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* septicaemia?
- (3) What was the cause of her left pyramidal deficit?

Broad complex tachycardia: a diagnostic dilemma

V Adhiyaman, S Froese, A Vaishnavi, R Cowell

Answers on p 803.

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?

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Submitted 22 June 1999
Accepted 16 November 1999

Figure 1 ECG record during tachycardia.

Bilateral fractures of the second metatarsals

Answers on p 805.

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Submitted 5 August 1999
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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

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Answers on p 806.

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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 exophthalmos, 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 $\mu g/dl$ (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 bendrofluzide 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 μ mol/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 μ mol/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 μ mol/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).

¹ Martin TJ, Grill V. Endocrine investigation: hypercalcaemia. *Clin Endocrinol* 1995;42:535–8.

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?

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Submitted 21 October 1999
Accepted 7 December 1999

Two cases of acute chest pain

Answers on p 808.

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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?

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Submitted 9 June 1999
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Figure 1 Computed tomography of chest in case 1. For description, see answers.

Figure 2 Computed tomography of chest in case 2. For description, see answers.

SELF ASSESSMENT ANSWERS

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.¹ 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*.² 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.³

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 electively and showed only gallstones. A barium enema was normal. The source was therefore assumed to be cholelithiasis. However, in view

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

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.

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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.¹

Q3: How would you treat this condition?

Ventricular tachycardia or supraventricular tachycardia associated with haemodynamic decompensation should be treated with electrical cardioversion.² If the patient is otherwise well ventricular tachycardia can be treated medically by various antiarrhythmic drugs, including intravenous lignocaine (lidocaine), amiodarone, disopyramide, and flecainide. 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.³ 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.¹

- 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.⁴

- 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.⁵ 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 if given during ventricular tachycardia.⁶ 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.⁷

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.

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