

HEADACHE, TINGLING NUMBNESS AND JUXTACORTICAL T2 HYPERINTENSE LESIONS: ATYPICAL CASE OF CELIAC DISEASE

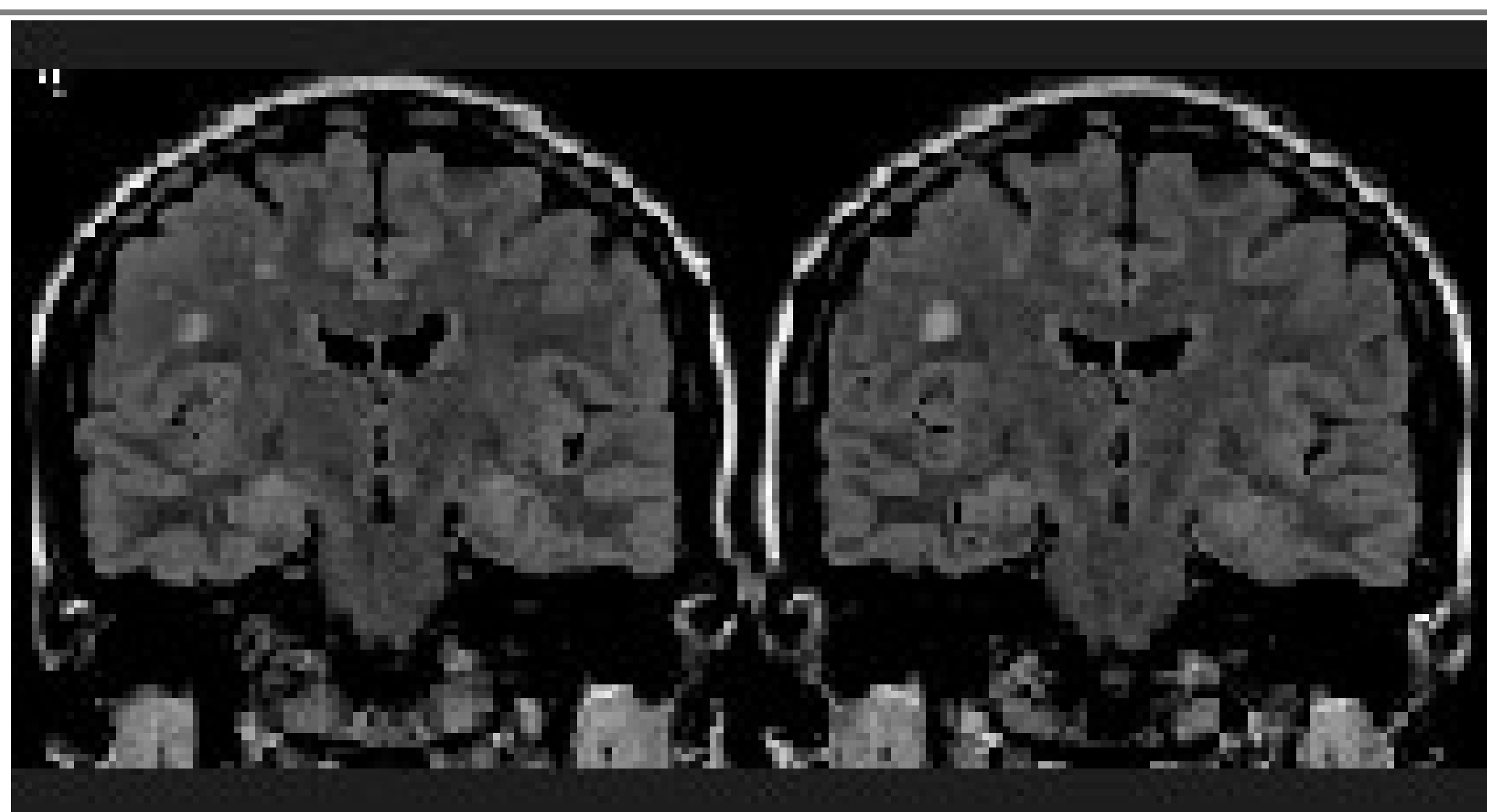
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INTRODUCTION: Celiac disease (CD) is an autoimmune bowel disease triggered by gliadine proteins¹, that can have systemic involvement with several neurological manifestations².

OBJECTIVE: The aim of the current work is to report an unusual neurological presentation of CD



DISCUSSION and CONCLUSION: The neurological symptoms more frequently described in relation to CD are ataxia, epilepsy, cognitive disorders, dementia, tremor, myelopathy, neuropathy, brainstem encephalitis, progressive leukoencephalopathy, occipital calcification, anxiety/depression, and myoclonic syndrome^{2,3}; for the remarkable migraine frequency in the population, the association with CD is still discussed. Focal brain white-matter lesions may be a rare extraintestinal manifestation of CD, they may be ischemic in origin as a result of a vasculitis or caused by inflammatory demyelination. This case report shows how poorly treatment responsive migraine associated with paresthesia and with brain T2 hyperintense lesions. should suggest further investigation including in the diagnostic panel a screening for CD.

CASE REPORT: A 27 years-old woman, affected by episodic headaches, since she was 8 years old, with unilateral location, pulsating quality, moderate or severe intensity, aggravated by routine physical activity, poorly responsive to non steroid antiinflammatory therapy, complained sporadic tingling numbness, not headaches related, located in the upper limbs and lasting about 24 hours. For these symptoms, in December 2015, her primary care physician prescribed a brain and spinal cord magnetic resonance imaging (MRI) with and without contrast enhancement that showed T2 hyperintense juxtacortical millimetre lesions, in frontoparietal regions bilaterally, without contrast enhancement. In January 2016 she was admitted to our Department and practiced a broad-spectrum screening: neurological examination, cell blood count (CBC), biochemical profile, coagulation profile, gammaglobulin immunofixation, thyroid hormones and autoantibodies, autoimmunity screening (ANA, ENA, ANCA, antiphospholipid antibodies in the suspicion of vasculitic disease), lumbar puncture (in the suspicion of demyelinating disease), transcranial Doppler with contrast agent (in the suspicion of patent foramen ovale). Neurological exam was negative. The only pathological findings were: iron deficiency anemia, high serum IgA (975 mg/dL), cerebrospinal fluid with oligoclonal band type II. In view of iron deficiency anemia and high serum IgA we investigated for CD and found positive IgA tissue transglutaminase (11.7U/mL) and IgA endomysial antibodies. Gastroscopy confirmed the diagnosis of CD. Headaches mitigated after three months of gluten-free diet and; in May 2016, MRI showed no new lesions.

1) R. John Presutti et al Celiac Disease. American Family Physician December 15, 2007 Volume 76, Number 12

2) Nikpour S et al. Neurological manifestations, diagnosis, and treatment of celiac disease: A comprehensive review Iran J Neurol. 2012

3) Hernandez-Lahoz C Mauri-CapdevilaG, Vega-Villar J, Rodrigo L. Neurological disorders associated with gluten sensitivity. Rev Neurol. 2011 Sep 1;53 (5):287-300.