Ascites and a raised serum Ca 125—confusing combination

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Measurement of serum Ca 125 in the initial evaluation of a woman with ascites can lead to unnecessary investigations or even surgery.

CASE HISTORY

A woman aged 56 was admitted with swelling of the lower limbs and the abdomen which had developed slowly. Previously she had been well; she had never smoked and drank little alcohol. Cytological and microbiological examination of ascitic fluid revealed nothing abnormal. An echocardiogram, however, showed dilated and poorly contracting cardiac chambers with moderate mitral and tricuspid regurgitation. Idiopathic dilated cardiomyopathy was diagnosed and the patient was started on diuretics and an angiotensin converting enzyme inhibitor.

In view of the gross ascites, serum Ca 125 was checked and proved to be greatly raised at 2150 IU/mL (normal <38). Although no malignant cells had been found in the ascitic fluid, a gynaecological opinion was sought. On vaginal examination, there were no adnexal masses palpable. A CT scan of the pelvis was normal. Transabdominal ultrasound revealed a normal right ovary but the left ovary was not clearly visualized. Because the cause of the raised serum Ca 125 remained unclear, exploratory laparotomy was decided upon. However, in the interim the patient responded to heart failure treatment with almost complete resolution of ascites and peripheral oedema. Serum Ca 125 fell to 240 IU/mL. Importantly, a transvaginal ultrasound showed normal ovaries. The laparotomy was therefore cancelled and the patient continued on heart failure treatment. When reviewed 2 months later, she was well and serum Ca 125 had returned to normal (31 IU/mL).

4 years later she returned with gross ascites and peripheral oedema. This time serum Ca 125 was 4020 IU/mL, but cytological and microbiological examination of ascitic fluid was again normal, as was a transvaginal ultrasound. She responded well to intravenous diuretics and fluid restriction. Maintenance doses of frusemide and enalapril were increased. 3 months later she was clinically euvolaemic and serum Ca 125 was normal.

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COMMENT

In a postmenopausal female with ascites, raised serum Ca 125 is frequently perceived as a diagnostic marker of ovarian cancer. This is a misconception. Although the Ca 125 antigen is present on more than 80% of malignant epithelial ovarian tissue of non-mucinous type, it is also found on both healthy and malignant cells of mesothelial (pleural, pericardial, peritoneal, endometrial) and non-mesothelial (amniotic membrane, tracheobronchial and cervical epithelium) origin. Raised serum Ca 125 levels have therefore been reported in various conditions involving these cells, including pleural and pericardial effusions and ascites[1].

The mechanism of raised serum Ca 125 in ascites is not fully understood. In culture, peritoneal mesothelial cells shed five times more Ca 125 than ovarian cancer cells[2], and one theory is that the antigen enters the blood via lymphatic absorption of ascites[3]. Peritoneal stretching seems relevant, since serum Ca 125 falls rapidly after paracentesis[4]. In patients with cirrhosis an additional factor could be low clearance of Ca 125 by the liver[5]; and in those with malignant ascites, infiltration of the peritoneal membrane could contribute.

Serum Ca 125 is therefore a non-specific marker. Currently it is used for monitoring response to treatment and detection of recurrent disease in patients with known ovarian cancer[6], and as an aid to differential diagnosis of adnexal masses[7]. Its value in screening for ovarian cancer is being investigated[8].

Our case illustrates that, for good management decisions, a rational order of investigations is crucial. In this patient, during investigation of ascites, serum Ca 125 was measured before pelvic ultrasound examination. This happened on two occasions and, unfortunately, each time this led to diagnostic confusion and concerns about ovarian cancer. On the first occasion surgery was scheduled, and this would have been particularly hazardous in view of the true cause of the ascites.

The exclusion of metastatic ovarian cancer as a cause of ascites is of great importance in view of the benefits of chemotherapy in advanced disease[9]. We suggest that every woman with first-onset ascites should have a pelvic ultrasound scan (preferably transvaginal) unless lymphoma cells are found in the ascitic fluid. Although not specifically evaluated in patients with ascites, a normal pelvic ultrasound scan in a symptom-free postmenopausal woman

with raised serum Ca 125 indicates an extremely low risk of ovarian cancer[10]. Pelvic ultrasound should also be considered in patients with an unexplained worsening of ascites the cause of which had been established previously. If an adnexal mass is identified, serum Ca 125 may be a useful diagnostic aid; in the absence of a mass, misinterpretation of a raised serum Ca 125 can lead to unnecessary and hazardous interventions.

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A missing vas

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The vas deferens is seldom palpated during routine physical examination, though absence of the vas has important implications.

CASE HISTORY

A man of 45 with moderate obesity came for routine vasectomy under local anaesthesia after an initial outpatient consultation at which no physical examination had been performed. The right vas was successfully ligated; the surgeon then explored the other side but had to abandon the operation because of persistent patient discomfort and inability to find the vas. At a second operation, performed under general anaesthesia, absence of the left vas was confirmed. An ultrasound scan subsequently demonstrated absence of the ipsilateral kidney.

COMMENT

John Hunter first described absence of the vas deferens in a cadaver in 1737[1], and an association with ipsilateral renal agenesis has been noted[2,3]. The reported prevalence of unilateral absence of the vas deferens is between 0.06% and 0.8%[4,5]. Unilateral renal agenesis is less common, with an estimated prevalence of 0.1%[6]. However, of patients with unilateral absence of the vas deferens, up to 80% have ipsilateral renal agenesis[4]. Even when the kidney is present, it may be affected by a wide range of anomalies such as ectopia, malrotation, fusion and polycystic disease. Abnormalities in the solitary contralateral kidney occur in nearly 10% of such patients, ranging from ureteric obstruction to vesicoureteric reflux[4]. In addition, ipsilateral absence of the adrenal gland has been reported in 15–25% of patients with renal agenesis[7].

Developmentally the Wolffian duct, which ultimately forms the vas deferens, is derived from the mesonephric duct. The ureteric bud which likewise develops from the mesonephric duct forms the ureter, renal pelvis, calyces, and collecting tubules. Thus congenital absence of the Wolffian duct precludes formation of a ureteric bud and results in renal agenesis. As in the case presented, the testicle is usually present in patients with a congenitally absent vas, since the testis is derived from the genital fold and not the mesonephric duct.

This case highlights the difficulties which may result from a congenitally absent vas deferens during vasectomy, which is increasingly performed as a day-case ambulant procedure in family planning clinics. We recommend that, when the vas cannot be felt on one side, on clinical examination, the patient should undergo scrotal exploration under general anaesthesia, to allow a thorough search before the vas is declared congenitally absent. All such patients should have imaging of the renal tracts. Because of the association between congenital absence of the vas deferens and defects in the cystic fibrosis transmembrane conductance regulator gene, these patients should also undergo genetic cystic fibrosis screening[8].

Absence of the vas deferens may be a useful warning sign in patients with blunt abdominal trauma requiring emergency surgery. Such patients should be assumed to have ipsilateral renal agenesis until proved otherwise.

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Suprachoroidal haemorrhage after addition of clarithromycin to warfarin

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In the presence of infection (and its treatment), anticoagulant therapy can go dangerously out of control.

CASE HISTORY

A woman aged 62 attended casualty because the vision in her only eye had suddenly deteriorated after a paroxysm of coughing. She gave a history of aortic and mitral valve replacements for rheumatic heart disease, and was taking warfarin with the aim of keeping the international normalized ratio (INR) between 2.5 and 3.5. A week before presentation she had begun a course of clarithromycin 250 mg twice daily for a chest infection. She had a complex ophthalmic history with bilateral cataract extraction in childhood and bilateral retinal detachment surgery at age 21. The left detachment repair had been successful with restoration of acuity to 6/18, but the right eye had become phthisical (blind and shrunken).

On examination she was pyrexial with signs of a resolving chest infection. There was a left hyphaema in the anterior chamber. The fundus could not be examined because of vitreous opacity. Ocular ultrasonography revealed a dense vitreous and suprachoroidal haemorrhage (Figure 1). Her INR was 8.2. Subsequent examination of the general practitioner's records showed an INR of 2.3 three days before clarithromycin and 2.9 three days into the course. The highest INR in the past four months had been 3.7.

The clarithromycin was stopped, the warfarin dose was adjusted and she was given 0.5 mg intravenous vitamin K. The chest infection cleared three days after admission. A vitrectomy with anterior chamber washout and partial drainage of the suprachoroidal haemorrhage was performed when her INR had fallen to 2.9. Unfortunately this did not improve her vision, which had now deteriorated to perception of light only.

COMMENT

A possible reason for the loss of anticoagulant control in this patient was the infection itself. However, we suspect that the cause was an interaction between clarithromycin and warfarin. This has been recorded in four previous case reports[1–3]. One described the interaction between clarithromycin, warfarin and digoxin in a woman with chronic atrial fibrillation[1]. Her INR at presentation was 7.3. She had gastrointestinal symptoms, weakness, dizziness and visual changes, but no permanent systemic or visual complications ensued. In the other three cases the INR rose to between 5.6 and >20 after taking clarithromycin and warfarin but no systemic symptoms developed[2,3]. Any of these patients could have suffered an intracerebral or other life-threatening bleed with an elevated INR.

The most common ocular haemorrhagic complication of warfarin is subconjunctival haemorrhage; a rarer one is spontaneous hyphaema. These almost always resolve without sequelae[4]. Suprachoroidal haemorrhage usually arises from

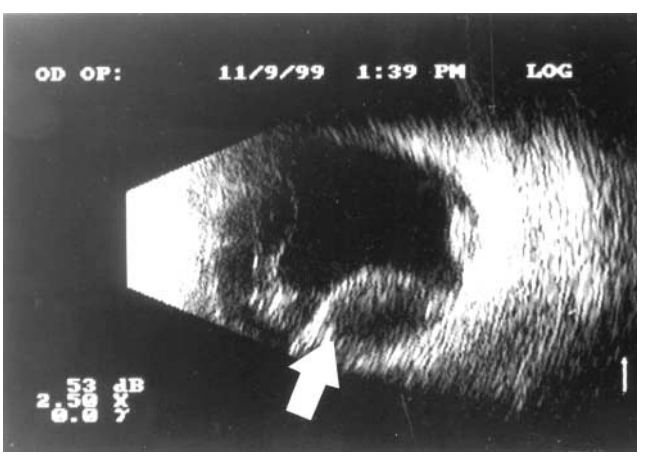

Figure 1 **Ocular ultrasound scan showing suprachoroidal haemorrhage**

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intraocular surgery or trauma[5]; we are not aware of any previous cases resulting from drug interaction with warfarin.

Antibiotic therapy in the preceeding four weeks is one of the major risk factors for a rise in INR to above 7 (at which point the risk of bleeding increases substantially[6,7]), in patients taking warfarin. Clarithromycin is thought to interact with warfarin through inhibition of the cytochrome p450 drug metabolizing system[7]. Cephalosporins inhibit vitamin K metabolism. Sulphonamides eliminate bacterial flora with consequent vitamin K deficiency. Ciprofloxacin and tetracyclines potentiate warfarin through unknown mechanisms[8].

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Persistent superior vena caval syndrome due to totally implantable venous access systems

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Totally implanted venous access systems are widely used in cystic fibrosis and have a low incidence of complications. Whilst local venous thromboses are described with these devices, we have found no previous reports of permanent superior vena caval occlusion.

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CASE HISTORY

A woman with cystic fibrosis (CF) had been colonized with *Pseudomonas aeruginosa* for many years. She required frequent intravenous antibiotic therapy and because of poor peripheral venous access a Port-a-Cath was inserted into the left subclavian vein in March 1994, when she was 19. When not in use, the port was flushed routinely every four weeks by a trained CF nurse using 5 mL heparin solution (1000 U/mL). During use, the device was flushed before and after each access with 5 mL heparinized saline (10 U/mL). In hospital this was done only by designated CF nurses; at home it was done by the patient, after full training.

In September 1995 the patient reported swelling of her arms and face and dilation of the veins on her chest. Superior vena-caval obstruction was diagnosed. Her coagulation profile was normal and blood cultures were negative. Angiography revealed thrombus in the superior vena cava (SVC). Streptokinase (25 000 U) was administered via the Port-a-Cath, and she was anticoagulated with heparin and then warfarin to maintain an international normalized ratio of 2–3 times normal. The symptoms of SVC obstruction resolved and the Port-a-Cath remained patent.

In March 1996 the Port-a-Cath became obstructed once again despite adequate anticoagulation and negative blood cultures, and patency could not be restored with urokinase (25 000 U) instilled into the Port-a-Cath line. The device was therefore removed and a new Port-a-Cath was inserted into the right subclavian vein at the same procedure. Thereafter, anticoagulation with warfarin was maintained. However, in October 1996 the replacement Port-a-Cath became occluded, with symptoms of recurrent SVC obstruction. Magnetic resonance scans revealed that the SVC was blocked in its middle third by organizing thrombus (Figure 1). Despite repeated streptokinase administration into the Port-a-Cath and adequate anticoagulation with warfarin, the catheter remained unuseable and was removed. In 1998 warfarin therapy was stopped and the patient has been maintained on aspirin ever since. The development of collateral venous drainage temporarily improved peripheral venous access. However, over the subsequent four years it has been increasingly difficult to achieve peripheral venous access, and a repeat MRI scan in December 2000 confirmed the persistence of SVC obstruction preventing the use of upper-body central venous cannulation.

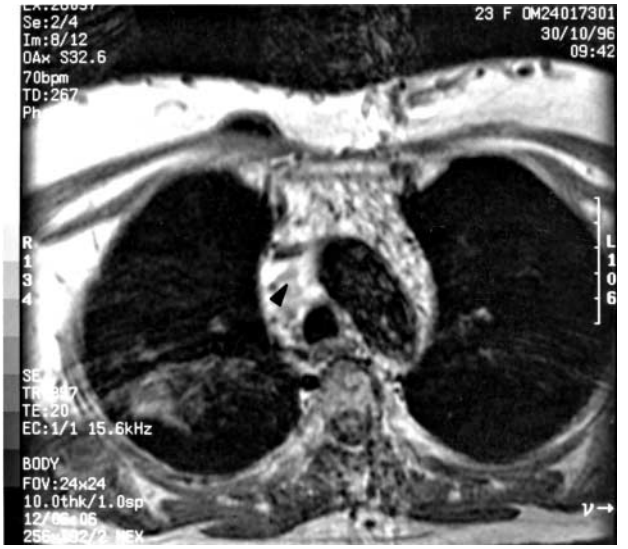

Figure 1 **Coronal section, T1 weighted, magnetic resonance image showing organized thrombus (arrow) occluding SVC**

COMMENT

Long-term venous access was first described in 1968, when total parenteral nutrition was administered via percutaneous cannulation of the subclavian vein with an externally exiting catheter. By 1973, less thrombogenic silicone rubber catheters were in use. These were further modified by increasing the bore size, and in 1979 Hickman[1] developed a novel subcutaneous placement, whereby subcutaneous tunnelling and the associated fibrous adhesions to the Dacron cuff helped reduce infection rates.

Subsequently, central venous access devices have been designed to be totally implanted beneath the skin, with access accomplished by a percutaneous Huber point needle which pierces the silicone septum without coring it out. These totally implanted venous access systems (TIVAS) are favoured by patients because they impose no restrictions on activity or clothing. Once inserted, TIVAS are designed to remain *in situ* for several years, with a mean survival time exceeding thirty months[2]. They also have a lower incidence of complications than their external ring counterparts.

Local sepsis has been reported in up to one-third of cases[3,4]; systemic sepsis is uncommon[4] but may require device removal[3]. Mechanical complications occur in up to half[2,4] and include migration, perforation and fragmentation of the septum. In one patient the catheter was severed at the point of entry into the subclavian vein, with migration of the distal 10 cm into the right ventricle[5].

Thrombotic events range from obstruction of the system with intraluminal thrombus, reported in 8–40% of cases, to subclavian vein thrombosis in 5%[2,3]. Thrombolytic agents restore patency in up to two-thirds of these cases[2]. One CF patient with protein S deficiency developed recurrent deep venous thrombosis when a TIVAS was placed in the left long saphenous vein[6]. Whilst SVC thrombosis is well described with Hickman catheters, it is rare with TIVAS and only three cases of transient SVC obstruction have been reported. In two of these the obstruction was not confirmed by imaging techniques; all three patients responded well to thrombolytic therapy and anticoagulation.

In our patient, we have demonstrated a new complication of TIVAS insertion—persistent SVC obstruction. This occurred despite adequate care of the port by both patient and CF staff, and persisted despite subsequent full anticoagulation. Whilst the SVC obstruction may have temporarily and paradoxically improved venous access by the development of collateral veins, it is a worrying complication in a CF patient who now once more has very poor peripheral venous access and who at some stage may need heart–lung transplantation. This patient requires frequent courses of intravenous antibiotics to treat her CF chest disease, and venous access has now become extremely difficult. In view of her persistent SVC obstruction, upper-body central vein cannulation is not a viable option. It has been suggested that permanent central venous access may be possible by inserting TIVAS into the inferior epigastric vein[7], but we are reluctant to consider this in view of her recurrent thrombotic events and the potential for infection associated with lower abdominal implantation.

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Pan-hypopituitarism and diabetes insipidus after a heart transplant

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The increased incidence of malignancy, particularly lymphoproliferative disorder, in organ transplant recipients[1] can present diagnostic and therapeutic challenges.

CASE HISTORY

A man aged 33 underwent cardiac transplantation for end-stage ischaemic cardiomyopathy, after which he was maintained on combined immunosuppression therapy with cyclosporin, azathioprine and prednisolone. 5 years after the transplant he was admitted with severe right temporal headache and blurring of vision. On examination his vision was diminished to perception of light bilaterally but the fundi and remaining neurological examination were normal. A computerized tomographic scan (CT) of the head and examination of cerebrospinal fluid (CSF) revealed no abnormalities. The headache and the visual disturbance resolved within 24 hours and he was discharged with a possible diagnosis of migraine. 3 months later he was readmitted: for 2 weeks he had had pain around the left eye and for 3 days he had noted drooping of the left eyelid, with diplopia. A history of polyuria and polydipsia was also noted. Examination revealed a left third-nerve palsy. Cerebral angiography showed nothing abnormal but magnetic resonance imaging (MRI) revealed a suprasella lesion, involving the pituitary stalk, which enhanced with gadolinium (Figure 1). Biopsy was deferred because of the position of the lesion. The third-nerve palsy gradually resolved. However, he returned shortly afterwards having suffered 24 hours of confusion, nausea, vomiting and photophobia. Polyuria and polydipsia were still troublesome, and additional complaints were anorexia, weight loss, malaise, loss of libido and decreased secondary sexual hair. There were no localizing neurological signs.

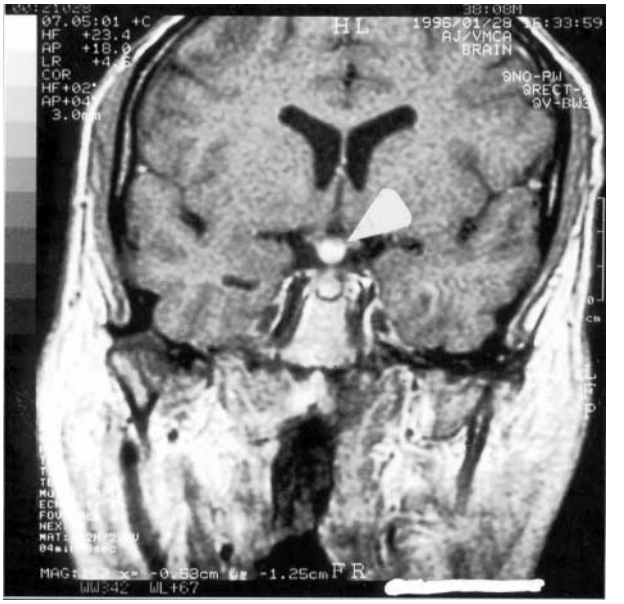

Figure 1 **Pituitary magnetic resonance scan at presentation showing high signal lesion (arrow) in pituitary stalk**

Routine haematology and biochemistry results were normal, apart from long-standing mild renal impairment (urea 11.0 mmol/L, creatinine 164 μmol/L). In particular, the serum sodium was 139 mmol/L (normal 134–147). Erythrocyte sedimentation rate (80 mm/h) and C-reactive protein (38 mg/L) were raised. Repeated CSF studies showed no conclusive evidence of infection or malignant disease. CSF protein was mildly raised at 0.55 g/L, glucose was normal and there were 5 lymphocytes/μL. CSF cytology showed lymphocytes mostly of mature type, but some cells had slight nuclear enlargement, possibly representing less mature forms, and the overall picture was thought to be of a mild lymphocytic reaction. Serum and CSF examinations for tumour markers (α-fetoprotein, human chorionic gonadotropin and carcinoembryonic antigen) and angiotensin converting enzyme were normal. CSF cultures for acid-fast bacteria and fungi, and tests for cryptococcus (cryptococcal antigen and Indian ink test) were negative. Autoantibody screening and serum protein electrophoresis gave normal results. Chest X-ray revealed cardiomegaly but no evidence of lymphadenopathy. Nothing abnormal was seen on abdominal ultrasonography.

Basal and dynamic endocrine tests confirmed hypopituitarism with biochemical evidence of thyrotroph and gonadotroph failure. The patient had a low free thyroxine (FT_4) of 10 pmol/L (normal 11–26) and a low thyrotropin (TSH) of less than 0.1 mU/L (0.2–4.0). The TSH did not rise in response to administration of thyrotropin-releasing hormone (TRH). His testosterone level was less than 0.7 nmol/L (9–25), luteinizing hormone (LH) less than

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0.5 IU/L (0.5–6.0), and follicle-stimulating hormone (FSH) less than 0.5 IU/L (0.8–9.0). LH and FSH did not respond to administration of gonadotropin-releasing hormone (GnRH). Corticotroph function was not assessable because of long-term treatment with prednisolone. Mild hyperprolactinaemia of 1104 mU/L (normal up to 450) was consistent with a stalk lesion. Basal growth hormone (GH) was 1.6 mU/L; in view of the previous cardiac transplantation, the insulin-stress test for dynamic assessment of the GH axis was not performed. His 24-hour urinary volume was 6.8 L. A hypertonic saline test was performed by infusing 5% saline at a rate of 0.06 mL/kg per minute for 2 hours and measuring plasma osmolality, urine osmolality and plasma arginine vasopressin (AVP) every 30 minutes. During the test, plasma osmolality rose from 296 to 317 mosm/kg, but urine osmolality remained less than 140 mosm/kg. Basal plasma AVP was 0.4 pmol/L and did not rise above 0.5 pmol/L, confirming the diagnosis of central diabetes insipidus (DI).

He was started on hormone replacement with thyroxine, testosterone and desmopressin. Symptoms improved initially but over the next three months he noted progressive weight loss and increasing flank pain. On repeat abdominal examination a mass was found in the left upper quadrant and CT revealed multiple abdominal and mediastinal lymphadenopathy. On mediastinal lymph node biopsy he proved to have a high-grade B-cell lymphoma. Initial treatment with high-dose acyclovir and reduction of immunosuppression yielded no clinical or radiological improvement and a course of gamma-interferon was similarly without benefit. He subsequently went on to six cycles of CHOP chemotherapy (cyclophosphamide, doxorubicin, vincristine, prednisolone) with prompt clinical improvement and complete radiological regression of abdominal and mediastinal disease. Repeat pituitary MRI revealed resolution of the stalk lesion (Figure 2), though the hypopituitarism and DI persisted.

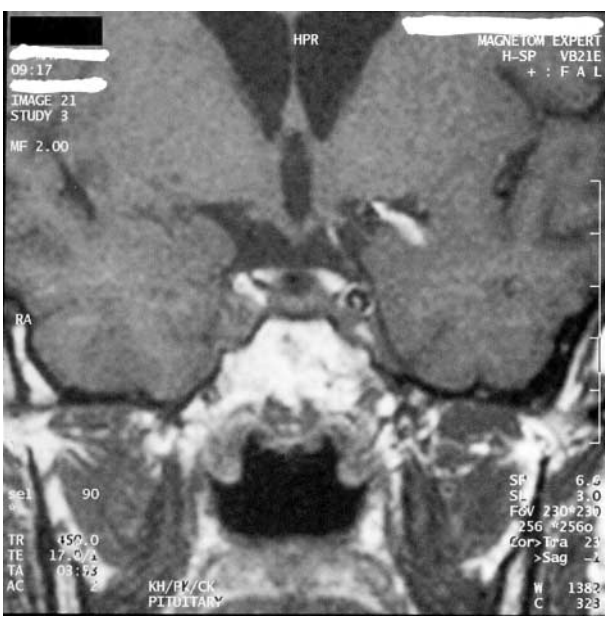

Figure 2 **Coronal view of pituitary MRI showing resolution of stalk lesion after chemotherapy**

COMMENT

Was the suprasella lesion a component of the post-transplant lymphoproliferative disorder (PTLD)? We think this likely in view of the close temporal relationship between presentation of the suprasella lesion (with pan-hypopituitarism and DI) and systemic lymphadenopathy, resolution of both following chemotherapy, presence of atypical lymphocytes in the CSF, and exclusion of other causes. To our knowledge, an association of PTLD with pan-hypopituitarism and DI has not been reported previously, though lymphomas have been described as a rare cause of these disorders in the non-transplant population[2–4].

The diagnosis of PTLD involving the central nervous system can be challenging[1]. In the present case treatment with exogenous glucocorticoids may have masked some of the common symptoms and signs of hypopituitarism, delaying presentation and diagnosis. The pituitary stalk lesion was not easily accessible to biopsy without substantial risk. The diagnosis remained ambiguous for several months until he developed abdominal and mediastinal lymphadenopathy.

Prompt diagnosis of PTLD is crucial for two reasons. First, it allows initiation of specific treatment for this life-threatening disease. PTLDs commonly regress when immunosuppressive therapy is reduced or discontinued. Other regimens include antiviral agents, chemotherapy, immunotherapy (alpha and gamma interferon, gamma-globulin, anti-B-cell monoclonal antibody), radiotherapy and surgery[1,5,6]. Currently aggressive polydrug chemotherapy and alpha-interferon are seen as the treatment of choice for PTLD refractory to reduced immunosuppression[6]. Secondly, it seems that some of the pituitary endocrinopathies produced by lymphoma may recover if treatment is initiated early. Both resolution of DI and improvement in hypopituitarism have been documented following treatment[2,4].

With the growing number of organ transplant recipients, and the increasing intensity of immunosuppression, the incidence of PTLD will rise. The diagnosis of lymphoma should be considered in a transplant recipient or any chronically immunosuppressed subject who presents with hypopituitarism and/or diabetes insipidus.

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Spontaneous extradural haematoma with sinusitis

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The most important causes of spontaneous intracranial extradural haematoma are coagulation disorders, dural vascular malformations and infection[1]. An association with frontal sinusitis has been reported on a few occasions.

CASE HISTORY

A man aged 17 had frontal headaches for three days. He attended the emergency department and was found to be neurologically normal. Oral temperature was 37.7°C and frontal tenderness was elicited on palpation. White cell count was 7.7×10^9/L (normal 4.0—11.0), erythrocyte sedimentation rate was 44 mm/h (2–8) and coagulation screen was normal. Frontal sinusitis was diagnosed, oral amoxycillin was prescribed and he was discharged home. Three days later he was admitted to hospital after a sudden exacerbation of the headache associated with nausea and vomiting. There was no history of head injury. He remained neurologically normal. A non-enhanced computed tomogram showed a right frontal extradural haematoma

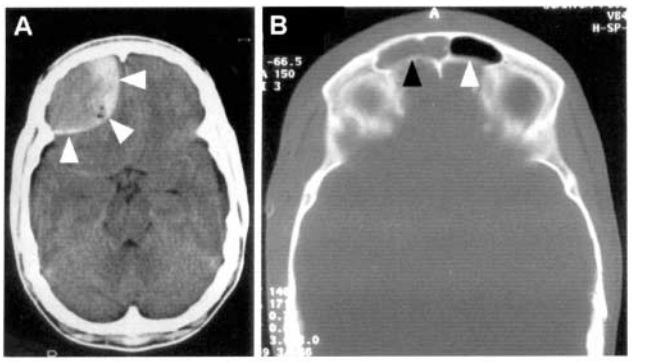

Figure 1 **Preoperative, non-enhanced, axial computed tomograms of the brain.** (A) Right frontal extradural haematoma (arrows). (B) Mucosal thickening/fluid in the right (black arrow) but not the left (white arrow) frontal air sinus, consistent with right frontal sinusitis

(Figure 1) which was then evacuated via a frontal craniotomy. No pus was seen and there was no apparent bony or dural abnormality. Histological examination showed a blood clot containing clusters of polymorphonuclear cells. No organisms were seen on Gram staining and culture yielded no growth. The patient received a two-week course of intravenous cefotaxime and metronidazole and recovered fully. Three months later he was symptom-free and a computed tomogram showed no recurrence of the haematoma.

COMMENT

Only eight cases[1–7] of spontaneous intracranial extradural haematoma complicating infection have been recorded to date. Four patients had chronic otitis[2,4,5,7], three had frontal sinusitis[3,4,6] and one had orbital cellulitis[7]. Seven of these cases[2–7] were diagnosed before the availability of computed tomography. A causal relation between infection and extradural haematoma is supported by the fact that, as in the present case, the extradural haematoma was always adjacent to the infected region.

We hypothesize that the extradural haematoma was caused by spread of inflammation beyond the confines of the sinus. This may explain the presence of clusters of neutrophils in the haematoma of our patient. The hypothesis is also supported by histological and radiological evidence of inflammation in contiguous bone and dura in patients with sinusitis[7]. Once the dural vessels adjacent to the infected region become inflamed, the vessel walls may weaken and the vessels may become prone to bleeding even after subclinical trauma. Progressive detachment of the dura from the inner table of the skull may also be facilitated by the accumulation in the extradural space of a sympathetic effusion, or of air through a bony defect[3].

When headache develops in a patient with sinusitis, otitis or facial cellulitis, brain abscess and subdural or extradural empyema should be considered. We suggest that

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a sudden-onset headache in these patients might also indicate a spontaneous extradural haematoma. Computed tomography of the brain should be performed urgently: deterioration in the patient's level of consciousness reflects delay in establishing the diagnosis. Prompt evacuation of the haematoma and a course of antibiotics should lead to complete recovery.

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