

Case Report

Multiple sclerosis or neurological manifestations of Celiac disease

Vahid Shaygannejad, Majid Ghasemi, Maedeh Mirmohamadsadeghi

Department of Neurology, Isfahan University of Medical Sciences School of Medicine, Isfahan Neuroscience Research Center
Isfahan, Iran

Abstract

Multiple sclerosis (MS) and celiac disease (CD) are considered to be T-cell-mediated autoimmune disease. We discuss about a known case of CD-showed relapsing – remitting neurological symptoms compatible with MS. In this rare co-occurrence subject, MS-CD patient, the interaction between MS – and CD-related inflammatory processes is open to discussion.

Key Words: Ataxia, celiac disease, diplopia, multiple sclerosis

Address for correspondence:

Prof. Vahid Shaygannejad, PO Box. 761, Department of Neurology, Alzahra Hospital, Soffeh Ave., Isfahan, Iran. E-mail: shaygannejad@med.mui.ac.ir
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INTRODUCTION

Multiple sclerosis is an inflammatory disease of central Nervous system (CNS) and it is sometimes difficult to differentiate from CNS involvement in systemic autoimmune diseases. Celiac disease (CD) is a gluten-sensitive enteropathy (GSE). Patients develop small bowel villous atrophy, malabsorption and weight loss, all reversed by a strict gluten-free diet (GFD).^[1]

Susceptibility to CD involved a combination of environmental genetic factors and immunologic mechanisms. It is activated by protein in the dietary cereal grains known as gluten and associated with specific major histocompatibility complex (MHC) II alleles that encode specific human leakage antigen (HLA) DQ2 or HLA DQ8 heterodimers.^[2]

Besides to enteropathy, CD has various extraintestinal expressions including neurologic symptoms, but it is uncertain whether these signs are caused by gluten ingestion.^[3]

Numerous neurologic conditions, including epilepsy, sensory ataxia, and neuropathy have been reported in association with established CD.^[4] We report a case of CD who was discovered to have MS.

CASE REPORT

Our patient is a 37-years-old woman with a history of chronic abdominal pain since 2004. Anti-endomysial antibody (Ab) of isotype IgA (16 u/mL), and anti-gliadin antibody of isotype IgA (67 u/ml), and IgA tissue transglutaminase antibody (186 u/mL) was present in the serum. Gastric and duodenal biopsy documented chronic gastritis associated with *Helicobacter pylori* and chronic duodenitis, but not celiac disease-related pathological findings. Gluten-free diet (GFD) was started and her GI symptoms subsided, so diagnosis of celiac disease was made.

In her past history, about 18 years ago, her left-eye became ophthalmoplegic and without a definite

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diagnosis, after corticosteroid therapy, it was recovered completely within 1 month. Five years ago, she suffered from diplopia that was binocular and continued until now. Few months before, she became ataxic. She thought that it has been related to diet. Although, it evolved and established completely within a few days, so she met a neurologist and her brain MRI showed demyelinating plaques compatible with multiple sclerosis [Figure 1], without any structural abnormality in cerebellum [Figure 2]. Her visual evoked potentials (VEP) were abnormal too (delayed responses with normal wave configuration). Oligoclonal band was seen in CSF examination. Therefore, the clinical diagnosis of MS was made and Avonex (Beta interferon 1-a, IM, weekly) was introduced to her. Till now, she has not had any relapse, and her neurological disability (EDSS) is fixed.

Now, she has episodic ataxia and slurred speech especially whenever she is tired. On neurological examination, she has binocular diplopia and fine horizontal nystagmus when she looks to the left side. Her plantar reflexes are upward.

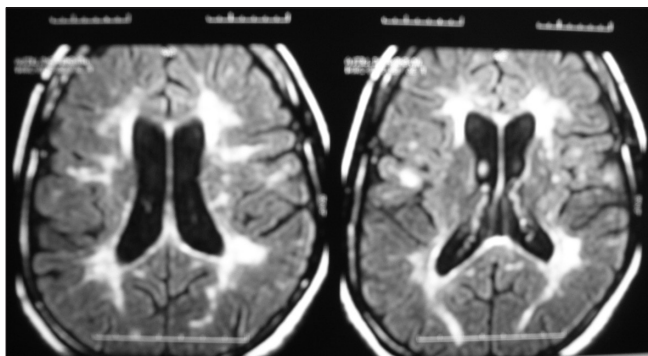


Figure 1: FLAIR-weighted MR imaging shows demyelinating plaques compatible with multiple sclerosis

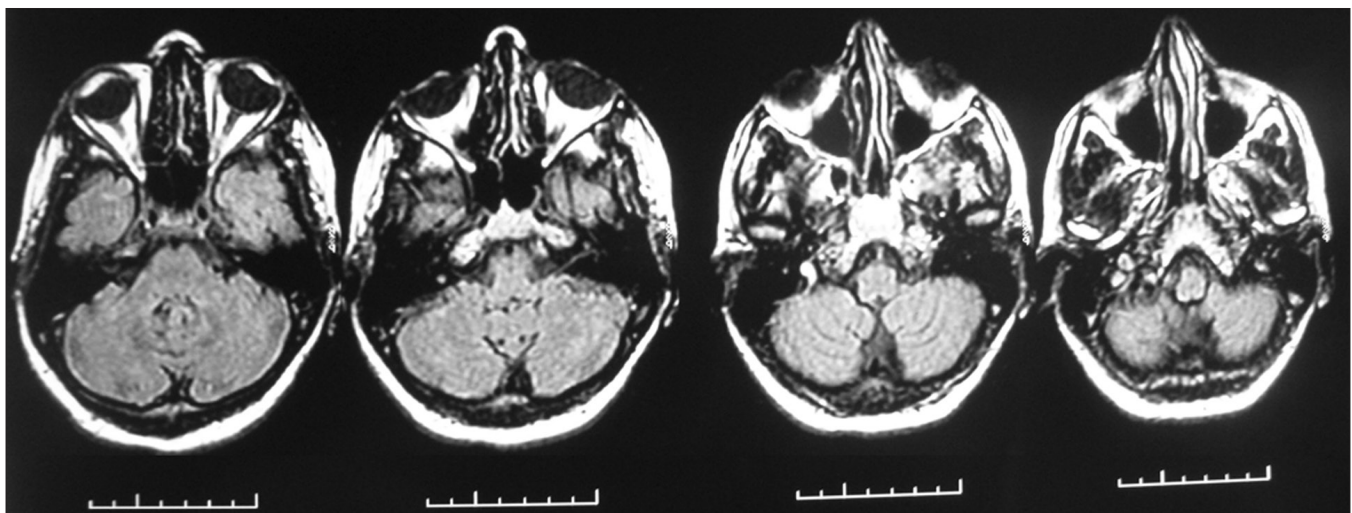


Figure 2: FLAIR-weighted MR imaging shows normal cerebellum

Abdominal reflexes are not detectable and her deep tendon reflexes are symmetric and exaggerated (3+). Her gait is ataxic and she falls when turns rapidly. Other neurological examinations are not remarkable.

DISCUSSION

MS is usually considered as a T cell-mediated inflammatory disease of the CNS, although B cell-mediated inflammation is also responsible in the development of the disease.^[5] Also, CD is considered a T-cell – mediated autoimmune diseases and the involvement of the Th₁ in its pathogenesis has been reported.^[6-8]

In spite of these similarities in pathogenesis between two disease, there was not any data that shows an increased frequency of celiac disease among patients with MS,^[5] and vice versa.

In the medical literature, there are few reports from some patients with MS-like disease together with gluten sensitivity accompanied by brain and spinal-cord white matter lesions on MRI that are indistinguishable from those seen in MS.^[9] Stefan Beyenburg *et al.* described a case of progressive leukoencephalopathy developed in a patient with adult celiac disease.^[10] In this case, brain MRI showed marked confluent white matter abnormalities, with progressive clinical course that is entirely different from our case.

Several studies showed the lack of significant association between MS and gluten sensitive enteropathy^[9-11] and jejunal biopsy specimen results in MS patients have been found to be normal.^[11] Previous researchers have investigated the role of a GFD in the treatment of MS and found no benefits.^[12]

The relapsing-remitting natural history of MS can make interpretations very difficult. So MS and CD are an uncommon association and their association is unclear.

So, we conclude that the concomitant presence of MS with atypical clinical course, and CD, likely shows an unusual chance association in our patient, but immune-mediated damage of the CNS triggered by gluten could not be excluded.

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