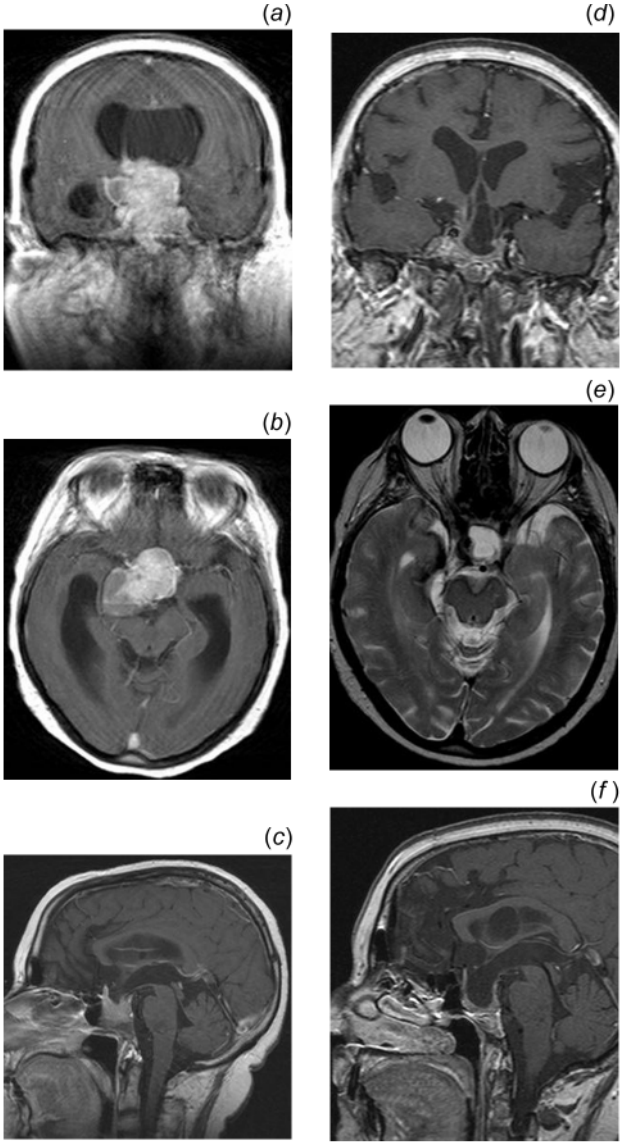

Figure 1 **MRI of head (*a*, *b*) on admission (with movement artefact); (*c*) immediately after cyst drainage; (*d–f*) after nine months' treatment with cabergoline**

the patient was taking half the correct thyroxine dose, and stopped taking thyroxine altogether for a short period, did the TSH rise, by which time the pituitary tumour had caused hydrocephalus severe enough to produce confusion, incontinence and falls. Although the dose of thyroxine required to maintain the euthyroid state has been reported to fall with age,[5] this would not have been a plausible explanation for the rapid decline in TSH seen in this patient. Had the free thyroxine also been measured, it might have prompted a search for other causes of a falling TSH during thyroxine replacement and let to diagnosis of the macroprolactinoma before the development of hydrocephalus.

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REFERENCES

1 Ladenson PW, Singer PA, Ain KB, *et al*. American Thyroid Association guidelines for detection of thyroid dysfunction. *Arch Intern Med* 2000;**160**:1573–5

2 Waise A, Belchetz PE. Lesson of the week: unsuspected central hypothyroidism. *BMJ* 2000;**321**:1275–7

3 Wardle CA, Fraser WD, Squire CR. Pitfalls in the use of thyrotropin concentration as a first-line thyroid-function test. *Lancet* 2001;**357**:1013–14

4 Shimon I, Cohen O, Lubetsky A, Olchovsky D. Thyrotropin suppression by thyroid hormone replacement is correlated with thyroxine level normalization in central hypothyroidism. *Thyroid* 2002;**12**:823–7

5 Sawin CT, Herman T, Molitch ME, London MH, Kramer SM. Aging and the thyroid. Decreased requirement for thyroid hormone in older hypothyroid patients. *Am J Med* 1983;**75**:206–9

Cold feet from adrenal leiomyosarcoma

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Among the rarer causes of cold feet is inferior vena cava thrombosis.

CASE HISTORY

A previously fit man of 57 was seen by the general physicians after acute onset of right groin pain with cold feet and altered sensation in both legs. On examination the feet were cold and cyanosed and the leg pulses were detectable only by doppler sonography. Examination was otherwise unremarkable. An ultrasound scan revealed bilateral femoral vein thrombosis and he was started on anticoagulants. A duplex scan then demonstrated thrombosis of the inferior vena cava (IVC) and both iliac veins. On abdominal CT he was found to have a massive left adrenal tumour involving the left kidney with tumour thrombus extending from the left renal vein into the IVC as far as the right atrium (Figure 1). A team including endocrine, vascular and cardiac surgeons was assembled. Additional investigations included a hormonal screen to exclude phaeochromocytoma, coronary angiography and

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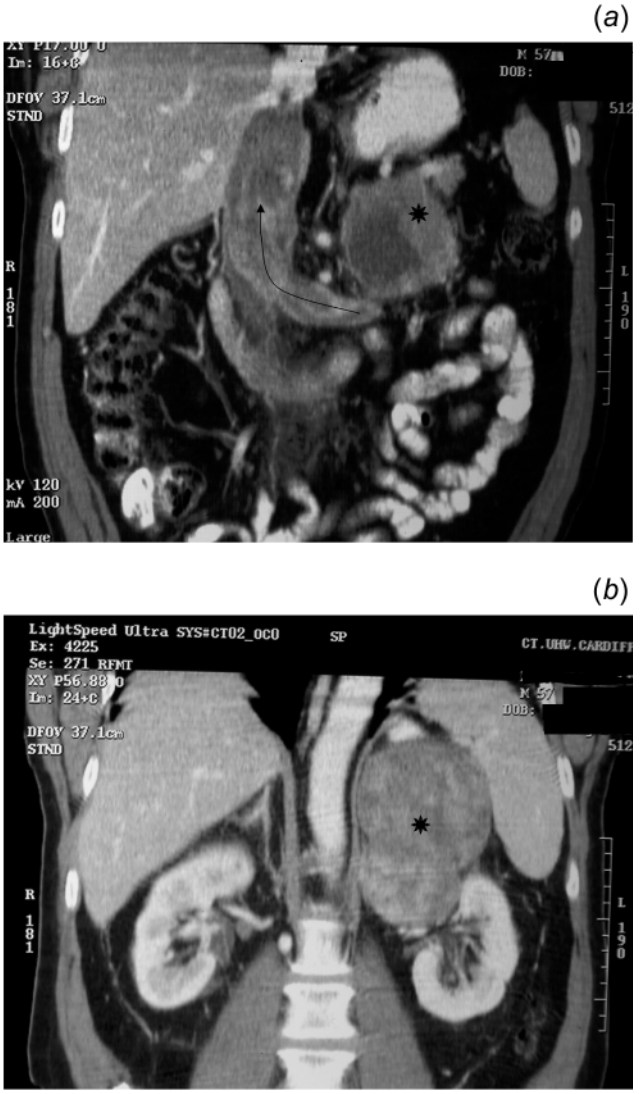

Figure 1 **Coronal reconstructions of CT scans.** (*a*) tumour thrombus is seen extending from left renal vein to inferior vena cava (arrow); ✱=left adrenal tumour. (*b*) ✱=left adrenal tumour

an echocardiogram to assess the patient's fitness for open-heart surgery when a patent foramen ovale was diagnosed incidentally. He was scheduled for elective surgery but required an emergency operation after a life-threatening pulmonary embolism. The patient was placed on cardiopulmonary bypass while the endocrine surgeon excised the adrenal tumour and left kidney en-bloc. The vascular surgeon then explored the IVC; tumour thrombus was extracted and embolectomy of the venous system was undertaken. This step was followed by exploration of the right atrium, pulmonary embolectomy and closure of the foramen ovale by the cardiac surgeon. On histological examination the tumour proved to be a high-grade leiomyosarcoma of the adrenal gland.

After a protracted stay in the intensive care unit, with complications including septicaemia from pneumonia, arrhythmia, seizures and infection with *Clostridium difficile*, the patient made an excellent recovery and underwent radiotherapy. He was able to work for more than six months before the development of widespread recurrence, from which he died.

COMMENT

Leiomyosarcoma of the adrenal gland is thought to derive from the smooth muscle wall of the central adrenal vein and its branches.[1] We have found nine previously reported cases, in three of which there was IVC tumour extension. The age of these patients was 30–63 years and tumour size was 11–25 cm; all were treated surgically, with extraction of tumour from the IVC.

Thrombosis of the IVC is seen in less than 2% of patients with deep vein thrombosis.[2] The usual cause is propagation from iliofemoral thrombosis, consequent upon tumour extension into the IVC from renal and adrenal tumours and consequent obstruction of venous return. Thrombosis of proximal deep veins can result in an oedematous blue leg (phlegmasia caerulea dolens) and venous gangrene. About 45% of patients with IVC involvement experience obstructive symptoms.[3] The tumour that most commonly invades the IVC is renal carcinoma, followed by adrenal cortical carcinoma and phaeochromocytoma.[3] The incidence of IVC invasion from adrenal cortical carcinoma has been estimated at around 9%.[3]

The treatment of choice for mesenchymal tumours remains radical resection. For tumour thrombus that remains below the diaphragm, the IVC may be cross-clamped above the hepatic veins. For tumour thrombus that extends above the diaphragm, cardiopulmonary bypass with deep hypothermic circulatory arrest is preferred.[4,5] Leiomyosarcoma is poorly responsive to adjuvant treatment. Little is known of the prognostic importance of intracaval tumour invasion from such a rare tumour: in renal cell carcinoma, provided local excision is complete, the level of caval tumour thrombus extension does not greatly influence long-term survival.[6] In the present patient the major operation gave a reasonable period of survival with satisfactory quality of life.

REFERENCES

1 Lujan MG, Hoang MP. Pleomorphic leiomyosarcoma of the adrenal gland. *Arch Pathol Lab Med* 2003;**127**:32–5

2 Gibbs NM. Venous thrombosis of the lower limbs with particular reference to bed-rest. *Br J Surg* 1957;**45**:209–36

3 Chesson JP, Theodorescu D. Adrenal tumor with caval extension—case report and review of the literature. *Scand J Urol Nephrol* 2002;**36**:71–3

4 Angermeier KW, Ross JH, Novick AC, Pontes JE, Cosgrove DM. Resection of nonrenal retroperitoneal tumors with large vena caval thrombi using cardiopulmonary bypass and hypothermic circulatory arrest. *J Urol* 1990;**144**:735–9

5 Marshall FF, Dietrick DD, Baumgartner WA, Reitz BA. Surgical management of renal cell carcinoma with intracaval neoplastic extension above the hepatic veins. *J Urol* 1988;**139**:1166–72

6 Moinzadeh A, Libertino JA. Prognostic significance of tumor thrombus level in patients with renal cell carcinoma and venous tumor thrombus extension. Is all T3b the same? *J Urol* 2004;**171**:598–601

Streptococcus mutans endocarditis: beware of the 'diphtheroid'

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Streptococcus mutans, a major cause of dental caries and endocarditis, is easily dismissed by the inexperienced as a common non-pathogenic skin bacterial contaminant termed 'diphtheroid'.

CASE HISTORY

A man of 62 was admitted after three months of lethargy, malaise, night sweats and weight loss. Type 2 diabetes mellitus had been diagnosed at about the time these symptoms began and, on a home visit, his general practitioner had thought they were due to poor diabetic control. The patient had not experienced chest pain, cough, rigors or arthralgia. His medical history included a Ross procedure (pulmonary valve autograft to aortic position, pulmonary valve homograft) at age 35 for a congenital abnormality of the aortic valve. His medication was gliclazide and aspirin.

On examination he was pyrexial (38.2°C), and tachycardic. The principal abnormal physical sign was a mid-systolic murmur. There was no rash or splenomegaly. Haemoglobin was 11.6 g/L, white cell count 21.6×10^9/L (neutrophils 95%), C-reactive protein 72 mg/L, erythrocyte sedimentation rate 94 mm/L, random blood glucose 13.1 mmol/L, HbA1c 9.6%. The initial diagnosis was hyperosmolar non-ketotic hyperglycaemia. Because of his persistent pyrexia, a set of peripheral blood cultures was taken which became positive within 24 h showing Gram-positive rods. Since a biochemical test for the identification of Gram-positive rods gave no profile, the isolate was reported as 'diphtheroids' and presumed to be a contaminant. However, because of his previous valve replacement and his ongoing fevers the clinicians were advised to take more blood cultures. Three further sets of blood cultures became positive within two days. Gram-positive rods were again seen; and, on review of the original Gram films, the organisms were deemed more likely to be streptococci, possibly *S. mutans*. It was strongly suggested to exclude endocarditis in this patient and to review the state of his teeth. A transthoracic echocardiogram showed a small mobile vegetation on a thickened pulmonary valve. He was then treated for presumed streptococcal endocarditis with gentamicin 80 mg twice daily for two weeks and vancomycin twice daily for five weeks (he was allergic to penicillin). Eight teeth with advanced caries were removed while he was in hospital. His infection markers became normal.

Originally, the laboratory performed a biochemical test (API) for the identification of streptococci, which named the organisms as *Leuconostoc* sp. This was unlikely to be correct since the isolate was vancomycin sensitive whereas *Leuconostoc* spp. are resistant. After two weeks, the reference laboratory confirmed the isolate to be a fully sensitive *S. mutans*.

COMMENT

S. mutans belongs to a group of non-haemolytic Gram-positive cocci which typically appear rod-shaped on acid culture medium but show a streptococcal appearance, in chains, when subcultured into a neutral or alkaline broth. The organism was first described by Clarke in 1924, who noted the variation of morphology with the pH of the medium.[1] On the basis of their rod-shaped appearance when isolated from blood culture medium they can be easily dismissed as diphtheroids—i.e. non-pathogenic skin bacteria. In 1977 Emmerson and Eykyn[2] drew attention to this hazard, by reporting two cases of *S. mutans* endocarditis in which the organisms had been initially misidentified as diphtheroid contaminants.

Although diphtheroids are the second most common bacterial contaminants in blood cultures, the case presented here illustrates that 27 years on we still fall into the same trap of misidentifying an important pathogen and potentially delaying the diagnosis and treatment of a serious infection.[3] Clinicians and microbiology staff need to be critical about the identification of 'diphtheroids' from blood cultures. New technology such as commercial identification kits may even add to the confusion.

REFERENCES

1 Clarke JK. On the bacterial factor in the aetiology of dental caries. *Br J Exper Pathol* 1924;**5**:141–7

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