Renal Infarction in a Patient Found to Have a Dysproteinemia

Jayesh Patel, Carol J. Holman, and Mony Fraer

Case Description
A 70-year-old man with coronary artery disease, hypertension, and a history of repaired abdominal aortic aneurysm presented to the emergency room with sudden-onset abdominal pain and nausea. His blood pressure was 185/96 mm Hg with stable other vitals on presentation. Physical examination was notable for diffuse abdominal tenderness to palpation with no additional abnormalities.

Laboratory investigation revealed hypotension (with normal plasma osmolality) and an elevated creatinine of 1.43 mg/dl (normal baseline, reference range 0.5–1.1). Complete blood count revealed leukocytosis, anemia, and thrombocytopenia. With a concern for acute intraabdominal process, abdominal computed tomography with intravenous contrast revealed intraventricular thrombus on the third day of systemic anticoagulation, requiring its discontinuation. After two cycles of chemotherapy, partial remission was achieved and creatinine returned to baseline levels.

Urinalysis revealed proteinuria and hematuria. Further workup revealed elevated prothrombin time, lactate dehydrogenase, and D-dimer levels, while haptoglobin was undetectable and fibrinogen was low (suggestive of ongoing red cell lysis). Procalcitonin level was normal and factor V Leiden mutation was absent. Blood cultures were negative. Electrocardiogram revealed sinus rhythm and transthoracic echocardiogram was negative for any valvular abnormalities or intraventricular thrombus. A peripheral smear revealed rouleaux formation and schistocytes. Serum protein electrophoresis was remarkable for a monoclonal protein in the serum protein electrophoresis was remarkable for a monoclonal protein in the serum. A bone marrow biopsy revealed 20% plasma cells in the bone marrow biopsy revealed 20% plasma cells (Figure 1C). The patient was considered to have acute renal infarction due to hypercoagulable state and (possibly) disseminated intravascular coagulation secondary to untreated multiple myeloma.

Systemic anticoagulation with intravenous heparin was initiated along with chemotherapy. The hospital course was complicated by the development of a left retroperitoneal hematoma on the third day of systemic anticoagulation, requiring its discontinuation. After two cycles of chemotherapy, partial remission was achieved and creatinine returned to baseline levels.

Renal infarction nearly always occurs in the setting of risk factors for thromboembolism, with atrial fibrillation being the most common (1). Due to the often vague presentation, acute renal infarction may be misdiagnosed and thus have prognostic implications. An acute segmental renal infarction appears as wedge-shaped areas of reduced intensity on computed tomography. A complete renal artery occlusion may cause decreased attenuation throughout the renal parenchyma due to reduced perfusion with a rim of viable tissue, termed as “rim sign,” which is believed to result from intact renal collateral circulation (2). Monoclonal plasma cells lead to an increased production of cytokines (such as vascular endothelial growth factor), which cause a hypercoagulable state through increased angiogenesis and tissue factor overexpression on endothelial cells (3,4). In addition, circulating monoclonal proteins cause hyperviscosity along with impairment of platelet and coagulation function which are considered to be the key mechanisms in hemostatic abnormalities. Patients with myeloma have probably the highest venous thromboembolic risk among those with hematologic malignancies, up to 7.5 fold higher compared to the general population (4,5).

Plasma cell diseases can lead to kidney injury, although, it is uncommon to have thrombosis in the renal vasculature being the initial presentation (6). Our goal is to bring to attention this uncommon scenario which could reveal the underlying primary process and, when missed, may have major consequences for renal function and overall morbidity. Renal infarctions in the context of untreated multiple myeloma have not been described in the literature to our knowledge.

Teaching Points
- Common risk factors for renal infarction include atrial fibrillation, infective endocarditis, hypercoagulable disease, hematologic malignancies, ischemic and valvular heart disorders, and acute renal artery dissection.
- Untreated multiple myeloma can cause arterial thrombosis at unusual locations secondary to a hypercoagulable state. It should be included in the differential diagnosis of a patient with renal infarction especially if the cause is not apparent during initial evaluation.
For a patient presenting with acute onset abdominal pain in the setting of uncontrolled hypertension, a vascular event should be considered (dissection, thrombosis/infarction etc.); in the same scenario, if hematuria is present, this would point to kidney’s involvement by this potential vascular event.

Disclosures
All authors have nothing to disclose.

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Author Contributions
J. Patel conceptualized the study and wrote the original draft; M. Fraer and C. Holman reviewed and edited the manuscript.

References

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Figure 1. (A) Abdominal CT revealing wedge-shaped infarcts within the right kidney. (B) A mural thrombus in the right renal artery. (C) Bone marrow core demonstrating hypercellular bone marrow with increased plasma cells which are atypical, with prominent nucleoli. (hematoxylin and eosin stain, ×100).