Case Report

A case of atheroembolic renal disease: Not uncommon but underdiagnosed

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ABSTRACT

Atheroembolic renal disease (AERD) is the renal component of the more generalized cholesterol embolization syndrome (CES) that results from rupture of the cholesterol-rich plaques in the larger arteries. Most cases are iatrogenic following invasive intravascular procedures, and >25% occur spontaneously. Cholesterol emboli that lodge distally cause ischemia, inflammation, and end-organ damage. We report a 60-year-old male diabetic, hypertensive with cardiovascular problems, presenting with rapidly worsening renal failure following an angiogram. Renal biopsy confirmed AERD associated with inflammation. He showed a very good response to a course of steroids. AERD, though a common problem, remains largely underdiagnosed. Recent years have seen a rise in the incidence, probably due to an increase in the atherosclerosis risk factors and more patients undergoing invasive intravascular procedures. A high index of suspicion is needed for timely diagnosis, as some of them show a good response to steroids.

Key words: Atheroembolic renal disease, Cholesterol embolization syndrome, Cholesterol plaques, Rapidly progressive renal failure

Atheroembolic renal disease (AERD) is the renal component of the more widespread cholesterol embolization syndrome (CES). They result from the rupture of the cholesterol-rich plaques in the aorta and larger arteries. The higher the plaque burden in vessels, the higher the risk for CES [1]. Plaque rupture is usually iatrogenic following intravascular procedure, thrombolytic therapy, or anticoagulant use, but sometimes CES can occur spontaneously in the absence of a triggering event in approximately 25% of cases [2]. Cholesterol emboli lodge distally in the smaller arteries, arterioles, and capillary bed of various organs and tissues, causing ischemia, inflammation, and organ dysfunction [3]. CES commonly affects the kidneys, intestines, and lower limbs because the abdominal aorta and ilio-femoral artery are the most common sites for plaque deposition. The severity of plaque deposition is less in the proximal aorta [4]. The kidneys are at high risk for embolization because of their proximity to the aorta and their increased blood supply [2]. An autopsy series involving the elderly population has shown the incidence of CES at 30% following angiography and 0.09–4.3% cases occurred spontaneously [5]. Incidence of clinically significant cholesterol embolism, including percutaneous intervention, is 0.6–0.9% [6].

Here, we report the case of an elderly man who presented with rapidly progressive renal failure due to AERD following angiography. Kidney biopsy helps make a definite diagnosis in cases that occur spontaneously or in the absence of extra-renal embolic features. In our patient, steroids were stopped once we had a diagnosis of AERD, but creatinine worsened, necessitating reinitiation of steroids. Renal functions then improved, emphasizing its role in selected cases. AERD should be considered in the differential diagnosis of acute kidney injury in a post-vascular procedure setting with a subacute rise in creatinine with livedo reticularis, digital ischemia, eosinophilia and low complements occurring more commonly in the elderly with diffuse atherosclerosis.

CASE REPORT

A 60-year-old male, diabetic for the past 8 years on glimepiride and vildagliptin, hypertensive for 2 months on cilnidipine, has peripheral vascular disease and is a reformed smoker.

He presented to the emergency unit with an acute onset of breathlessness, blood pressure (BP) was 210/100 mmHg, mild edema, and basal crackles.

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He was admitted for severe hypertension and acute left ventricular failure.

Chest X-ray showed pulmonary edema, and two-dimensional echocardiography showed reduced left ventricular function with an ejection fraction of 42%, with regional wall motion abnormalities. He had an estimated glomerular filtration rate of 66 mL (serum creatinine 1.2 mg/dL). Lower limb pulses were weak, but there were no skin changes.

He underwent a coronary and peripheral angiogram through the femoral route using 50 mL of iodinated non-ionic contrast. Angiogram revealed triple-vessel coronary disease with diffuse involvement of both lower limb arteries. The patient was advised of coronary artery bypass graft and, in the meantime, put on cardiac medications which included aspirin 150 mg, clopidogrel 75 mg, atorvastatin 40 mg, and furosemide 40 mg/day.

Investigations done on a follow-up visit 1 week later revealed mild renal failure (creatinine 1.5 mg/dL, day 7 post-angiogram). Urine routine showed albumin nil, and microscopy was normal. Ultrasound abdomen showed normal-sized kidneys with a mild increase in cortical echotexture. At this point, the rise in creatinine was thought to be due to diuretic-related volume depletion. Based on clinical assessment, the diuretic was withdrawn, and the patient was advised to follow up after another week. The patient's heart failure symptoms had resolved by now. Serum creatinine had increased to 3.5 mg/dL; serial reports are depicted in Table 1.

The patient was advised to consult a nephrologist; however, he delayed and came to us when his creatinine had increased to 4.5 mg/dL. He complained of easy fatigability and mildly decreased appetite. He was clinically euvolemic, with body weight of 60 kg, urine output of 1.5 L/day, and BP of 140/80 mmHg. Clinical examination and fundoscopy were normal; repeat urine showed trace albumin and normal microscopy. Antinuclear antibodies and antinuclear cytoplasmic antibodies were negative; serum complement C3 and C4 were normal. Renal biopsy was deferred at this stage as the patient was on antiplatelets, which were first withheld. In view of rapidly worsening renal failure, he received three doses of pulse methylprednisolone followed by oral steroids at 0.5 mg/kg/day.

A renal biopsy was done 1 week later. The biopsy core, which had 10 glomeruli, was adequate to make a diagnosis. A few glomeruli were sclerosed, all viable glomeruli were normal, and there was tubular atrophy and interstitial fibrosis in 30–40% cortical area. There was a lymphocytic interstitial inflammatory infiltrate; the arcuate artery and arteriole showed transparent needle-shaped clefts (cholesterol cleft) surrounded by an inflammatory reaction, which was suggestive of AERD (Figs. 1 and 2). Kidney functions showed improvement, and his creatinine at the last follow-up at 8 weeks was 1.6 mg%.

DISCUSSION

The risk factors for CES include diabetes, hypertension, age above 60, smoking, intravascular procedures, thrombolytic therapy, anticoagulants, and blunt trauma. One study showed no increased risk with thrombolytic therapy. Radial artery approach carries a lesser risk compared to the femoral [7,8].

A high index of suspicion is required for early diagnosis. AERD presents as acute/subacute renal failure or as a slowly progressive decline in renal function over several months, leading to chronic kidney disease. Contrast nephropathy and acute tubular necrosis (hypovolemia, hypotension) occur in the periprocedure period and should be considered in the differentials. Still, they present more acutely and improve by 2 weeks, unlike AERD, which manifests after 2 weeks. Drug-induced allergic interstitial nephritis also has an acute/subacute onset of renal failure.

The clinical features, such as livedo reticularis, nail fold infarcts, purple toes, and gangrene, if present, help in diagnosis. Other manifestations are fever, myalgia, anorexia, nausea, vomiting, abdominal pain, gastrointestinal hemorrhage, hepatic involvement, pancreatitis, altered mental status, stroke, mononeuropathy, and retinal emboli.

Urine shows mild to moderate proteinuria and bland sediment, but nephrotic-range proteinuria and hematuria are documented, especially in glomerular embolism [7,8]. Urine eosinophils accounting for more than 5% of the urine white blood cell count are seen in 33% when the Hansel stain is used. Raised erythrocyte sedimentation rate, C-reactive protein, peripheral blood eosinophilia in 14–70%, decreased serum complement, and a weakly positive ANA may be seen. Most cases of AERD are diagnosed on clinical grounds, considering the risk factors with skin, muscle, and ophthalmology findings.

Renal biopsy may be required for definitive diagnosis, especially in spontaneous cases. Cholesterol crystals are seen as biconvex needle-shaped clefts (appear as empty space as crystals dissolve during routine preparation) in smaller arteries, arterioles, and glomeruli. Initially, there will be neutrophilic and eosinophilic infiltration around the occluded vessel, which over time gets replaced by mononuclear cells. Over time, blood vessels get completely occluded, causing ischemia, glomerulosclerosis, tubular atrophy, and interstitial fibrosis. Similar pathology is seen in other organs.

Table 1: Number of days post-angiogram

Investigations	D0	D7	D14	D21	D28
Serum creatinine mg/dL	1.2 (106.8 μmol/L)	1.5	3.5	4.5	4.9 (433.16 μmol/L)
Blood urea mg/dL	20	45	60	70	102 (17.85 mmol/L)

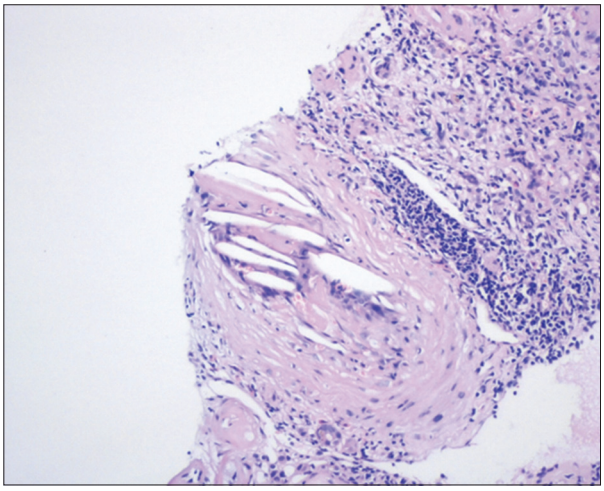


Figure 1: Hematoxylin-Eosin stain, small artery showing transparent needle-shaped clefts (cholesterol clefts) with surrounding inflammation

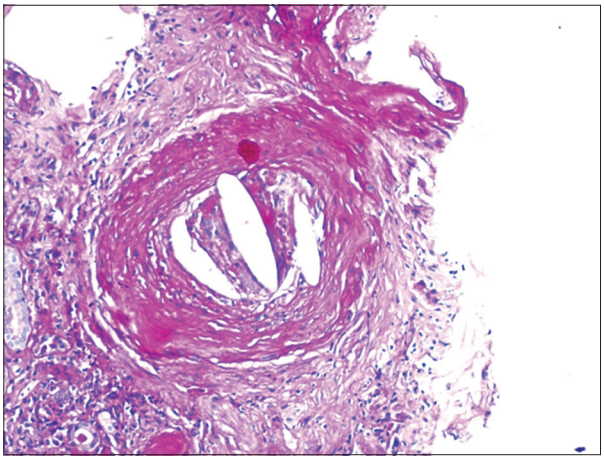


Figure 2: Periodic acid-schiff stain-vascular sclerosis of media with intimal thickening. Cholesterol clefts are also seen

Due to the high prevalence of hypertension, arteriolar nephrosclerosis is seen in many patients.

Patient and renal survival are poor, and the most common cause of death is cardiovascular events. 30% of patients require dialysis, some require long-term dialysis, and 30% show improvement in creatinine over a variable time if there are no fresh showers of emboli to the kidneys [5,7].

There is no consensus or guidelines for treatment, and different treatment approaches are described in the literature. In addition to BP and diabetes control, smoking cessation and the use of high-dose statins, other treatments that have been tried include steroids, prostaglandin analogs, cyclophosphamide, colchicine, and interleukin-1 inhibitor [5,9,10]. These may be beneficial in the acute/subacute forms of disease. Since inflammation is a key component of the pathology in CES, the use of anti-inflammatory agents may find rationale [11-13]. Jung, in a case report, has shown the benefit of short-term steroids in selected cases [11]. Our patient had all risk factors for CES, presented subacutely, and the biopsy showed AERD. His creatinine improved with steroids but worsened when stopped after 2 weeks (creatinine 4.2 mg/dL). Steroids were restarted at 1 mg/kg for 1 week, followed by a slow taper, and his creatinine at the end of 8 weeks was 1.6 mg/dL.

CONCLUSION

AERD is common but remains an underdiagnosed disease; a high index of suspicion is needed for early diagnosis, especially in patients without any precipitating events. Steroids, if initiated early in the course, may improve the outcomes in AERD with acute/subacute presentation.

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