prompt diagnosis and withdrawal of the offending agent should help prevent chronic renal damage while averting unnecessary investigations.

Concomitant illness or other drugs may aggravate the hypercalcaemia associated with this syndrome. In our first patient, dehydration associated with a urinary tract infection, and treatment with a thiazide diuretic may have contributed to her episodes of hypercalcaemia. Although thiazides reduce renal calcium excretion, their role in the pathogenesis of hypercalcaemia is disputed. Loop diuretics, in contrast, have calcicuric properties which are sometimes beneficial—in conjunction with measures such as rehydration and bisphosphonates—in the management of severe hypercalcaemia. However, caution is required as dehydration and pre-renal uraemia may be potentiated.

Although the term milk-alkali syndrome is now outdated, it continues to be encountered in modern medical literature. The original components of the syndrome have now been superseded and a more descriptive term such as “calcium-alkali” syndrome would seem more appropriate. The relatively high, and possibly increasing, incidence of calcium-alkali induced hypercalcaemia in hospital inpatients suggests that a higher level of awareness of this syndrome is required among clinicians. This view is reinforced by our experience with these patients who also illustrate the potential hazards of self medication.

Learning points
- Otherwise unexplained hypercalcaemia in conjunction with a suppressed plasma parathyroid hormone concentration and a metabolic alkalosis is suggestive of the “calcium-alkali” syndrome.
- Ingestion of excessive quantities of preparations containing antacid and calcium carbonate for dyspeptic symptoms may lead to recurrent hypercalcaemia with reversible, or sometimes irreversible, renal impairment.
- Patients in whom the commonest causes of hypercalcaemia (primary hyperparathyroidism and malignancy) have been excluded should be questioned about treatment with preparations containing antacid and calcium.
Two cases of acute chest pain

Q1: What is your diagnosis in both the cases?
Dissecting thoracic aortic aneurysm (type A) in case 1 and dissecting thoracic aortic aneurysm (type B) in case 2.

The clinical features which support the diagnosis are the acute crushing pain in the chest radiating to the back and the absent pulses in the upper limbs in case 1. The previous history of an abdominal aortic aneurysm and a large unfolded thoracic aorta on chest x ray without ECG changes of a myocardial infarction should arouse suspicion of a dissecting thoracic aneurysm in case 2.

Q2: What investigations would you order to obtain a correct diagnosis?
The diagnosis is made most accurately by aortography or computed tomography of the chest. Cross sectional and transoesophageal echocardiography support the diagnosis. In the cases presented here, figure 1 shows dissection flap in a type A aneurysm (arrow), with differential enhancement rates; figure 2 shows dissection flap in a type B aneurysm (arrow).

Q3: How are the two cases managed in clinical practice?
An early diagnosis is crucial. Intensive care units are the ideal place to manage these cases. While type A dissections (acute and chronic) benefit from operative intervention, conservative treatment in the form of controlling blood pressure is usually sufficient for type B dissections (acute and chronic). It is imperative that these patients are followed up on discharge and good control of blood pressure is maintained.

Discussion
Dissecting thoracic aneurysms can often be mistaken clinically for myocardial infarction, and treatment with thrombolytic agents such as streptokinase can have disastrous consequences and even lead to death. A high index of suspicion is necessary to avoid missing the diagnosis. Clinical symptoms to bear in mind are acute crushing pain in the anterior chest which often radiates to the back and lower abdomen, worsening with each heart beat. Discrepancy in blood pressure between the two arms, or absent pulses in the upper limbs, should also draw suspicion to the condition. Less common presentations include stroke, syncope, or paraplegia as described by Cohen.1

Dissecting aneurysms have now been classified as types 1 and 2 (also called type A), which involve an intimal tear in the ascending aorta within a few centimetres of the aortic valve, and type 3 (also called type B), which involves an intimal tear in the descending aorta distal to the left subclavian artery. The classification is outlined in box 1. The diagnosis is usually made by aortography or computed tomography of the chest, preferably with contrast. Cross sectional echocardiography and transoesophageal echocardiography (TOE) support the diagnosis. With newer advances in technology, magnetic resonance imaging, spiral computed tomography, and electron beam computed tomography (EBCT) will become the procedures of choice in the diagnosis of this condition.

A meta-analysis by Gysi et al of 40 years of experience with thoracic aortic dissections has shown that operative intervention is usually beneficial in both acute and chronic type A dissections. Furthermore, the analysis concluded that acute and chronic type B dissections usually benefited from conservative treatment, especially control of blood pressure.2 Surgical intervention may, however, be necessary for complicated cases. The overall prognosis remains poor if untreated, with 20% dying within 24 hours, 60% in two weeks, and 90% by 12 months. Follow up of these patients involves strict control of blood pressure, as discussed by Wheat.3

Final diagnosis
Case 1: type A dissecting thoracic aortic aneurysm; case 2: type B dissecting thoracic aortic aneurysm.

Box 1: Classification of dissecting thoracic aneurysms

- Type A: all dissections in volving the ascending aorta.
- Type B: all dissections not involving the ascending aorta.
- Type I: originates in the ascending aorta and propagates at least to aortic arch and often beyond.
- Type II: originates in and confined to ascending aorta.

Learning points

- Dissecting thoracic aortic aneurysms can be easily missed unless a high index of suspicion is maintained.
- Patients presenting with atypical chest or abdomen pain should be carefully examined for discrepancies in blood pressure in the upper limbs.
- Computed tomography of the chest or an aortogram are the best investigations to diagnose the condition.
- Management is best carried out in an intensive care unit.
- Surgical intervention is needed in type A dissections.

Two cases of acute chest pain

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