Spinal cord compression secondary to a thoracic meningeal cyst after subarachnoid haemorrhage: a case report

Spinal meningeal cysts are rare and their aetiology uncertain, although an association with congenital abnormalities and familial tendency favours a congenital origin.1 Spinal cord is very rare, although intermittent symptoms of back pain and fluctuating spinal cord compression can occur and may be explained by emptying and filling of the cyst.2 We report the case of a thoracic intradural cyst causing painless spinal cord compression after subarachnoid haemorrhage. This has not to our knowledge been previously described.

A 70 year old woman was admitted to the Wessex Neurological Centre in September 1991 with difficulty walking and poor balance. Ten months earlier the patient had collapsed at home with severe headache and a diagnosis of subarachnoid haemorrhage was confirmed on lumbar puncture, which showed uniformly heavy blood staining. A cranial CT showed no evidence of haemorrhage and bilateral carotid angiography was normal. The patient had no abnormal neurological signs and recovered with conservative measures. After discharge she remained well for three weeks and then experienced sudden weakness in her left leg for which she did not seek any medical attention. This resolved after three days and was not associated with any sensory disturbance.

In June 1991 the patient developed a progressive weakness of her right leg followed by increasing left leg weakness. She noticed an ascending numbness involving both legs and by the time of her admission this had reached her umbilicus. She had no sphincter disturbance or back pain.

On admission the patient was ataxic and unable to walk unaided. Clinical examination showed a spastic paraparesis with grade 3 power, the weakness being slightly more pronounced on the right. Both knee jerks were brisk and the planters extensor; the ankle jerks were absent. There was a sensory level at T8; light touch pinprick and temperature were all reduced below this level and joint position sense was absent in both legs. The gait was ataxic and Romberg's test was positive.

Routine blood tests and plain spinal radiographs were reported as normal. MRI of the dorsal spine showed a multiloculated intradural cystic lesion from T4 to T8 having the same signal intensity as CSF on the T1 and T2 weighted images with evidence of spinal cord compression (figure (A), (B)).

Two days after admission a thoracic laminectomy from T4 to T8 was performed. There was an extensive cyst lying mainly on the right side. Using a Valsalva manoeuvre the cyst was shown to gradually fill over a period of time. The cyst was removed throughout the length of the exposure although a portion of the cyst below T8 was left.

Pathological examination of the specimen showed a meningeal cyst with a thickened fibrocollagenous outer wall containing areas of calcification with no inner arachnoid membrane. There was no evidence of previous haemorrhage. Postoperatively there was an initial deterioration of the patient's paraparesis that has since improved although the patient has remained ataxic due to poor joint position sense.

Previous classification of meningeal cysts has been histologically misleading with intradural cysts being designated arachnoid cysts or arachnoid diverticula. A recent classification by Nabours et al, however, suggested that diagnosis and classification should be based on radiological, histological, and operative findings. They suggested dividing meningeal cysts into extradural and intradural and recommended MRI as a preoperative assessment to define accurately the anatomy and tissue structure of the cyst.

Further reports with MRI of sacral meningeal cysts have shown flow sensitive sequences useful in differentiating communicating from non-communicating cysts.4 We consider that it is better to regard a communicating cyst as in our case as a pouch rather than a true cyst.

Extradural cysts have been described more commonly than intradural cysts and whereas intradural cysts are usually asymptomatic,1 presentation with back pain is much more common than cord compression. This is by contrast with our case where the initial symptomatology was of sudden but transient left leg weakness possibly due to filling and then emptying of the cyst or as a result of a vascular event. This was followed later by painless progressive spinal cord compression.

We believe that it is likely that the patient's initial symptoms were related to a change in the CSF dynamics after subarachnoid haemorrhage. This caused a rise in the overall CSF pressure and allowed its
Bilateral chronic subdural haematoma: an unusual presentation with isolated oculomotor nerve palsy

Isolated third nerve palsy is a common presentation of intracranial aneurysms, diabetes mellitus, chronic lymphocytic meningeal inflammation, and cavernous sinus lesions. Bilateral subdural haematomas presenting with an isolated oculomotor paralysis, however, without any other notable symptoms or signs except for mild headache, are unusual.

We report a 60 year old man referred to us with a three week history of mild generalised headache, two weeks of visual blurring, and diplopia for two days. He was known to be hypertensive and on treatment with metoprolol. Relevant medical history included two episodes of transient ischaemic attacks in the form of transient left hemiparesis in 1982 and 1989, for which he was taking warfarin (5 mg per day). The patient was alert and orientated and the only deficit was a complete right oculomotor nerve paralysis. Clinically an aneurysm of the right internal carotid artery was suspected. Surprisingly, CT showed bilateral chronic subdural haematoma (fig. 1). The haematomas were evacuated through bilateral frontal and parietal burr holes. Immediately after the operation the ptosis recovered partially, the pupil reacted sluggishly to light, and six hours later resolution of the third nerve palsy was complete. After the operation he had a transient left hemiparesis that was presumed to be caused by a transient ischaemic attack. Cerebral angiograms performed before discharge did not show any abnormality.

One of us (MMC) previously reported on 114 cases of chronic subdural haematoma and in that series no patient presented with an isolated oculomotor palsy.1

One of the most common pathogenic mechanisms of isolated oculomotor palsy is microvascular infarction of the nerve, which may be associated with diabetes mellitus, hypertension, atherosclerosis, or collagen vascular disease.2 Under these circumstances there is usually partial or complete sparing of the pupil.3 Our case did not have pupillary sparing. When mydriasis is present, compression of the nerve must be considered, as it is the earliest sign of compression.4 The cause of the oculomotor paralysis in our case was presumably pressure of the herniating uncus of the right temporal lobe, a false localising sign, common in raised intracranial pressure due to head injuries and intracranial tumours causing brain shift. Chronic subdural haematoma may also present this way, usually with other localising signs, impairment of higher mental functions, or a deteriorating sensorium. The fact that only the right third nerve was paralysed led us to believe that the right side subdural haematoma was larger. In fact, the CT and findings at operation showed that both were of similar size. Perhaps slight anatomical variation in the position of the third nerve in relation to the tentorial edges and uncal sulci, and also minor asymmetry of the perimesencephalic cistern explains the lateralisation to the right. Rapid recovery of the third nerve after evacuation of the subdural haematoma lends support to our contention that the palsy was due to distortion of the nerve, and not from another cause.

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Intrathecal baclofen pump infection treated by adjunct intraventricular antibiotic instillation

The delivery of intrathecal baclofen via subcutaneous pumps is gaining increasing use in the management of intractable spasticity of spinal origin.5 An uncommon but potentially fatal complication is infection within the pump of the catheter that connects the pump to the intraspinal space.6 We report the case of one such infected pump that was successfully sterilised in situ by the combined use of systemic and intraventricular infection of antibiotics.

A 68 year old man had been receiving intrathecal baclofen via a manually controlled subcutaneous Cordis Secor pump for severe bilateral spasticity and muscle spasms secondary to multiple sclerosis. The pump was operated by carers and medical staff at the nursing home where the patient lived. He was admitted from the nursing home suffering from urinary retention and had no other new symptoms. On examination, he had an oral temperature of 38°C, but was not clinically toxic. He was alert, cooperative, and obeyed commands. He had a severe spastic tetraparesis with power in his left arm only (grade 3/5). He had no meningism and no obvious source of infection.

Microscopy of the urine and three sets of blood cultures were negative. A chest radiograph was normal. Aspiration, microscopy, and culture of the residual baclofen in his reservoir confirmed the presence of a Staphylococcus aureus infection within the reservoir.

His clinical condition was such that immediate removal of the device was not considered mandatory. An attempt was made to sterilise the pump while in situ as the patient was unwilling to undergo surgery to replace the pump if the present one had to be removed. The delivery of intrathecal baclofen was stopped and oral baclofen was started to prevent troublesome spasms in the legs, but this was ineffective. Despite receiving treatment with high dose intravenous fluoxacillin (1g four times daily) and fucidin (500 mg three times daily).