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Letter to the Editor

Exploring the iceberg of gluten

Sir,

Gluten, a protein present in wheat, rice or barley shares the reputation of common etiological agent for childhood disease 'Coeliac Disease' (CD) and adult 'Non-Coeliac Gluten Sensitivity' (NCGS).

Affecting around 1% of children worldwide, CD is an intestinal form with predominant gastrointestinal manifestations. It is characterized by histopathological triad of duodenal villous atrophy, cryptal hyperplasia and intense intra epithelial lymphocytic response with or without transglutaminase (TG) or endomysial antibodies (EMA) as auto antibodies in serum.[1]

While in adults, NCGS is around six times more common with its neuropsychiatric presentation than CD. NCGS lacks histopathological duodenal features but is frequently associated with IgA or IgG antigliadin antibodies due to intense immune response.[2]

Both the central and peripheral nervous system are affected in NCGS but predominantly it presents as Cerebellar Ataxia (Gluten Ataxia).

An irreversible loss of purkinje fibres in vermis part of cerebellum with associated atrophy and gliosis is manifested clinically as limb or gait ataxia or even dysartheria. It has been evident that institution of gluten free diet (GFD) with elimination of antigliadin antibody improves or stabilizes the ataxia on clinical ground.[3]

The other common presentation of NCGS is chronic progressive, symmetrical, sensory motor, axonal peripheral neuropathy which is seen in approximately 70%-75% cases anytime during course of disease. At times small fibre neuropathy as painful burning sensation in distal parts of upper and lower limb is also associated.[4]

Similarly, gluten sensitivity might be the underlying cause of medically refractory epilepsy which may or may not be associated with neuroimaging features. These patients respond well to anti-epileptic drugs if tagged with gluten free diet.[5] With extended involvement of central nervous system gluten sensitivity produces features of severe headache, vertigo, insomnia, anxiety, depression along with cognitive and memory impairment.

The diagnosis of NCGS is generally delayed by an average of 8-9 years due to predominant neuropsychiatric manifestations in absence of gastrointestinal features. These symptoms as initial presentation are also present in other commonly prevalent diseases.

The clinical suspicion of NCGS rests upon unexplained neurological features like gait ataxia, peripheral neuropathy and refractory seizures. If these symptoms are further supported by seropositivity of antigliadin antibodies or more recent tTG6 antibodies, the suspicion becomes much stronger.

A strict gluten free diet is the simplest and the cheapest method to ameliorate the troublesome symptoms associated with gluten hypersensitivity.[6] These unexplained common neuro psychiatric symptoms should be evaluated in light of underlying gluten sensitivity early in the course of disease so that abstinence from gluten as therapeutic measure could be imposed for a better outcome.

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