pulsatility and peak pressure to increase. In the presence of a valvular mechanism the communicating pouch would remain at a higher pressure compared with the CSF pressure and so cause spinal cord compression. This led 10 months later to progressive spinal cord compression due to continued filling and enlargement of the pouch resulting in greater pressure on the cord by virtue of its increased surface area.

Histologically in this case there was absence of the inner arachnoid membrane noted in previous studies and no evidence of arachnoiditis or haemosiderin within the cyst making subarachnoid haemorrhage from the cyst or a traumatic aetiology unlikely. This is by contrast with arachnoid cysts that have been described after trauma, inflammation, or haemorrhage,[5] where the cyst wall consists of a delicate connective tissue with a coating of mengiothelial cells.

This case demonstrates the rare association of an intradural meningeal cyst with painless thoracic cord compression. It supports previous studies suggesting the congenital nature of these lesions and the possibility of fluctuating cord compression caused by volume changes in the cyst. We found no evidence of a direct association with subarachnoid haemorrhage, although changes in the CSF dynamics after haemorrhage may lead to pressure changes within the cyst. Our histological evidence points to the presence of a single arachnoid layer as being an inconsistent finding in making the diagnosis of a meningeal cyst.

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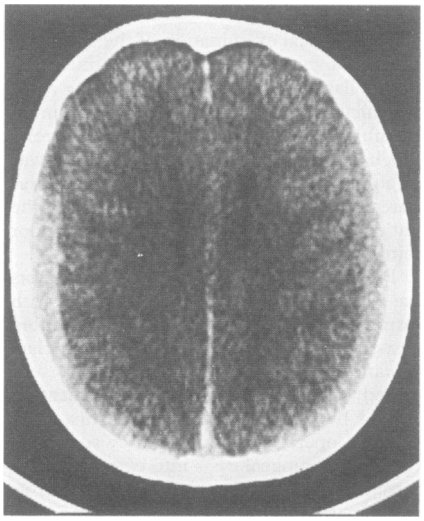
Bilateral chronic subdural haematoma: an unusual presentation with isolated oculomotor nerve palsy

Isolated third nerve palsy is a common presentation of intracranial aneurysms, diabetes mellitus, chronic lymphocytic meningeal infiltration, 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 paralysis. Clinically an aneurysm of the right internal carotid artery was suspected. Surprisingly, CT showed bilateral chronic subdural haematomas (figure). 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 haematomas 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 haematomas 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 unci, 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 any other cause.



Contrast enhanced CT showing bilateral chronic subdural haematomas.

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Intrathecal baclofen pump infection treated by adjunct intrareservoir antibiotic instillation

The delivery of intrathecal baclofen via subcutaneous pumps is gaining increasing use in the management of intractable spasticity of spinal origin.[1] An uncommon but potentially fatal complication is infection within the pump or the catheter that connects the pump to the intrathecal space.[2] We report the case of one such infected pump that was successfully sterilised in situ by the combined use of systemic and intrareservoir injection 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 careworkers and medical staff at the nursing home where the patient lived. He was admitted from the nursing home suffering from a pyrexia, but 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 flucloxacillin (1g four times daily) and fucidin (580 mg three times