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Aplastic anemia in a patient with autoimmune Hepatitis

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INTRODUCTION/OBJECTIVES: Aplastic anemia is a rare stem cell disorder in which the bone marrow stops making cells, leading to pancytopenia. It can be inherited, but most cases are autoimmune, triggered by drugs, viruses, or irradiation

CASE PRESENTATION: A 47-year old woman was diagnosed with autoimmune hepatitis in 2019 after suffering from subacute hepatic failure. TPMT enzyme activity was normal. She was put on corticosteroid therapy followed by azathioprine 200 mg. The patient initially had a good response to therapy with normalization of transaminase levels. During the administration of prescribed therapy, pancytopenia was found. Azathioprine therapy was discontinued and the patient was put on dexamethasone monotherapy. The blood cell count didn't improve on corticosteroid therapy. Due to comorbidities, the patient was not a candidate for splenectomy so eltrombopag was introduced into therapy. Bone marrow biopsy revealed aplastic anemia in development caused by azathioprine and secondary immune mechanisms following autoimmune hepatitis. The introduction of cyclosporine instead of azathioprine combined with corticosteroids and eltrombopag resulted in an initial positive hematopoietic response. After the initial positive outcome of the combined therapy, the patient's thrombocytopenia worsened so anti-thymocyte globulins were included in therapy. Symptomatic thrombocytopenia, such as menometrorrhagia, was managed with platelet transfusions. During the hospitalization, HLA typing was done for bone marrow transplant planning.

CONCLUSION: Azathioprine-induced aplastic anemia is not so common but it is a serious complication. Treatment with the immune system – suppressing therapy or a bone marrow transplant is necessary for patients with severe aplastic anemia.

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Atypical Raynaud syndrome and skin changes caused by late stage of Lyme disease with cryoglobulinemia Paola Negovetić^a, Ena Parać^a, Tin Šklebar^b, Ljiljana Smilianić Tomičević^{a,c}

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Keywords: Atypical Raynaud Syndrome, Cryoglobulinemia, Lyme disease

INTRODUCTION/OBJECTIVES: Raynaud's syndrome is a disease in which the peripheral blood vessels contract, reducing the blood supply to the affected region. It manifests with coldness, colour changes, and a sensation in the affected digits. Cryoglobulinemia is a presence of cryoglobulins in the serum that cluster together during cold temperatures limiting blood flow and generating damage to muscles, skin, organs, and nerves.

CASE PRESENTATION: A male patient reports changes in the skin color of his right hand, which turned purple when exposed to cold temperatures. On examination, the digits were thickened, the skin in the right hand, was livid, warm, and without clinical symptoms of Raynaud's syndrome. Clinically, acrodermatitis chronica atrophicans was suspected. The patient's serology was positive for Borrelia burgdorferi and histopathology of skin changes points to the chronic acrodermatitis found in the late stages of Lyme disease. The patient was diagnosed with Raynaud's syndrome by computerized color telethermography findings. In patient's serum, cryoglobulins type 3 were found. The infection probably caused mixed cryoglobulinemia and consequently atypical Raynaud's syndrome in this patient.

CONCLUSION: Secondary Raynaud's syndrome can be caused by cryoglobulinemia due to an infection with Borrelia burgdorferi and, with typical skin changes, appears at a late stage of Lyme disease. The patients infected with Borrelia often do not know that they have been exposed to it, and the disease can sometimes come to late stages until it is detected. Hence, a thorough history must be taken, and if there are any doubts, the doctor conducts an extensive examination.