

DDSIG New and Noteworthy

APTA Neurology Section

8/10/2017

Volume 2 Edition 6

New

Title [The neurologic significance of celiac disease biomarkers.](#)

McKeon, Lennon, Pittock, Kryzer, and Murray. Neurology. 2014 Nov 11; 83(20): 1789–1796.

INTRODUCTION: The authors report neurologic phenotypes and their etiologies determined among 68 patients with either (1) celiac disease (CD) or (2) no CD, but gliadin antibody positivity.

METHODS: Neurologic patients included both those with the CD-prerequisite major histocompatibility complex class II human leukocyte antigen (HLA)-DQ2/DQ8 haplotype, and those without. The 3 groups were as follows: group 1 (n = 44), CD or transglutaminase (Tg)-2/deamidated gliadin immunoglobulin (Ig)A/IgG detected; group 2 (n = 15), HLA-DQ2/DQ8 noncarriers, and gliadin IgA/IgG detected; and group 3 (n = 9), HLA-DQ2/DQ8 carriers, and gliadin IgA/IgG detected. Neurologic patients and 21 nonneurologic CD patients were evaluated for neural and Tg6 antibodies.

RESULTS: In group 1, 42 of 44 patients had CD. Neurologic phenotypes and causes vitamin were diverse. In groups 2 and 3, 21 of 24 patients had cerebellar ataxia; none had CD. Causes of neurologic disorders in groups 2 and 3 were diverse. One or more neural-reactive autoantibodies were detected in 10 of 68 patients, all with autoimmune neurologic diagnoses. Tg6-IgA/IgG was detected in 7 of 68 patients (cerebellar ataxia, 3; myelopathy, 2; ataxia and parkinsonism, 1; neuropathy, 1); the 2 patients with myelopathy had neurologic disorders explained by malabsorption of copper, vitamin E, and folate rather than by neurologic autoimmunity.

CONCLUSION: Our data support causes alternative to gluten exposure for neurologic dysfunction among most gliadin antibody-positive patients without CD. Nutritional deficiency and coexisting autoimmunity may cause neurologic dysfunction in CD.

Noteworthy

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Link to article:

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4240435/>

CONCLUSIONS and CLINICAL IMPLICATIONS: For neurologic patients with positive CD serologies, CD should be confirmed histologically before attributing neurologic disorders to gluten exposure. Alternative etiologies for neurologic symptoms need to be considered.