malignancies, KS has also been reported in patients receiving Janus kinase (JAK) inhibitors. We present a case of HHV-8-positive cutaneous Kaposi sarcoma developing in a patient with primary myelofibrosis under ruxolitinib treatment. **Methods:** A 72-year-old female with a 2-year history of intermediate-2 risk primary myelofibrosis, positive for the JAK2 V617F mutation, was being followed in our hematology department. She had been on ruxolitinib (2×10 mg/day) for symptom control, which provided initial improvement in systemic complaints and splenomegaly. However, after 14 months of treatment, she developed painless violaceous plaques and nodules on her lower extremities, raising suspicion for Kaposi sarcoma. Dermatologic examination confirmed the presence of multiple dark purple nodules predominantly on the left lower leg. A punch biopsy was performed, and histopathological examination revealed spindle-cell proliferation consistent with Kaposi sarcoma. Immunohistochemical staining was strongly positive for HHV-8. **Results:** Laboratory evaluation revealed hemoglobin of 9.2 g/dL, white blood cell count of 13,000/mm^3, and platelet count of 120,000/mm^3. Peripheral smear showed typical findings of myelofibrosis, including teardrop-shaped erythrocytes. HIV, HBV, and HCV tests were all negative. Abdominal ultrasonography confirmed stable splenomegaly (19 cm). The ruxolitinib treatment was discontinued, and hydroxyurea was initiated as an alternative. Given that the Kaposi lesions were localized and the patient remained asymptomatic, systemic chemotherapy was not started. The patient is being followed with close dermatological and hematological monitoring. **Discussion:** This case highlights a rare but clinically significant complication of ruxolitinib therapy in a patient with primary myelofibrosis. JAK inhibition may lead to immune dysregulation, impaired antiviral T-cell responses, and viral reactivation—particularly HHV-8 in susceptible individuals. Although KS is commonly associated with HIV, this patient had no underlying immunodeficiency other than the JAK inhibitor–mediated suppression. The temporal relationship between ruxolitinib exposure and KS onset, combined with HHV-8 positivity and regression of symptoms after discontinuation of the drug, supports a probable causal association. Clinicians should remain vigilant for unusual infections or neoplasms in patients undergoing JAK inhibitor therapy.

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OP 7

Sequential Autoimmune Hematological Manifestations: From Isolated Lupus Anticoagulant to Post-COVID-19 Autoimmune Hemolytic Anemia

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Introduction: Autoimmune hematological disorders can present with variable phenotypes over time, suggesting underlying B-cell dysregulation. We report a unique case of sequential, distinct autoimmune manifestations occurring five years apart in the same patient, highlighting the heterogeneous nature of autoimmune hematological conditions and their potential triggers. **Case Presentation:** A middle-aged woman with no history of systemic autoimmune disease presented in 2019 with incidentally discovered coagulopathy. Routine laboratory evaluation revealed an INR >3 without bleeding symptoms. Further workup showed prolonged PT with normal aPTT, normal liver function, and normal levels of factors VIII, IX, and XI. Lupus anticoagulant testing was positive, while anticardiolipin and β2-glycoprotein I antibodies were negative. The patient did not meet criteria for systemic lupus erythematosus or antiphospholipid syndrome. Without specific treatment, the coagulation abnormalities spontaneously resolved within three months. Five years later, in 2024, the patient developed fatigue, jaundice, and anemia two weeks after COVID-19 infection. Laboratory findings revealed: markedly decreased hemoglobin, elevated LDH, suppressed haptoglobin, and elevated indirect bilirubin. Direct antiglobulin test (DAT) was strongly positive for IgG. Interestingly, lupus anticoagulant and other antiphospholipid antibodies were negative at this presentation. Bone marrow aspiration showed normocellular marrow with erythroid hyperplasia, excluding malignancy or dysplasia. The diagnosis of COVID-19-associated autoimmune hemolytic anemia (AIHA) was established. Treatment with rituximab 375 mg/m^2 weekly for four doses was initiated. The patient demonstrated dramatic response within two weeks, with rapid normalization of hemoglobin, LDH, and bilirubin levels. The DAT became negative, and the patient remains in remission on regular follow-up. **Discussion:** This case illustrates the dynamic nature of autoimmune hematological disorders. The initial presentation of isolated lupus anticoagulant positivity with spontaneous resolution, followed years later by post-viral AIHA, suggests an underlying predisposition to B-cell mediated autoimmunity with variable clinical expression. COVID-19 has been increasingly recognized as a trigger for autoimmune phenomena, including AIHA. The temporal relationship between COVID-19 infection and AIHA development in our patient, combined with the excellent response to B-cell depletion therapy, supports this association. The contrasting immunological profiles between episodes—positive lupus anticoagulant initially versus positive DAT with negative antiphospholipid antibodies later—demonstrates that autoimmune manifestations can evolve independently over time. This heterogeneity poses diagnostic and therapeutic challenges but also provides insights into the complexity of autoimmune regulation. **Conclusion:** Sequential development of distinct autoimmune hematological disorders in a single patient underscores the importance of comprehensive evaluation and long-term monitoring. The dramatic response to rituximab in the second episode highlights the central role of B-cell dysregulation in these conditions. This case emphasizes the need for heightened awareness of post-viral autoimmune complications and the potential for evolving autoimmune phenotypes over time.

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