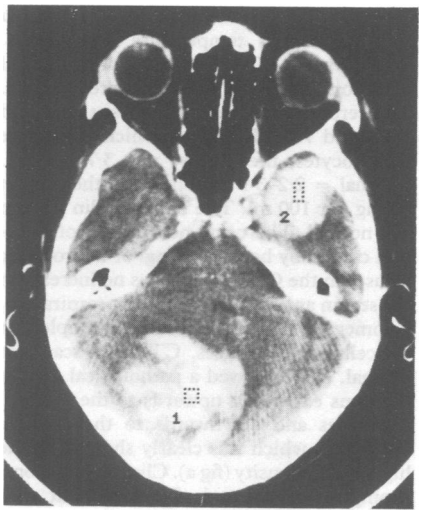

Figure CT scan showing right temporal lobe and left cerebellar hemisphere haemorrhages.

Her limbs were flaccid with symmetrically diminished reflexes and equivocal plantar responses.

She made no response to deep painful stimuli. Initial blood results revealed a leucocytosis of 48·9 × 10^9/l with a differential of eosinophils 32·81 and neutrophils 12·18 × 10^9/l respectively. Her haemoglobin, biochemical screen and liver function tests were all normal. A full coagulation screen including platelets, KPPT, fibrinogen and Protein C were also normal.

A chest radiograph showed cardiomegaly and old calcified apical foci. Her CT scan (figure) showed a right temporo-parietal haematoma with spread into the right lateral ventricle and associated mass effect, and a left cerebellar haematoma with compression of the fourth ventricle but without resultant hydrocephalus. She failed to regain consciousness and died 10 weeks later. Permission for a necropsy examination was refused.

HES is a term which was introduced by Hardy and Anderson in 1968.[1] It is defined as persistent eosinophilia (> 1500 mm^3) in a patient in whom no known underlying cause can be found and who develops end organ injury. Central nervous system disturbance is the second most important clinical manifestation.

In a recent study[2] 65% of patients were affected. Central nervous system dysfunction was seen in seven patients (15%), four of whom had a distinctive encephalopathy. Peripheral neuropathy was found in 27 patients (52%). Embolic phenomena including anterior and middle cerebral artery occlusions were seen in six (12%).

This is the first reported case of HES presenting with intracranial haemorrhages.

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1 Hardy WR, Anderson RE. The hypereosinophilic syndrome. *Ann Intern Med* 1968;65: 1220–29.
2 Moore PM, Harley JB, Fauci AS. Neurologic dysfunction in the idiopathic hypereosinophilic syndrome. *Ann Intern Med* 1985;102: 109–14.

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Meningitis and spinal subdural empyema as a complication of sinusitis

Spinal subdural empyema is a rare event. We describe the case of a young man who developed sinusitis while recovering from a routine operation followed by a dramatic meningitic illness with formation of a spinal subdural empyema.

A previously fit 23 year old man had an elective left temporal mandibular joint meningoplasty for Costen's syndrome. Two days later he developed a mild headache with a slightly purulent nasal discharge. Within the following 24 hours his headache became bifrontal and more severe. He developed visual hallucinations though he remained orientated and there was marked neck stiffness, severe low back pain with very limited straight leg raising and a high fever. There was no previous history of sinus disease or immunosuppression. His lumbar puncture showed turbid green cerebospinal fluid (CSF) with 284 polymorphs and 134 lymphocytes with a CSF protein of 1·5 grams per litre and a low CSF glucose.

Plain skull radiographs showed thickening of the right maxillary sinus with a small gas bubble in the supra-sella cistern. A CT scan confirmed these findings and showed a completely opaque sphenoid sinus. Cultures from his CSF grew *streptococcus milleri* (a microaerophilic haemolytic streptococcus) and a bacteroides of the corrodens type. Both these organisms are compatible with an origin in the upper respiratory tract. Ampicillin, metronidazole and gentamicin were given intravenously. His sinuses were drained and a considerable amount of green pus was obtained. His temperature gradually settled though his severe back pain persisted and his tendon jerks in both lower limbs became depressed. A cervical myelogram was performed and this showed somewhat irregular subarachnoid space in the lower cervical region and a complete block at the level of D7. A subsequent CT scan of the same region confirmed a posteriorly situated mass causing forward displacement of the cord but no bony erosion. He had a laminectomy from D7 to D10. A subdural mass with much granulation tissue was identified and suitable decompressive measures were undertaken. Following this he made a slow but progressive recovery although he was troubled with low back pain and was discharged home after completion of a month of antibiotic therapy.

The association between sinusitis and intracranial empyema is well established. Spinal subdural empyema is a much rarer occurrence and has not been previously described as a complication of sinusitis.

In a 1973 review of the literature only ten cases were described.[1] Five of these patients died, three were left with significant neurological deficits and only two fully recovered. Prompt surgical drainage and antibiotics have obviously improved prognosis, and more recently eight additional cases have been reported.[2] Seven of these patients made full or fair recoveries.

Typically, spinal subdural empyema presents with a fever, backache, and radicular symptoms and signs. There have been claims that the absence of percussion tenderness helps to distinguish it from an epidural collection of pus[1 3 4] which is usually associated with vertebral osteomyelitis or bacteremia. The diagnosis has recently been facilitated with the introduction of spinal CT.[5 6]

In our patient no clear collection of intracranial subdural pus was identified with a CT brain scan with contrast though this may appear normal in the early stages.[7]

The striking feature in this case is the finding of an extensive spinal subdural empyema. It remains unclear whether it was formed by haematogenous spread from an intracranial or paranasal source despite the relative avascularity of the subdural space. The empyema may have derived from extension through the arachnoid in the presence of meningitis. Seeding of the subdural space by lumbar puncture has been described as a possible cause.[8]

Paranasal sinus pathology should be excluded as a cause of pyogenic meningitis even in the absence of a clear history of sinusitis, and should now be considered as a possible cause of spinal subdural empyema.

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1 Fraser RAR, Ratzan K, Wolpert SM, Weinstein L. Spinal subdural empyema. *Neurology* 1973;28:235–8.
2 Probst von CH, Wicki G. Spinale subdurale empyeme und abszesses. *Schweizer Archiv fur Neurologie, Neurochirurgie und Pschiatre Band 134* 1984;1:53–70.
3 Dacey RG, Winn HR, Jane JA, Butter AB. Spinal subdural empyema: report of two cases. *Neurosurgery* 1978;3:400–3.
4 Case records of the Massachusetts General Hospital (Case 47). *N Engl J Med* 1984; 311(21):1365–70.
5 Theodotou B, Woosley RE, Whaley RA. Spinal subdural empyema: diagnosis by spinal computed tomography. *Surg Neurol* 1984; 21:610–12.
6 Lomholdt Knudsen L, Voldby B, Stagaard M. Computed tomographic myelography in spinal subdural empyema. *Neuroradiology* 1987;29:99.
7 Hodges J, Anslow P, Gillet G. Subdural empyema—continuing diagnostic problems in the CT scan era. *Q J Med* 1986;288:387–93.
8 Negrim J, Clark RA. Pyogenic subdural abscess of the spinal meninges. Report of two cases. *J Neurosurg* 1952;9:95–100.

Familial cerebellar ataxia and possible cosegregation with an inversion in chromosome 4

We have recently encountered a family which may shed light on the gene locus of an inherited form of late onset cerebellar ataxia.

The family pedigree (fig) reveals that three of seven siblings have or have had late onset cerebellar ataxia. Case II2 died aged 65 years. From the age of 55 years relatives noticed that she had a progressively unsteady gait, and slurred speech. None of her medical records can be traced. Case II4 died aged 74 years. She was first referred to a neurologist at the age of 60 years with a one year history of unsteady gait and dysarthria, which showed slow but steady deterioration culminating in marked disability. Relevant findings on examination at referral included nystagmus on lateral gaze, normal optic discs, and normal tone, power, tendon reflexes and sensation in all limbs. Air encephalography did not show any obvious cerebellar abnormality nor was any noted at necropsy examination.

The necropsy report recorded that this woman had a chromosome abnormality although the precise nature of this was not specified. Review of her medical records and personal contact with all known locally sited