(S60) SEVERE ANEMIA ASSOCIATED WITH NATALIZUMAB THERAPY: A REPORT OF TWO CASES

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Background: Natalizumab is a humanized monoclonal antibody to the VLA-4 antigen for treatment of relapsing-remitting multiple sclerosis (MS) and Crohn's disease. Overall, natalizumab is well tolerated, with known side effects including infusion reactions, hepatic transaminase elevations, antibody formation, and progressive multifocal leukoencephalopathy. Severe anemia has not previously been reported in association with natalizumab therapy. Objectives: To describe two cases of severe anemia during treatment with natalizumab in MS patients. Methods: Descriptive case reports and discussion based on review of the literature. Results: Two female patients with relapsing-remitting MS were referred to our center after development of severe anemia requiring packed red blood cell transfusions while on natalizumab. MS disease duration was 18 years (age 50) and 7 years (age 45). Prior disease-modifying treatments included interferon beta-1a, interferon beta-1b, and glatiramer acetate, without exposure to myelosuppressive agents. Numbers of monthly natalizumab infusions were 30 and 13 prior to onset of symptoms. The patients presented with shortness of breath and severe fatigue within 3 days of the last infusion. The hemoglobin nadir was 7 and 4.1 (g/dL). One patient stabilized after a single transfusion without further treatment. The other underwent bone marrow biopsy, which revealed normal cellularity, focal lymphoid aggregates, and a shift toward immaturity in erythroid lineage consistent with regeneration after a toxic event. She required repeated transfusions and was treated with oral prednisone (60 mg/day) and stabilized after 3 months. Other etiologies for anemia including infection, gastrointestinal bleeding, iron deficiency, and autoimmune hemolysis were ruled out. Natalizumab antibodies were absent. Natalizumab therapy was discontinued in both patients, with subsequent normalization of hemoglobin. Conclusions: Severe anemia has not previously been reported in association with natalizumab treatment and does not have a higher incidence in MS patients. The currently known mechanism of action of natalizumab does not explain the development of severe anemia in these two patients, although a rare, transient idiosyncratic reaction is possible. Further cases of anemia while on natalizumab therapy should be reported to improve medical knowledge.

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