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Complement-Mediated Thrombotic Microangiopathy Presenting with Dialysis-Dependent Acute Kidney Injury and Multiorgan Ischemia After Hematopoietic Stem Cell Transplantation

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Case Study : Thrombotic microangiopathy (TMA) is a serious complication following allogeneic hematopoietic stem cell transplantation (HSCT) and is frequently associated with complement dysregulation. Renal involvement may predominate and rapidly progress to dialysis-dependent acute kidney injury (AKI). We report a case of complement-mediated TMA after HSCT presenting with severe AKI and multiorgan ischemia. A 56-year-old man with prior allogeneic HSCT for myelodysplastic syndrome (MDS) presented with rapidly worsening renal function. Tacrolimus was discontinued due to suspected chronic graft-versus-host disease, and he had been on ruxolitinib for eight months. He was admitted for suspected acute pericarditis and treated with nonsteroidal anti-inflammatory drugs and colchicine. Ten days after treatment initiation, renal function rapidly deteriorated, with serum creatinine rising from 0.77 to 7.40 mg/dL. Hemoglobin declined to 7.6 g/dL, platelets to 30,000/ μ L, and lactate dehydrogenase increased to 1,800 IU/L. Peripheral smear showed numerous schistocytes (20–25 per high-power field). ADAMTS13 activity was above 40%, complement C3 was decreased, and other serologic markers for glomerulonephritis were negative. No evidence of infection or MDS relapse was identified. During evaluation, visual disturbance acutely worsened. Ophthalmologic examination revealed bilateral retinal hemorrhages and ischemic lesions, consistent with Purtscher-like retinopathy associated with systemic TMA. Plasma exchange, renal replacement therapy, and C5 inhibition with Ravulizumab were initiated. However, renal recovery was not achieved and dialysis dependence persisted. Multiorgan complications subsequently developed, including ischemic ileocolitis, pancreatic necrosis, hepatic ischemic changes, and acute left frontal cerebral infarction, followed by secondary pneumonia. The patient ultimately died. Complement-mediated TMA following HSCT can present with rapidly progressive, dialysis-dependent AKI and evolve into widespread ischemic organ injury despite timely multimodal therapy, including complement inhibition. This case underscores the aggressive nature of post-transplant TMA and highlights the need for early recognition and prompt intervention to mitigate irreversible kidney damage and systemic complications.