Short report

Epilepsy and coeliac disease

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SUMMARY A number of neurological disorders have been described in association with coeliac disease, including epilepsy. A review of 177 patients with coeliac disease failed to show an increased prevalence of epilepsy. This is contrary to the findings of other workers, and requires further investigation.

Since coeliac disease was first described by Samuel Gee in 1888¹ there have been several reports of various neurological disorders associated with it. These include peripheral neuropathy, demyelination of the posterior columns, depression, deterioration in mental function, schizophrenia and epilepsy.²⁻⁷ Morris *et al* described "epileptiform convulsions" in two out of thirty patients, while Cooke and Smith noted "attacks of unexplained loss of consciousness" in five out of sixteen patients, giving a prevalence of 6% and 31% respectively. Chapman *et al*, looking specifically at the association with epilepsy, found a prevalence of 5.5%, with a high proportion of temporal lobe epilepsy (77%). We report here the prevalence of idiopathic epilepsy in a review of 177 patients with coeliac disease.

Patients, methods and results

One hundred and ninety-seven patients with coeliac disease were circulated with a questionnaire inquiring about the occurrence of faints, fits, blackouts and other common symptoms of epilepsy. The criteria for coeliac disease were taken as an original abnormal jejunal biopsy of the coeliac type, and clinical response to gluten-free diet. The diagnosis of epilepsy as a basis for episodes of loss of consciousness was based on the presence of seizure activity in the EEG, together with clinical details obtained from the patient, and when available, details obtained from an independent witness. When the EEG failed to reveal seizure activity, the diagnosis was based on clinical details obtained from the patient, and when available, from an

independent witness of the attacks. Of the 177 patients (mean age 21 years; range 2-76; 91 males, 86 females) who completed and returned the questionnaire, thirty-six had one or more symptoms suggestive of epilepsy, and these were reviewed under the supervision of a consultant neurologist (NC). A further nine patients had a family history of epilepsy, but were not included in the review. Two out of thirty-six patients were considered to have idiopathic epilepsy, on the basis of history and epileptic feature in the EEG. This gives a prevalence of 1% compared to 0.5% in the general population.⁸ The difference between the two groups is not statistically significant (to test significance the normal approximation to the binomial was used, assuming p to be 0.005). The diagnosis in the remaining thirty-four patients was vasovagal attacks (20), anxiety states (7), febrile convulsions in childhood (2), post-traumatic epilepsy (2), vertigo (1), and cerebral concussion (1). One other patient had a history of blackouts in childhood, strongly suggestive of epilepsy, but at the time of the study, she had been free of symptoms for over three years, and had a normal EEG; consequently, she was considered not to have epilepsy.

Discussion

The explanation for the supposed association between coeliac disease and epilepsy is far from clear. Pyridoxine deficiency has been shown to cause convulsions in childhood,^{9,10} and although low serum levels of pyridoxine have been found in coeliacs, they have rarely been low enough to cause convulsions.³ Other explanations include magnesium deficiency, calcium deficiency and genetic factors. If an increased prevalence of epilepsy does exist among patients with coeliac disease, there are a number of important conclusions. Subclinical cases of coeliac disease may account for poor seizure control, through impaired drug absorption. Other neuro-

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logical disorders, such as schizophrenia,^{11 12} metal deterioration¹² and mood changes,^{13 14} sometimes improve on gluten withdrawal; could the same be true of epilepsy? This study suggests that there is no increased prevalence of epilepsy in patients with coeliac disease. These findings are contrary to those of a similar study by Chapman *et al* (1978). This may be due to the relatively small numbers involved, and we feel that a larger study is needed to resolve this problem.

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