Superior sagittal sinus thrombosis, an unusual presentation of acute myeloid leukaemia: a case report

S Navaratne, C J Blakeley, K Hashemi

A 47 year old woman presented to the emergency department with a two week history of a global headache and a one day history of vomiting, photophobia, and blurred vision. She also reported night sweats during that period. Her general practitioner had prescribed simple analgesics for the headache, but they had had little effect.

She had been healthy previously and was on no regular medication. General examination revealed temperature 36.8 °C, pulse 84 per minute, and BP 120/90 mm Hg, bilateral subconjunctival haemorrhages, and left axillary lymphadenopathy. Chest and abdominal examinations were unremarkable. Neurological examination revealed the patient to be fully alert and orientated with a Glasgow Coma Scale score 15/15. She had neck stiffness and positive Kernig's sign suggesting “meningism”, but there were no signs of raised intracranial pressure at this stage.

Routine blood investigations revealed haemoglobin 8.9 g/dl, white blood cell count $23.0 \times 10^3$, platelets $18 \times 10^6$, international normalised ratio 1.59, and prothrombin time 16.3 seconds. An initial blood film identified blast cells of myeloid origin, later confirmed by bone marrow biopsy. An urgent CT scan was unremarkable with the exception of some fluid in the right sphenoid sinus.

A provisional diagnosis of acute myeloid leukaemia with sepsis was made and the patient was commenced on a regimen of broad spectrum antibiotics, cefazidime, and gentamicin, and then later, cytotoxic agents. Despite treatment she continued to deteriorate with decreasing level of consciousness and a magnetic resonance imaging (MRI) scan (fig 1) confirmed superior sagittal sinus thrombosis with extensive surrounding oedema and an area of venous infarction. One week after admission, in spite of treatment, she developed a further large intracerebral bleed (fig 2) and died.

DISCUSSION
Superior sagittal sinus thrombosis is a rare but serious condition presenting with symptoms and signs of raised intracranial pressure, such as headache, vomiting, and papilloedema. Convulsions and haemorrhagic cerebral infarction are well recognised complications. The thrombosis may be a primary event or secondary to a neighbouring focus of infection. The investigation of choice for suspected cerebral venous thrombosis is MRI since a significant number will be missed on computed tomography. Initial treatment of a confirmed thrombosis involves administration of intravenous heparin followed by oral anticoagulation with warfarin. More recently locally introduced thrombolytics have been used with some success in deteriorating patients.

Although thromboembolic events in haematological malignancies are well known, the complication of superior sagittal sinus thrombosis is an extremely rare event and is usually secondary to treatment. In one retrospective study of 283 patients (11%) experienced a thromboembolic event over a 10 year period. These consisted of deep vein thrombosis, pulmonary embolism, and ischaemic stroke; there were no reports of intracranial venous sinus thrombosis.

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Figure 1 MRI scan showing superior sagittal sinus thrombosis, oedema, and venous infarction.

Figure 2 MRI scan showing intracerebral bleed.
A young man with a headache
E Redfern, O Warren

A previously well 24 year old man presented to the emergency department with a two week history of worsening temporal headache and non-specific coryzal symptoms. Due to the protracted course of his symptoms he was initially triaged to minors. On medical assessment, it emerged he had consulted his general practitioner earlier in the course of the illness. He had been prescribed and had completed a course of oral amoxicillin. Twenty four hours before attendance, he developed persistent vomiting and an occipital headache. He complained of neck pain and also of photophobia.

On examination, he had a temperature of 38 °C, and was supporting his head in extension with his hands, while vomiting continuously. He could not tolerate funduscopy because of the photophobia, and his headache worsened on lying down. There were no other significant findings.

Due to his presentation, his case was discussed with the on-call radiologist, who was initially reluctant to perform an emergency CT head scan due to the long history and normal Glasgow Coma Scale score. However, while in the department, the patient developed a mild degree of confusion, (GCS 14, E4V4M6) and the scan was performed. It showed a large, right sided, subdural collection in the frontal region. It was 1 cm deep, with associated mass effect, but no midline shift, and contained a 3 mm fleck of gas. There was dural enhancement (fig 1). The right maxillary sinus was also opacified with pus (fig 2).

The patient was immediately referred to the neurosurgical team at an adjacent hospital. However, prior to transfer, he deteriorated further (GCS 12, E3V3M6). He also developed a right sided ptosis with a dilated, sluggish right pupil. After further discussion with the neurosurgical team, the patient was given intravenous ceftriaxone, metronidazole, dexamethasone, and mannitol.

After transfer the patient underwent a right frontal microcraniotomy and bilateral antral washouts; 60 ml of foul yellow pus was drained. The patient was ventilated overnight. The pus culture revealed *Streptococcus milleri* and mixed anaerobic species. The organisms were sensitive to penicillin and metronidazole. The patient was discharged back to the referring unit a week later, on oral co-amoxiclav and phenytoin.

**DISCUSSION**

Subdural empyema is a collection of pus in the space between the outermost layers of the meninges, the dura, and the arachnoid. Young male patients in their second or third decade of life are most commonly affected. It is mainly a complication of sinusitis, less frequently otitis media or neurosurgical procedures. With prompt diagnosis and
aggressive treatment, overall mortality is still as high as 20%. Recurrence is common requiring further surgery. It has high morbidity of neurological sequelae, particularly epilepsy, and fits may occur years later.

Subdural empyema has a rapid, fulminating course and presents with headache, fever, vomiting, impaired consciousness, and focal neurology. As in our case, a history of preceding sinusitis is not always obvious. CT scan is nearly always diagnostic but can be equivocal and further imaging with repeat CT or magnetic resonance imaging may be warranted. Subdural empyema is a neurosurgical emergency, the treatment of choice being prompt surgical drainage, simultaneous eradication of the primary source of sepsis, and systemic antibiotics.

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An unusual cause of an acutely locked knee
S Blake, C Wright, C Edwards

A 54 year old man presented to the emergency department with an acutely locked left knee. He had had a unicompartmental knee replacement done on this knee 18 months earlier. Plain x rays were taken (fig 1A) which confirmed the dislocation of the meniscal bearing. An attempt to reduce the knee under sedation in the emergency department was unsuccessful and an orthopaedic consultation was requested. An attempted manipulation under anaesthetic to reduce the dislocated bearing also failed. An open reduction was required. A miniarthrotomy was performed using the previous incision. The surface of the dislocated bearing was found to have been damaged (fig 1B) and therefore a new bearing was inserted.

DISCUSSION
To our knowledge this is the first report of a dislocated bearing of a unicompartmental knee replacement presenting as an acutely locked knee. Thousands of knee replacement operations are carried out each year in the UK. Of these, up to 5% are suitable for unicompartmental knee arthroplasty. The vast majority of patients remain satisfied with their unicompartmental knee replacement at long term follow up. The commonest long term complication is wear of the polyethylene liner. It is already recognised that most locked knees are caused by a mechanical block to full extension. Most are caused by torn menisci, 10% are caused by cruciate ligament ruptures, and loose bodies are an occasional finding. It is difficult to identify those patients who will require surgical intervention for their locked knee. Many are able to either unlock their knee spontaneously or are able to do so after adequate analgesia has been provided and they have been encouraged to flex and extend the knee. Arthroscopic assessment of the joint is obviously required for those knees that remain locked. Repeated attempts to unlock the knee by manipulation risks damaging the components of a joint replacement. This was clearly demonstrated in our case. It is proposed that these patients should be referred to orthopaedics early on, with a low threshold for open reduction of the dislocated meniscal bearing.

Figure 1  (A) Plain x ray demonstrating the dislocated meniscal bearing (white arrows). (B) The meniscal bearing after removal, note the damaged edge (black arrow).
An unusual cause of an acutely locked knee

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