Letters

Tuberculous meningitis presenting with trismus

Sir: Trismus may be the result of local pathological processes affecting the jaw or teeth, tetanus or phenothiazine medication. We have recently seen a patient in whom trismus was the presenting feature of tuberculous meningitis. A 48-year-old man was admitted with a 4 week history of involuntary episodic twitching of the right side of his face. The episodes had lasted about one minute then subsided for minutes or hours. One week before admission he developed painful spasms of the jaw muscles on the right lasting several hours during which he was unable to open his mouth. Simultaneously he noticed numbness of the right side of his face, initially in the distribution of the ophthalmic division of the trigeminal nerve spreading to involve the maxillary and mandibular areas. His personal, family and drug history were not contributory. On examination there was increased lacrimation on the right and complete anaesthesia in the distribution of the right trigeminal nerve. The jaw was tightly closed due to spasm of the right masseter and temporalis muscles which were slightly wasted. Occasional twitching of the right platysmal muscles was noted. The remainder of examination was normal. Extensive laboratory investigations revealed only an elevated erythrocyte sedimentation rate (45 mm/h, Westergren). Radiographs of the skull, including basal views, and chest were normal. CT scan of the posterior fossa was normal. Lumbar puncture yielded clear fluid under slightly raised pressure (230 mmH2O) containing protein 94 mg/l, glucose 1.01 mmol/l (blood glucose 6.4 mmol/l), and chloride 102 mmol/l. Microscopy revealed 248 lymphocytes/mm3, 70 neutrophils/mm3, and acid fast bacilli. Mycobacterium tuberculosis was later cultured from the CSF. Treatment was started with isoniazid (400 mg/day), rifampicin (600 mg/day) and ethambutol (1200 mg/day). Marked relief of the trismus was obtained after 10 weeks on this regimen; the facial numbness, however, persisted until discharge three months after admission.

Chronic meningitis may present with a wide variety of subtle signs and symptoms. In this case the findings were of right trigeminal nerve involvement in the absence of other signs of meningeal irritation or systemic features and proved highly misleading.

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Transient global amnesia — familial incidence

Sir: Transient global amnesia1 2 is thought to be due to transient ischaemia of the medial aspects of both temporal lobes or medial thalamic nuclei3 4 and to be encountered mainly in middle-aged or elderly men who are assumed to have atherosclerosis affecting the vertebrobasilar circulation.5 A recently published report6 recorded a familial incidence of this condition and we wish to report a further example.

A 53-year-old man was admitted for investigation of two amnesic episodes. In the first one, three years previously, he had been found by the police standing by his motor-cycle on a bridge not knowing how he had got there or what he was doing. There were no signs of injury and he behaved normally though being left with a memory deficit lasting eight hours. The second and recent episode occurred at home whilst he was working in his own garage servicing a small but intricate steam engine. He was heard by his wife to switch the engine on and off and one hour later he went in the house and told his wife that he felt as though he had had a stroke. He could not remember being in the garage, he looked pale, appeared confused, could not retain information that his wife had just given him, and kept repeating the same questions. However, he ate his lunch, went back into the garden covered sensibly with his neighbour and three hours later he became normal, though with memory deficit affecting events of the previous four hours. On physical examination he was normal and a number of investigations including ECG, EEG and CT were normal. The patient described similar episodes in his family. At the age of 56 the father had an episode of amnesia lasting three hours during which he was able to drive his car and was noted to be in a “vague mood” subsequently recovering fully. He had two further similar episodes and was investigated at a London hospital with negative results. He died in his eighties.

A sister had a 12-hour long episode of amnesia at the age of 66: she woke up one morning confused and in an “odd mood”. She was able to dress and cook breakfast and when later seen by the general practitioner the only abnormality found was that of impaired memory. When fully recovered her comment was that she felt as though she had been “a long way off”. There have been no further recurrences. Another sister has so far remained asymptomatic.

It is difficult to explain such familial incidence of transient global amnesia other than by postulating atherosclerosis in the vertebrobasilar system with chance susceptibility of the inferolateral temporal lobes in these three relatives. Alternatively, it is also possible that familial transient global amnesia is a common phenomenon, which simply has not been reported as a significant finding until recently.5 It would be of interest to see whether such indeed is the case.

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References


Diagnostic significance of free sialic acid in cerebrospinal fluid in meningitis

Sir: Earlier workers have reported on the levels of free and bound cerebrospinal fluid (CSF) sialic acid in various neurological disorders.1–6 We were prompted to examine the claim that determination of free sialic acid in CSF can be used as an aid in the diagnosis of pyogenic meningitis and its differentiation from tuberculous meningitis.4 6 Using essentially Warren’s method7 free and