Letters

Transient global amnesia and migraine: familial incidence

Sir: The pathophysiology of transient global amnesia is unclear; often an ischaemic aetiology has been suggested, but epileptic and migrainous mechanisms have also been invoked. A familial incidence in this syndrome has rarely been reported. We describe a family with an unusual incidence of transient amnic attacks and migraine.

ER, a 72-year-old woman with 10 years of schooling, was admitted to hospital in March 1985, after a temporary attack of altered behaviour dominated by disturbance of memory. Two hours before she had been at home, conversing with her daughter-in-law, when suddenly she became strange, astonished and did not recognise some household objects as familiar; she asked her daughter-in-law about her presence in the house. She was not able to remember having had a walk in the morning and having prepared a trip to Milan for the next day in order to attend an opera at La Scala Theatre (she had been very excited about this on the previous day). On admission she was still confused, disoriented in time but not in space. General examination, ECG and neurological examination were normal. She asked the same questions over and over again, showing great difficulties in grasping simple information. Other high cerebral functions, clinically evaluated, were preserved. Retrograde amnesia was present, covering a period of several weeks. During the examination she began to recover and after 1 h she was apparently normal. Blood parameters, EEG, CT brain scan and cerebro-vascular Doppler sonography, subsequently performed, were normal. Neuropsychological tests for memory (verbal and spatial span, Rey 15 Words, Rey Complex Figure), administered a month later, gave normal findings; at this time, only amnesia for the period of the attack still remained. The patient's history revealed that she had suffered from common migraine until the menopause, and her mother and two siblings had suffered from vascular headaches (common and classic migraine). Furthermore, a brother of hers, MR, a 76-year-old healthy man, with 7 years of schooling, had suffered from an acute, transient attack of amnesia at the age of 52: after a windy day spent hunting with some friends, he had a 6–8 hour long episode of amnesia, without loss of consciousness, during which he was able to speak, walk and drive. We examined this man, who had no physical abnormalities. Neuropsychological tests for memory proved normal. He was still able to describe the amnesic episode in sufficient detail, even though it happened more than 20 years before. His medical history was uneventful except for that episode and for migraine which in adolescence had been so marked that he had to interrupt high school. A sister had also had an episode of memory disturbances when she was young, but she had died many years before and we had no details about her. We think that, while ER suffered from a classical transient global amnesia attack, MR had probably had transient global amnesia with partial recollection of the episode, analogous to other rare observations.

Previous reports about familial transient global amnesia suggest that familial incidence may be explained by a high frequency of risk-factors for cerebro-vascular disease in the same family. Alternatively, it is possible that episodes of transient global amnesia in members of a same family are a coincidence, because of the frequent occurrence of this condition, which is certainly more frequent in medical practice than has been described. However, we would argue that the migraneous hypothesis of transient global amnesia is also compatible with a familial incidence of this syndrome and the presence of transient global amnesia and migraine in the same family supports this explanation. Even though the high frequency of both conditions can also be invoked to explain their association, this argument is not very convincing. We think that the hypothesis of a single mechanism underlying both transient global amnesia and migraine (the transient global amnesia-migraine connection), as suggested by their presence in the same family, is an interesting one and should be the subject of further investigations.

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References


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Taurocyamine in cerebrospinal fluid of neurological and psychiatric patients.

Sir: In 1981 Mori et al. reported that o-amino acids could be the precursors of the corresponding guanido compounds. Taurocyamine could be derived from taurine. Taurine, considered as an inhibitor of impulse transmission in the central nervous system, occurs also in cerebrospinal fluid (CSF). Taurocyamine has been demonstrated in the CSF of uraemic patients and in some epileptics but not in normal neurological patients. The purpose of our study was to evaluate the presence of taurocyamine in CSF of patients with neurological and psychiatric diseases.

The study includes the analysis of 206 cerebrospinal fluid samples of patients with neurological and psychiatric diseases (see table). The neurological patients were classified according to the Ninth Revision of the International Classification of Diseases. The psychiatric patients were classified according to the Third Edition of the Diagnostic and Statistical Manual of Mental Disorders. CSF was obtained by lumbar puncture. 3 ml of CSF were dried on a rotatory vacuum evaporator at 30°C and dissolved in 0.8 ml 0.2 N sodium citrate buffer, pH 2.2. The sample was directly applied to the chromatographic system.

The separation of taurocyamine was carried out with 0.2 N sodium citrate buffer, pH 3.28. The column was a 69 × 0.9 cm chromatographic column with 55 cm bed of spherical resin (flow rate 50 ml/hr, 30°C) in an automatic amino acid analyser (Beckman, Unichrom) modified for guanido compounds analysis. The color reaction developed with Sakaguchi Reagen has been described by Durzan.

Taurocyamine was eluted after 48 min. Under our conditions taurocyamine can be estimated at a concentration as low as 4 nmol/ml when using 3 ml CSF and read out of the chromatographic pattern of a CSF specimen with and without addition of standard.

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