Correspondence

Coeliac disease: does it always present with gastrointestinal symptoms?

Sir,
Coeliac disease (CD) is an immune-mediated enteropathy that was once perceived to be primarily a paediatric disorder is now being diagnosed with increasing frequency in the adult population. Although it primarily affects the gastrointestinal (GI) tract, several patients may be asymptomatic or have extra intestinal symptoms at the onset. Neurological involvement like epilepsy has been associated with the disease but the cause is far from clear although an autoimmune mechanism has been often hypothesized. Improvement of symptoms and subsidence of seizure activity has been often successfully achieved with a gluten-free diet. We present one such patient with CD with epilepsy and also a brief discussion about the recent literature.

Case
A 43-year-old Caucasian gentleman with no significant past medical history was brought into hospital by his wife. She noticed her husband on bed during the night hours in a rigid state with uprolling of eyeballs, which lasted for about 5 minutes. He was unresponsive during this phase but gradually recovered with a mild headache and fatigue. On further enquiry, he confirmed that there was no recollection of the event and he never had a similar experience in the past. There was no history of any febrile illness, tongue bite, frothing from mouth, incontinence, speech abnormality, focal weakness or any injury, and the clinical examination was unremarkable.

His investigations revealed a normal full blood count, urea and electrolyte’s, glucose, liver function and thyroid function but with a slightly low level of corrected calcium of 2.07 mmol/l (normal albumin) and phosphate of 0.60 mmol/l which led to the estimation of vitamin D and parathormone levels and both of which were within normal limits. This was followed with a computed tomography scan of brain and chest X-ray, which did not show any abnormality. Taking things forward to investigate a slightly low level of calcium in a Caucasian patient, IgA tissue transglutaminase (TTG) antibody test was requested and this showed an abnormal value of 138 U/ml (0–10). CD was confirmed with an upper GI endoscopy and biopsy, which showed a benign gastric mucosa but a subtotal villous atrophy in the duodenum. The patient was referred to the dietetics service and was advised for gluten-free diet. His blood tests were repeated after 2 months which showed an improved corrected calcium level of 2.18 mmol/l and phosphate level of 1.04 mmol/l. He is presently under regular review and no further recurrence of seizure activity has been reported.

Discussion
CD is an immunologically mediated condition related to intolerance to dietary gluten. Although classical signs are related to the GI tract, extra intestinal manifestations like anaemia, coagulopathy, metabolic bone disease, neurological disorders, psychiatric syndromes and infertility are also encountered.1 The prevalence of epilepsy in CD has been suggested to be high2,3 but other neurological disorders like cerebellar ataxia, peripheral neuropathy, dementia, myoclonus and myelopathy are also noted commonly.4,5

The precise mechanism of association between CD and epilepsy is unknown but several hypotheses have been proposed. Some suggest that the antibodies associated with CD may be themselves neurotoxic or, alternatively, may be a marker for a neurotoxic immunological process.6 Several authors have successfully demonstrated autoantibodies like antiendomysium, anti-TTG and anti-reticulin in their cohort of epileptic patients.7–9 Other explanations include deficiency of calcium and magnesium, suboptimal drug absorption and genetic factors10 or due to oxidative stress and free radical accumulation.1
The onset of the seizures are quite variable in CD as the duration of gluten exposure is important and a correlation can be found between the duration of the disease preceding the diagnosis (and treatment) and neurological findings as detected even years after the diagnosis. The two conditions may coexist for a prolonged period of time before the actual clinical manifestations which may be till the late middle age as in our patient.

Our case demonstrates that neurological symptom such as epilepsy may be the first manifestation of CD and the diagnosis may be often challenging as the frequency of undiagnosed CD in neurological manifestations of unknown origin may be as high as 16%. A high index of suspicion for CD should be borne in mind in patients with epilepsy with nonspecific GI or constitutional symptoms. Although routine screening for CD in all patients with epilepsy is not cost-effective, it is reasonable to perform it in patients with neurological dysfunctions of unknown cause and in patients with intractable epilepsy. Early institution of a gluten-free diet in combination with antiepileptic treatment is beneficial as delay in diagnosis of CD in epilepsy patients or poor dietary compliance may adversely influence the overall outcome and lead to complications.

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References

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Follow-up of muscular sarcoidosis using fluorodeoxyglucose positron emission tomography

Sir,

Sarcoidosis is an immune-mediated condition affecting numerous organs, especially the lungs, lymph nodes, skin and eyes. Although asymptomatic muscle involvement is common in sarcoidosis (50–80% of histological cases), symptomatic myopathy is rare, being encountered in 0.5–2.3% of the patients. We recently observed a case of symptomatic myositis revealing a recurrence of sarcoidosis with favorable outcome after initiation of infliximab; our case is of particular interest, as it indicates that whole body fluorodeoxyglucose positron emission tomography (FDG-PET) is a useful test for both diagnosis and follow-up of myopathy in sarcoidosis.

A 43-year-old man was diagnosed as having both biopsy-proven pulmonary and muscle sarcoidosis in October 2005. In October 2008, the patient was...