Anuria and Acute Renal Failure Caused by Renal Artery Occlusion

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CASE PRESENTATION

Initial Presentation

A 66-year-old woman presented to the emergency department with a 3-day history of vomiting and diarrhea and a 1-day history of anuria. She had been in her usual state of health until 3 days prior to presentation when she began to experience diarrhea and vomiting. The patient initially did not seek treatment, as several family members had similar symptoms. After her third day of illness, the patient noted that she had not urinated during the entire day and therefore sought medical attention.

Past Medical History

The patient's past medical history was significant for hypertension and chronic ischemic renovascular disease. Five years ago, she had increasingly difficult-to-control hypertension. Further evaluation revealed anomalous renal artery supply (3 arteries supplying the left kidney and 2 partially occluded arteries supplying the right kidney) and a 3-cm abdominal aortic aneurysm. She subsequently underwent abdominal aortic aneurysm repair with left renal artery bypass. The right kidney was not bypassed due to its smaller size and difficulty in exposing its vessels. The left renal graft required angioplasty 4 years ago for stenosis. The patient also experienced a right frontoparietal cerebrovascular accident 17 years ago with no residual effects and a transient ischemic attack 4 years ago.

The patient's medications included metoprolol, simvastatin, clopidogrel, aspirin, and amitriptyline. She had no recent changes to her medication regimen. The patient had a limited history of smoking and quit 15 years prior to this admission. She had an extensive family history of vascular disease, with her mother, father, and sister having a history of hypertension and coronary artery disease and/or stroke.

Physical Examination

Physical examination was essentially unremarkable. The patient denied fever, chills, weight loss, chest pain, abdominal pain, flank pain, or hematuria, although she complained of rhinorrhea, nausea, and general body aches. Her vital signs were: temperature, 97.6°F; blood pressure, 147/83 mm Hg; heart rate, 104 bpm; and respiratory rate, 20 breaths/min. She was in no acute distress. Her lung, abdominal, and neurologic examinations were normal. Cardiac examination was normal except for mild tachycardia and a systolic ejection murmur with radiation to her right carotid artery. She had no edema.

Laboratory Studies

Laboratory results on admission are shown in **Table 1**. In addition, complete blood count revealed a white blood cell count of $6.2 \times 10^3/\mu L$ (normal, 4.5–11.0 $\times 10^3/\mu L$), hemoglobin of 11.2 g/dL (normal, 12–15 g/dL), hematocrit of 33.4% (normal, 35%–45%), and platelet count of $121 \times 10^3/\mu L$ (normal, 150–450 $\times 10^3/\mu L$). There were no schistocytes on microscopy. Both serum protein and albumin levels were decreased. Alkaline phosphatase, aspartate aminotransferase, and alanine aminotransferase were normal. Of note, her serum creatinine level on admission was 8.5 mg/dL as compared with 1.6 mg/dL 2 months prior to admission.

Diagnosis

Given the marked increase in serum creatinine, the patient was diagnosed with acute-on-chronic anuric renal failure. Bladder catheterization confirmed no

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Table 1. Laboratory Values for the Case Patient

Study	Results
Alanine aminotransferase, U/L	28 (normal, 9–52)
Albumin, g/dL	2.6 (normal, 3.9–5.0)
Alkaline phosphatase, U/L	57 (normal, 38–126)
Aspartate aminotransferase, U/L	24 (normal, 14–56)
Bicarbonate, mEq/L	15 (normal, 22–31)
Blood urea nitrogen, mg/dL	86 (normal, 7–17)
Calcium (total), mg/dL	6.6 (normal, 8.4–10.2)
Chloride, mEq/L	101 (normal, 98–107)
Creatinine, mg/dL	8.5 (normal, 0.7–1.2)
Lactate dehydrogenase, U/L	655 (normal, 50–200)
Phosphorus, mg/dL	5.3 (normal, 2.3–4.7)
Potassium, mEq/L	3.9 (normal, 3.6–5.0)
Protein, g/dL	5.1 (normal, 6.3–8.2)
Sodium, mEq/L	131 (normal, 137–145)

Table 2. Differential Diagnosis of Anuria

Complete urethral or bladder outlet obstruction
Bilateral renal calculi
Operative ligation of the ureters
Bilateral renal artery or vein occlusion
Aortic dissection at the level of the renal arteries
Vasculitis involving the renal arteries
Renal hypoperfusion secondary to shock
Rapidly progressive glomerulonephritis
Hemolytic-uremic syndrome
Nonsteroidal anti-inflammatory drug use
Bilateral cortical necrosis
Infiltrating cancer of the retroperitoneum
Acute tubular necrosis

Data from references 1–4.

urine output despite intravenous administration of several liters of normal saline. Given the patient's history of renal artery bypass, renal artery occlusion was pursued as the cause of the patient's symptoms and was confirmed by renal duplex ultrasound, which showed no blood flow in the left renal graft.

Treatment and Follow-up

The patient underwent an emergent renal graft angioplasty with stent placement in the thrombosed left renal artery graft. Postangioplasty, the patient received 1 hemodialysis treatment immediately to correct for electrolyte abnormalities. The patient's urine output normalized, and she did not require further dialysis treatment. Her electrolyte levels remained within normal limits and urine output stabilized at 100 mL/hr. She was discharged 11 days after admission. Her serum creatinine level had decreased to 3.2 mg/dL at discharge and returned to baseline 1 month later. A repeat duplex ultrasound at discharge showed a patent left renal graft with normal velocities.

DISCUSSION

Differential Diagnosis of Anuria

Anuria is defined as the total absence of urine output, although in clinical practice it is usually defined as urine output less than 100 mL in 24 hours.[1] Anuria is most commonly associated with complete lower urinary tract obstruction and has a limited differential diagnosis, which includes complete urinary tract outlet obstruction, poor renal perfusion secondary to severe shock, total occlusion of the renal arteries or veins, cortical necrosis, acute tubular necrosis, hemolytic-uremic syndrome, and rapidly progressive glomerulonephritis (**Table 2**).[1–4]

Complete urinary tract outlet obstruction is the most common cause of anuria and should be investigated first. In the case patient, urethral and bladder outlet obstruction were ruled out with bladder catheterization, which showed no urine output. Her history, physical examination, and laboratory studies helped to rule out shock, rapidly progressive glomerulonephritis, cortical necrosis, and acute tuberal necrosis, as the patient did not have hypotension or inadequate oxygen delivery, systemic symptoms, edema, or any other signs of vasculitis. Although she was mildly anemic and thrombocytopenic on admission, the patient was afebrile, and microscopy showed no schistocytes, thus ruling out hemolytic-uremic syndrome and thrombotic thrombocytopenic purpura. Given the patient's history of a solitary functioning kidney with stenosis requiring angioplasty, renal artery occlusion became the most likely diagnosis.

Renal artery occlusion is a rare cause of anuria, although its incidence is not reported in the primary literature. Commonly used references list this cause of anuria for completeness but do not discuss it in depth,[2,3] and renal artery occlusion is often not included in the differential for anuria.[4] Although renal artery obstruction is a rare cause, it should be included in the differential because treatment can result in a return to baseline renal function.

Clinical Presentation of Renal Artery Occlusion

The varying presentations of renal artery occlusion can make the diagnosis difficult. Classically, patients with acute thrombosis present with flank pain, nausea, vomiting, and hematuria.[4] However, if thrombosis occurs over time, the development of collateral circulation may result in few symptoms.[4] Physical examination may reveal signs of atherosclerosis, such as hypertension or carotid or abdominal bruits.[5] Laboratory analysis will reveal evidence of acute renal failure with elevated serum creatinine levels and possibly microscopic hematuria.[4] If the kidney becomes infarcted, serum levels of lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase may become elevated, and the release of renin typically results in hypertension.[4] Renal arteriography is diagnostic and allows for possible therapeutic angioplasty and stent placement.[5]

Treatment

Restoration of renal blood flow reverses renal failure and results in rapid return of renal function if infarction has not occurred. One case report suggested that collateral circulation may allow kidney preservation and resumption of renal function when blood flow is restored in patients despite prolonged ischemia and anuria (31 days in this report),[6] which is confirmed by reports from a 30-year period (1965–1996) involving similar patients.[7–34]

Renal percutaneous transluminal balloon angioplasty with or without stenting is commonly used to treat renal artery stenosis. Renal artery stenting has a high success rate initially, usually cited as 95% to 100%.[35] Long-term prognosis is much more difficult to predict. Several studies have attempted to determine which factors are predictive of a better outcome following angioplasty and stent placement. Burket and colleagues[36] found that higher baseline systolic blood pressure may be a predictor of better postprocedure reduction in blood pressure. They found that age, sex, race, diastolic blood pressure, number of vessels stented, and severity of stenosis were not predictive of improved blood pressure or renal function.[36] Radermacher and colleagues[37] used Doppler ultrasonography to measure resistance to flow in the segmental arteries of both kidneys. They found that a resistance index value of 80 or greater was predictive of a poor response to angioplasty or surgery, whereas lower values predicted an improvement in blood pressure control and renal function.

Renal artery angioplasty with stenting has a complication rate of about 10%.[35] The most common complication is hematoma formation at the catheter site. Other more severe complications include pseudoaneurysm, renal artery thrombosis, renal failure, cholesterol embolus, renal infarction, renal artery rupture, renal artery dissection, and death.[38] In our patient with a solitary kidney, the benefits of this procedure clearly outweighed the risks, as the only other option would have been dialysis.

CONCLUSION

The differential diagnosis of anuria is limited. Most causes can be quickly determined by history, physical examination, and bladder catheterization. Although renal artery occlusion is a rare cause of anuria, physicians should consider this diagnosis, especially in patients with known renovascular disease or a history of renal artery surgery, as prompt treatment can be essential to preserving renal function. **HP**

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