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Case Report: Parvovirus B19 Associated Large-vessel Vasculitis in an Immunocompromised Patient.

Large vessel vasculitis (LVV) cause inflammation of the walls of large arteries. Autoimmune mechanisms typically underlie LVV, but infectious triggers are increasingly recognized, particularly in immunocompromised patients. We present a rare case of LVV in a patient with relapsing-remitting multiple sclerosis (RRMS) treated with cladribine, a lymphocyte-depleting agent, and explore the potential role of Parvovirus B19 (B19V) in LVV pathogenesis.

A literature review was conducted on vasculopathies in immunocompromised patients, the immunological effects of cladribine, and the role of B19V in LVV.

A 64-year-old female with RRMS consulted due to acute cervical pain, submandibular lymphadenopathy, and severe headache nine days after completing her first cladribine cycle. Imaging revealed circumferential thickening of the brachiocephalic artery and proximal large vessels, consistent with LVV. Laboratory investigations showed lymphopenia, elevated inflammatory markers, and B19V IgM positivity. Extensive infectious and autoimmune workups were negative, except for significantly elevated procalcitonin levels. Given the patient's lymphopenia and imaging findings, an infectious vasculitis was suspected. Empirical antibiotics led to rapid clinical, biochemical, and radiological improvement, supporting an infectious etiology.

This case met the ACR/EULAR 2022 criteria for extracranial giant-cell arteritis (GCA), with imaging demonstrating characteristic concentric arterial involvement. While primary LVV was considered, particularly in the context of lymphopenia resembling advanced HIV or a cladribine-induced autoimmune reaction, the acutely positive B19V IgM and rapid antibiotic response suggested an infectious etiology. Cladribine-induced lymphocyte depletion likely increased susceptibility to viral or bacterial vasculopathy. B19V, known to be associated with polyarteritis nodosa and Kawasaki disease, may contribute to LVV through molecular mimicry, immune complex deposition, or direct endothelial infection, bypassing adaptive immune mechanisms suppressed by cladribine. The elevated procalcitonin and prompt improvement with antibiotics further supported an infectious process, although no bacterial pathogen was definitively identified.

This case highlights a rare presentation of LVV in an immunosuppressed patient, emphasizing the need to consider infectious triggers in vasculitis differentials. This case also raises awareness of the potential contributions of B19V to LVV pathogenesis through non-adaptive immune pathways. Further studies are needed to better understand the mechanisms of infectious vasculitides in immunosuppressed populations.