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Case Report

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A Case of AKI Exposes Underlying Danger

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Abstract

A 66-year-old male with Acute Kidney Injury (AKI) after cardiac catheterization is discovered to have other comorbidities such as vascular disease, ectopic kidney, and hydronephrosis of the ectopic kidney due to a common iliac artery aneurysm. Discovery of the new comorbidities created a therapeutic challenge which concluded successfully.

Keywords: AKI; Cholesterol emboli; Ectopic kidney; Internal iliac artery aneurysm

Case report

A 66-year-old male with a history of hypertension, hyperlipidemia, decades of smoking, intermittent claudication and chronic kidney disease presumed to be due to hypertensive and vascular disease was admitted with new onset of angina pectoris, dyspnea, and ischemic electrocardiographic changes. Pre-cardiac catheterization labs showed serum creatinine of 1.7 mg/dl (eGFR 55 cc/min) and urinalysis revealing +1 proteinuria by dipstick. The urine did not reveal red blood cells or white blood cells. On March 17, the patient underwent cardiac catheterization, via the right femoral artery, which required i.v. contrast. Two stents were placed, in the left circumflex and the right coronary artery. During the coronary angiography, films of the aorta revealed mild right renal artery stenosis, an ectopic left kidney in the true pelvis, with hydronephrosis of the pelvic kidney due to compression of its ureter by a 3.3 cm left common iliac artery aneurysm. The patient felt well after the procedure, but within 12 hours he complained of nausea, abdominal pain, and paresis of the left upper extremity.

The neurological symptom resolved within 20 minutes. On day 2, the serum creatinine rose to 2.3 mg/dl. On day 3, a CT of the brain without contrast showed white matter ischemic changes and mild cerebral atrophy, without evidence of hemorrhage or infarct. However, an MRI of the brain (without contrast) on the same day revealed a small right posterior frontal cortical infarct as well as multiple lacunes in the right basal ganglia. We considered that AKI was due to contrast nephropathy, but in the context of neurological sequelae we considered cholesterol emboli, or less likely an atheroembolism from a ruptured plaque in the carotid artery. On day 5 the serum creatinine increased to 2.6 mg/dl. Which we considered a delayed AKI, which was more consistent with Cholesterol Emboli Syndrome (CES). On day 6 a radionuclide renal scan was performed showing the normally positioned right kidney, and a smaller left kidney just above the bladder in the pelvis. The right kidney contributed 52% while the left contributed 48% to overall renal function. On day 7 an MRA of the neck showed 60% stenosis of the origin of the right internal carotid with an ulcerated plaque. Low dose anticoagulation was initiated. On day 14 the serum creatinine rose to 4 mg/dl with little tendency to decrease, and by day 19 the patient exhibited livedo reticularis of the lower extremities, with blue toes and an elevated eosinophil count to 8 % of the total white blood count. Peripheral pulses were retained. Because of the delay and saw tooth pattern in creatinine elevation, elevated eosinophils, and livedo rash we diagnosed the cause of AKI as a shower of cholesterol emboli, rather than contrast mediated nephropathy. The patient was discharged on day 22. Over the next two weeks, the serum creatinine elevation revealed small peaks and valleys before reaching the peak of 5.2 mg/dl. Medical management, including fluid restriction, potassium restriction, a statin, amlodipine, metoprolol, nitroglycerine, furosemide, pentoxyphilline and oral corticosteroids was enough to avoid initiation of dialysis. His blood pressure was 160/88 with clear lungs, trace edema. Renal function partially improved over the next 3 months, with creatinine decreasing to 2.9 mg/dl. (eGFR 24 cc/min) and remained stable, permitting carotid repair.

112 days after the cardiac catheterization the patient had right carotid endarterectomy, which allowed us to proceed with repair of the common iliac artery aneurysm, an aneurysm which if left untreated could burst and result in sudden death. By this time, the livedo reticularis had completely disappeared. Serum creatinine

remained stable at 2.8-3 mg/dl. Pre-operative angiography with less than 30 cc. contrast dye, revealed that the renal artery to the left pelvic kidney derived from the right common iliac artery, but the pelvic kidney vein emptied into the left common iliac vein – an unusual cross-over circulation anomaly. The left CIA aneurysm was ligated, and an 8 mm. Dacron graft was placed from the right common iliac artery, distal to the take-off of the renal artery going to the left pelvic kidney. The Dacron graft extended across the pelvis to the left external iliac artery which delivered blood in an antegrade manner to the femoral artery and in a retrograde manner to the left internal iliac artery. The left common iliac artery aneurysm was ligated both distally and proximally and allowed to collapse relieving pressure on the ectopic ureter, partially relieving the hydronephrosis.

Eight months after vascular repair of the CIA aneurysm, a subsequent technetium 99m, Mag-3 renal scan showed no significant hydronephrosis of the pelvic kidney, with 57% function of the right kidney and 43% function of the left pelvic kidney. Serum creatinine stabilized at 2.5 mg/dl at 12 months and remained at this level of GFR (25 cc/min) over the next 5 years. A CT of the pelvis performed 5 years after vascular repair showed a 2.6 cm calcified left common iliac artery aneurysm, with slightly dilated right common iliac artery. Attention to blood pressure control in the range of 120-130 systolic and 80-90 diastolic, lipid control, continuation of pentoxyphilline and cessation of smoking continued. Eight years after ectopic kidney vascular surgery a Lasix renal scan showed no signs of hydronephrosis, but creatinine increased to 3.5 mg/dl. The patient moved to another state and was lost to follow-up.

Discussion

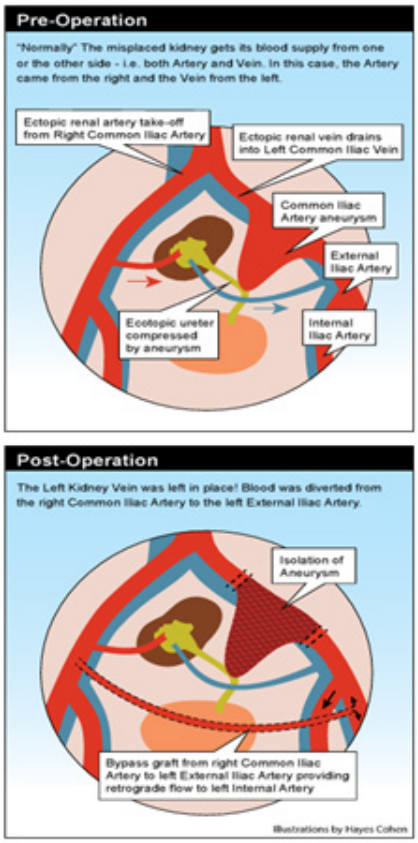
The initial differential diagnosis of AKI after cardiac catheterization includes Contrast Induced Nephropathy (CIN) and multiple Cholesterol Embolization Syndrome (CES). In CIN the trend of serum creatinine is a quick increase within 24-48 hours, with no skin manifestations, or systemic organ system symptoms. Serum creatinine usually descends to baseline levels within 3-4 days. In CES the serum creatinine elevation is delayed by 2-3 days, shows no sign of decreasing and often shows a "saw tooth" or "staircase" pattern of ups and downs over the course of weeks. It is often accompanied by an increased Erythrocyte Sedimentation Rate (ESR), and eosinophilia. Early symptoms of ischemic pain, such as leg pain or cramps, livedo reticularis, "blue-toe syndrome", abdominal discomfort, nausea, anorexia, neurologic changes may occur [1]. Our patient showed a delayed increase in creatinine, livedo reticularis and elevated eosinophils confirming the diagnosis of CES. Urinalysis in either CIN or MCES is not specific or sensitive, as either may include red blood cells, granular casts, or proteinuria. The treatment of CES is controversial. Anticoagulants and fibrinolytics have been discouraged, especially when patients experienced AKI during vascular intervention such as angiography or cardiac surgery. Since the pathophysiology of CES includes small cholesterol crystals inducing microvascular obstruction, ischemia and a profound "foreign body" giant cell inflammatory component, it was decided to treat with pentoxyphilline and corticosteroids. Pentoxyphilline is a competitive non-selective phosphodiesterase inhibitor which inhibits tumor necrosis factor (TNFα) and leukotriene/cytokine synthesis, suppressing inflammation [2-4].

In addition, it improves red blood cell deformability, reduces blood viscosity, and decreases platelet aggregation [5]. It is presumed that the addition of this medication to the patient's long-term care provided additional protection against the progression of renal failure. Corticosteroids, at 1 mg/kg/day of prednisone, was initiated on day 20, and slowly tapered over the course of the next 60 days to stop. Steroids have been shown to have a beneficial effect in controlling the inflammatory reaction induced by the cholesterol crystals [6]. The serendipitous discovery of the pelvic kidney and common iliac artery aneurysm were concerning. Pelvic kidneys account for ~1 of 2500 live births and result from failure of the fetal kidney to ascend to the normal location [7,8]. Patients may have urinary tract infections, vesico-ureteral reflux, nephrolithiasis, and hypertension or remain asymptomatic as this patient did throughout life. The blood supply of a pelvic kidney is atypical and highly variable, often with the vasculature crossing the midline [9]. Clinicians must be aware of unusual clinical presentations or trauma to pelvic kidneys, as well as the potential for surgical misadventure during abdominal or pelvic surgery for other pathology [10]. Iliac artery aneurysms are exceedingly rare, occurring in 0.1% of people. Almost 75% of iliac artery aneurysms occur in the common iliac artery [11]. They are most often due to atherosclerotic vascular disease occurring in males in the 7^{th} or 8^{th} decades of age, but may also be due to connective tissue disorders, fibromuscular dysplasia, or cystic medial necrosis. An iliac artery diameter >1.7cm. is aneurysmal and is found in only 0.03% of the population in autopsy studies.

Iliac artery aneurysms may be asymptomatic but symptoms that may occur are vague and often misinterpreted as being related to other organ systems. The symptoms of the aneurysm may affect the bladder ("pulsatile micturition", frequent urination), hypertension, low abdominal pain, leg, hip, or back pain due to impingement of nerves, or pain during bowel movements, or renal colic pain due to partial obstruction of the ureter from a normally situated kidney [12,13]. These symptoms are related to pressure by the aneurysm on the internal iliac vein, the lumbosacral nerve trunk (posteriorly), the external iliac vein and obturator nerve (laterally) and the colon (medially) [14]. The natural history of an aneurysm is to increase in size, impinge on adjacent structures and possibly rupture in nearly 33% of patients [15,]. The prognosis of patients with untreated iliac artery aneurysms is poor. Rupture is associated with mortality rates 90%. A Common Iliac Artery [CIA] aneurysm of >3.5 cm. presents a risk of rupture [16]. The average size of a ruptured isolated iliac artery aneurysm is between 5 and 7 cm [1,15]. Over 50% of undiagnosed internal iliac artery aneurysm

patients present with spontaneous rupture and rapid death [15]. Preemptive surgery markedly reduces mortality to <10% while the operative mortality in emergent operations is reported to be between 30-50% [16]. Deadly complications such as rupture of the aneurysm into the bladder, colon, or peritoneal cavity have been reported.

A diagnosis of CIA can be suggested by the finding of a curvilinear calcified structure in the lower quadrant on abdominal or pelvic plain films. Intravenous pyelogram IVP s or CT urogram may show the location of an ectopic kidney and its functionality, as well as any degree of obstruction. Abdominal and or pelvic sonography may be useful but are limited by the deep location of these aneurysms and by operator dependency. CT with contrast is the most useful test for the detection and diagnosis of iliac artery aneurysms and ectopic kidneys. Treatment depends on comorbidities and the complexity of the anatomy. Since most iliac aneurysms are discovered in the 6th and 7th decade of life the treatment is usually endovascular repair [17]. AKI in this patient exposed a potentially deadly common iliac artery aneurysm which had been completely silent for the patient except for the overlooked intermittent claudication. The finding represents an urgent intervention, especially if the aneurysm is >3.5 cm. The serendipitous finding of an IAA is a red flag to the physicians who discover it and requires timely referral and treatment.



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