HSPI The Open Access Publisher

JNND JOURNAL OF NEUROSCIENCE AND NEUROLOGICAL DISORDERS
ISSN 2639-3220

Letter to Editor

Autoimmune Diseases Associated with Iron Deficiency Anemia; Coexistence of Demyelinating Disease and Inflammatory Bowel Disease

Hatice Hamarat[1]*, Berrin Yalinbaş Kaya[2] and Özlem Şahin[3]

[1]Internal Medicine Department, Eskişehir City Hospital, Eskişehir, Turkey
[2]Gastroenterology Department, Eskişehir City Hospital, Eskişehir, Turkey
[3]Neurology Department, Eskişehir City Hospital, Eskişehir, Turkey

More Information

***Address for correspondence:**
Hatice Hamarat, İnternal Medicine Department, Eskişehir City Hospital, Eskişehir, Turkey,
Email: hklncal@hotmail.com

Submitted: January 06, 2024
Approved: January 17, 2024
Published: January 18, 2024

How to cite this article: Hamarat H, Kaya BY, Şahin Ö. Autoimmune Diseases Associated with Iron Deficiency Anemia; Coexistence of Demyelinating Disease and Inflammatory Bowel Disease. J Neurosci Neurol Disord. 2024; 8: 008-009.

DOI: 10.29328/journal.jnnd.1001089

https://orcid.org/0000-0001-8694-5686

In a young patient with iron deficiency anemia and low hemoglobin levels, we identified a very rare association: Demyelinating disease with Inflammatory Bowel Disease (IBD). A 20-year-old female patient presented to the emergency department with weakness, headache, sudden onset of forgetfulness, and impaired speech. She had no known medical history, alcohol or drug abuse, or family history of coagulation disorder, blood diseases, or stroke.

Upon examination, the patient was conscious, alert, and oriented, but appeared distracted and unable to focus. All other neurological examinations were normal. The abdominal examination revealed tenderness in the pelvic region but no abdominal defense.

During the psychiatric examination, the patient exhibited limited affect, slow speech, and amnestic findings when recalling recent events. The cardiac examination was normal. The ophthalmologic examination revealed mild swelling and hyperemia in the nasal portion of the right optic disc.

During the patient's hospitalization, vital signs remained stable and there was no fever. Laboratory analysis revealed a hemoglobin level of 5.4 g/dL, a hematocrit level of 23.3%, an MCH of 13.9 pg, and an MCV of 59.7 fL. Direct Coombs, LDH, and Haptoglobin levels were normal. Ferritin levels were at 3 ug/L and Vitamin B12 levels were at 236 ng/L. Biochemical parameters were normal. Coagulation tests, acute phase reactants, and hepatitis and HIV markers were negative. No pathological cells were detected in the peripheral smear, which was consistent with iron deficiency anemia. CMV, EBV, and COVID PCR tests were also negative. Additionally, suspected diffusion restriction was observed in the basal ganglia.

The patient's drug tests in urine were negative. Brain Computed Tomography (CT) was normal. Diffusion-weighted Magnetic Resonance Imaging (MRI) revealed multiple ischemic foci measuring up to 1cm in size in the frontoparietal lobes at the level of bilateral centrum semiovale. In the contrast-enhanced brain MRI, some T2A hyperintense signals were observed perpendicular to the corpus callosum in the periventricular white matter. Mild contrast enhancement was observed around the lesions in the left and right frontal lobes on the contrast series. The possibility of multiple sclerosis was considered. The imaging findings were consistent with Multiple Sclerosis (MS), but we were unable to confirm the diagnosis of MS in our hospital by immunochemical analysis of patient plasma for common pro- and anti-inflammatory factors (INF-β, IL-4, etc.) and specific antibodies (IgG1) (against MBP or MOG protein antigens) in patient plasma obtained during the acute phase of the disease. The abdominal CT scan of the patient with distension revealed diffuse thickening of the colon and transverse colon walls. The pathology was consistent with ulcerative colitis. Upper gastrointestinal endoscopy was performed to investigate the cause of anemia, which revealed minimal antral hyperemia. Colonoscopy showed widespread deep ulcers in the cecum, ascending colon, and transverse colon, as well as severe hyperemia and edema. Additionally, widespread post-inflammatory polyps were found in the ascending colon and cecum.

The patient was administered intravenous iron therapy and erythrocyte suspension. As the hemoglobin level

increased, the patient's consciousness began to improve and their attention span increased. Although lesions were still present in the cerebral MRI taken 3 months later, there was no neurological pathology found during the physical examination. The pathophysiology of neurological diseases caused by anemia is interesting [1,2].

Endothelial damage occurs in cases of anemia and secondary thrombocytosis occurs, increasing the risk of developing ischemic stroke due to anemia. Endothelial dysfunction itself leads to more inflammation and in an anemic hypoxic environment, especially in the watershed area of the brain, it leads to ischemic brain tissue damage. Many studies have reported cases of ischemic stroke in young patients with anemia [2-6]. Epidemiological studies suggest a relationship between Inflammatory Bowel Disease (IBD) and Multiple Sclerosis (MS) [7,8]. This has been particularly attributed to the use of anti-tumor necrosis factor alpha (anti-TNFa) agents for IBD treatment. However, the number of studies on this topic is insufficient. We could not perform sufficient laboratory analysis to diagnose MS in our patient, but we followed the patient for the presence of a neurological demyelinating disease caused by IBD-related anemia.

It is important to note that after treatment of the anemia, the patient's neurological symptoms may disappear, but the radiological imaging may remain unchanged. Inflammatory bowel diseases such as ulcerative colitis may be accompanied or coexist with demyelinating diseases. Endothelial damage and hypoxia due to iron deficiency can especially trigger existing demyelinating diseases. It is important to screen young IBD patients for demyelinating diseases to prevent permanent disability.

References

1. Mehta PJ, Chapman S, Jayam-Trouth A, Kurukumbi M. Acute ischemic stroke secondary to iron deficiency anemia: a case report. Case Rep Neurol Med. 2012; 2012:487080. doi: 10.1155/2012/487080. Epub 2012 May 9. PMID: 22937352; PMCID: PMC3420649.

2. Kaiafa G, Savopoulos C, Kanellos I, Mylonas KS, Tsikalakis G, Tegos T, Kakaletsis N, Hatzitolios AI. Anemia and stroke: Where do we stand? Acta Neurol Scand. 2017 Jun;135(6):596-602. doi: 10.1111/ane.12657. Epub 2016 Aug 1. PMID: 27480069.

3. Boehme AK, Esenwa C, Elkind MS. Stroke Risk Factors, Genetics, and Prevention. Circ Res. 2017 Feb 3;120(3):472-495. doi: 10.1161/CIRCRESAHA.116.308398. PMID: 28154098; PMCID: PMC5321635.

4. Hartfield DS, Lowry NJ, Keene DL, Yager JY. Iron deficiency: a cause of stroke in infants and children. Pediatr Neurol. 1997 Jan;16(1):50-3. doi: 10.1016/s0887-8994(96)00290-1. PMID: 9044402.

5. Azab SF, Abdelsalam SM, Saleh SH, Elbehedy RM, Lotfy SM, Esh AM, Srea MA, Aziz KA. Iron deficiency anemia as a risk factor for cerebrovascular events in early childhood: a case-control study. Ann Hematol. 2014 Apr;93(4):571-6. doi: 10.1007/s00277-013-1922-y. Epub 2013 Oct 19. PMID: 24141332.

6. Kandinata NN, Breehl L, Chhetri B, Paudel S. Stroke Secondary to Iron Deficiency Anemia: A Case Report. Cureus. 2021 Nov 13;13(11):e19526. doi: 10.7759/cureus.19526. PMID: 34804746; PMCID: PMC8592313.

7. Bernstein CN, Wajda A, Blanchard JF. The clustering of other chronic inflammatory diseases in inflammatory bowel disease: a population-based study. Gastroenterology. 2005 Sep;129(3):827-36. doi: 10.1053/j.gastro.2005.06.021. PMID: 16143122.

8. Kimura K, Hunter SF, Thollander MS, Loftus EV Jr, Melton LJ 3rd, O'Brien PC, Rodriguez M, Phillips SF. Concurrence of inflammatory bowel disease and multiple sclerosis. Mayo Clin Proc. 2000 Aug;75(8):802-6. doi: 10.4065/75.8.802. PMID: 10943233.