Letters

question as to why the phenomenon is not seen more widely, since many patients with subclinical tremor must suffer wrist injuries of this type.

JD COLE
LS ILLIS
EM SEDGWICK
Wessex Neurological Centre,
Southampton General Hospital,
Southampton,
SO9 4XY, UK

References


Accepted 21 October 1988

Superior sagittal sinus thrombosis in Wegener's granulomatosis

SIR: We report the occurrence of sagittal sinus thrombosis in a patient with systemic micropolyarteritis, an association we believe not previously described.

A 57 year old man had developed insulin dependent diabetes mellitus at the age of 19. He first experienced headaches in 1977. He became systematically unwell in 1981 with a rising plasma creatinine and an ESR of 123 mm/h. A renal biopsy was performed, and an arteriovenous fistula was fashioned but not used because renal function reverted to normal spontaneously.

In 1983 he again became unwell with repeated epistaxes, facial numbness, headache and persistently high ESR. His symptoms responded to corticosteroids.

In 1984 he developed visual distortions and subsequently visual obscurations. CSF pressure was normal though protein was 0·66 g/l.

In 1987 he presented with papilloedema. CSF pressure was 30 cm H2O. There were 7 white cells/mm³, protein 1·77 g/l. CSF electrophoresis showed an oligoclonal pattern indicative of local synthesis. Cranial CT (fig. a) revealed thickening of the meninges and low density in the sinus confluence after intravenous contrast, consistent with sagittal sinus thrombosis ("empty delta" sign). This was confirmed by angiography (fig. b).

A lumbo-peritoneal shunt produced an improvement in the visual symptoms and headaches, though recovery was complicated by two focal seizures.

Further renal biopsy and review of the 1981 histology showed a focal necrotising micropolyarteritis. Anti-neutrophil cytoplasmic antibodies were positive at 20% (normal <1%). Other auto-antibodies including anti-nuclear antibodies were negative. Coagulation was normal and the lupus anticoagulant was absent. Examination of the nasal septum revealed bilateral ulcerating lesions. Biopsy showed chronic inflammatory changes. Otological examination was normal.

These clinical features and investigations suggest a systemic micropolyarteritis of the Wegener's type. Wegener's granulomatosis involves the nervous system in up to 50% of cases, typically causing cranial and peripheral neuropathies, aseptic meningitis, diabetes insipidus and focal lesions of brain and spinal cord. The pathogenesis of these complications is by granuloma formation, or more commonly by focal arteritis. Small arteries are most commonly affected.

In our patient we postulate the sagittal sinus thrombosis to be a result of vasculitis, either by extension of thrombus from inflamed small vessels, or as a consequence of chronic aseptic meningitis. Lateral sinus thrombosis has been described in Wegener's granulomatosis but only in a septicaemic patient with otitis media and mastoiditis. We are not aware of any other previous report of involvement of the large intracranial veins and sinuses in Wegener's granulomatosis, though systemic lupus erythematosus may rarely produce this complication.4

We thank Dr R Ross Russell and Dr D Taube for permission to publish details of a patient under their care.

S R HAMMANS
L GINSBERG
The National Hospital for Nervous Diseases,
Queen Square, London,
WC1N 3BG UK

Correspondence to Dr L Ginsberg, Queen Square, London WC1N 3BG

References