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Neurological Disorders Revealing Celiac Disease

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Abstract **Case Report**

Background: Celiac disease is a chronic autoimmune enteropathy triggered by gluten exposure in genetically predisposed individuals. Although gastrointestinal manifestations are classically predominant, atypical and extraintestinal presentations are increasingly recognized. Neurological and psychiatric manifestations may occasionally represent the sole presenting features, leading to diagnostic delays. ***Case Presentation:*** We report the case of a 56-year-old man with no significant medical history who presented with progressive neuropsychiatric symptoms over a four-month period, including behavioral changes, aggressiveness, psychomotor agitation, incoherent speech, and memory impairment. Physical examination revealed no focal neurological deficits or gastrointestinal symptoms. Brain magnetic resonance imaging showed diffuse cortico-subcortical atrophy, while cerebrospinal fluid analysis and electroencephalography were unremarkable. Laboratory investigations revealed iron-deficiency anemia suggestive of malabsorption. Serological testing demonstrated strongly positive anti-tissue transglutaminase IgA antibodies. Upper gastrointestinal endoscopy showed a pseudonodular duodenal mucosa, and histopathological examination of duodenal biopsies revealed total villous atrophy classified as Marsh 3c. A diagnosis of celiac disease was established. The patient was started on a strict gluten-free diet with nutritional and neurological follow-up, resulting in favorable clinical improvement. ***Conclusion:*** This case highlights an unusual presentation of celiac disease revealed exclusively by neuropsychiatric manifestations in the absence of digestive symptoms. It underscores the importance of considering celiac disease in the differential diagnosis of unexplained behavioral and cognitive disorders, particularly when associated with iron-deficiency anemia or other signs suggestive of malabsorption. Early diagnosis and treatment may improve clinical outcomes and prevent long-term neurological complications.

Keywords: Celiac disease; Neuropsychiatric manifestations; Behavioral disorders; Iron-deficiency anemia; Gluten-free diet; Malabsorption.

INTRODUCTION

Celiac disease is a chronic autoimmune enteropathy induced by gluten exposure in genetically predisposed individuals [1,2]. Although the classical presentation is characterized by chronic diarrhea and malabsorption syndrome, atypical, silent, or extraintestinal forms are increasingly recognized [2,3]. Neurological and psychiatric manifestations, such as ataxia, peripheral neuropathy, as well as mood and behavioral disorders, may constitute the sole presenting feature of the disease [4,5].

CASE PRESENTATION

Mr. M.E., a 56-year-old man with no significant past medical history, was referred to the Neurology Department for progressive neuropsychiatric symptoms evolving over a four-month period. The clinical presentation was dominated by behavioral and personality changes, characterized by unusual aggressiveness, psychomotor agitation, incoherent speech, and marked memory impairment with recurrent forgetfulness. These manifestations occurred in the absence of fever and with preserved general health status, without focal neurological signs or gastrointestinal symptoms.

Brain magnetic resonance imaging [MRI] revealed diffuse cortico-subcortical atrophy without any other significant abnormalities. Cerebrospinal fluid analysis obtained by lumbar puncture was unremarkable. Electroencephalography showed a normal interictal

tracing. HIV and syphilis serologies were negative. Laboratory investigations revealed microcytic hypochromic anemia with a low ferritin level of 14 µg/L, suggestive of a malabsorption syndrome.

Celiac serology demonstrated strongly positive anti-tissue transglutaminase IgA antibodies at 169 U/mL. Upper gastrointestinal endoscopy revealed erythematous atrophic pangastritis and a pseudonodular appearance of the duodenal mucosa. Histopathological examination of duodenal biopsies demonstrated total villous atrophy classified as Marsh 3c, associated with intraepithelial lymphocytosis estimated at 35%.

The diagnosis of celiac disease was established based on clinical, biological, serological, and histological findings. A strict gluten-free diet was initiated, together with close nutritional and neurological follow-up, resulting in a favorable clinical outcome.

DISCUSSION

Celiac disease is a chronic autoimmune enteropathy triggered by gluten exposure in genetically predisposed individuals carrying HLA-DQ2 and/or HLA-DQ8 haplotypes [1,2]. Although gastrointestinal manifestations remain common, atypical and extraintestinal forms account for a substantial proportion of clinical presentations in adults [2,3]. These extraintestinal manifestations include hepatic, dermatological, endocrine, neurological, and psychiatric disorders, which may occasionally represent the initial presentation, as observed in our patient [3,4].

Neurological manifestations associated with celiac disease are well documented and include gluten ataxia, peripheral neuropathy, epileptic seizures, chronic headaches, and cognitive impairment [4–6]. From a psychiatric perspective, patients may present with depression, anxiety, attention disorders, irritability, and, more rarely, behavioral disturbances and confusional states [7].

In a systematic review and meta-analysis published in 2021, Clappison *et al.,* demonstrated that depressive and anxiety disorders were significantly more frequent among patients with celiac disease compared with the general population [7].

Mild cognitive impairment, “brain fog,” working memory deficits, and attention disorders have also been reported in patients with celiac disease, particularly when the condition remains undiagnosed or inadequately controlled [4,5]. Hadjivassiliou *et al.,* suggested a direct autoimmune involvement of the central nervous system mediated by antibodies directed against transglutaminase 6 [TG6], a cerebral isoform of the target enzyme implicated in celiac disease [5,8].

Several studies have shown that neurocognitive manifestations may occur even in the absence of gastrointestinal symptoms and tend to improve following the initiation of a strict gluten-free diet [4,5,9].

From a behavioral perspective, cases of confusional syndromes, psychotic disorders, and rapidly progressive dementia associated with celiac disease have been reported in the literature [10]. Consequently, many authors recommend including celiac disease in the differential diagnosis of atypical neuropsychiatric disorders, particularly when accompanied by nutritional deficiencies or suggestive biological abnormalities [4,10].

In our case, the isolated behavioral presentation, without gastrointestinal symptoms or focal neurological deficits, together with iron-deficiency anemia, prompted investigations for a malabsorption-related etiology. The diagnosis was confirmed by positive anti-tissue transglutaminase IgA antibodies and duodenal histology demonstrating total villous atrophy classified as Marsh 3c [1,2].

Strict adherence to a gluten-free diet remains the cornerstone of treatment for celiac disease [1,2]. Several studies have reported improvement in neurological and psychiatric manifestations following the introduction of a gluten-free diet and correction of associated nutritional deficiencies [5,11].

CONCLUSION

This case illustrates an atypical presentation of celiac disease revealed by isolated behavioral disturbances in the absence of any gastrointestinal manifestations. It highlights the importance of maintaining a broad clinical approach when evaluating unexplained psychiatric symptoms and underscores the need to consider an underlying organic etiology, particularly in the presence of suggestive biological findings such as iron-deficiency anemia.

The increasing recognition of extraintestinal manifestations of celiac disease requires heightened awareness among neurologists, psychiatrists, and internists. The favorable clinical response observed following the implementation of a gluten-free diet further emphasizes the value of early diagnosis, which may help prevent the progression of potentially irreversible neurological damage. In a context where the gut–brain axis is receiving growing scientific attention; this observation raises an important question: how many so-called “functional” neuropsychiatric disorders may actually conceal an underlying silent celiac disease?

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