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. The striking feature in this case is the finding of an enigmatic subdural empyema. It remains unclear whether it was formed by haematogenous spread from an intracranial or parasanal source despite the relative avascularity of the subdural space. The empyema is thus a complication through the arachnoid in the presence of meningitis. Seeding of the subdural space by lumbar puncture has been described as a possible cause. Parasanal 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.

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. The empyema may have derived from extension of the infection of the subdural empyema.

Correspondence to: Dr R Harries-Jones, Hereford General Hospital, Hereford HR1 2PA.


3 Correspondence to: Dr Roche, Department of Medicine for the Elderly, St Charles Hospital, Exmouth Street, London W10, United Kingdom.