Severe Abdominal Pain Eight Years after Renal Transplant: A Case of Renal Transplant Atherosclerosis

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INTRODUCTION

Chronic mesenteric ischemia (CMI), also known as intestinal ischemia, is a condition that occurs when plaque builds up in the major arteries that supply the small intestine. When discussing CMI, it is important to distinguish between acute and chronic mesenteric ischemia. Acute mesenteric ischemia is a medical emergency caused by an acute loss of blood flow to the small intestine, leading to bowel infarction. This is either secondary to arterial emboli, likely originating from the heart, or soft plaque rupture leading to arterial thrombosis. In contrast, CMI is a constant hypoperfusion of the small intestine due to significant atherosclerosis and vessel narrowing, without plaque rupture.

CMI presents with weight loss, pain with eating, and food aversion often resulting in significant morbidity and a delayed diagnosis. The common risk factors that increase the likelihood of chronic mesenteric ischemia include age greater than 60 years, smoking history, uncontrolled dyslipidemia, diabetes, and hypertension. This is a case of an elderly female who was diagnosed with a 90% stenosis of the superior mesenteric artery (SMA), despite having few well-known risk factors.

CASE REPORT

A 72-year-old female presented to the emergency room (ER) with severe abdominal pain after oral intake and significant malnutrition (BMI of 17.5 on admission). Her history included end stage renal disease status-post renal transplant in 2012, coronary artery disease status-post stenting of the left anterior descending coronary artery (LAD) in 2019, and dyslipidemia. She was a lifetime non-smoker, non-diabetic with no history of hypertension. Her dyslipidemia was well controlled (most recent lipid profile demonstrated a cholesterol of 170 mg/dL and low-density lipoprotein of 75 mg/dL).

Upon presentation to the ER, the patient underwent emergent CT abdomen/pelvis imaging, which demonstrated non-calcified plaque at the origin and proximal SMA resulting in a high-grade stenosis. There was no evidence of bowel ischemia or bowel obstruction.

Given her abdominal symptoms and imaging findings, vascular surgery was consulted but did not recommend acute surgical intervention. The following morning, interventional radiology performed a mesenteric angiogram demonstrating a 90% stenosis of the SMA, and the SMA was stented successfully (Figure 1) with improved distal flow. Following successful endovascular revascularization, patient's abdominal pain following oral intake immediately and completely resolved.

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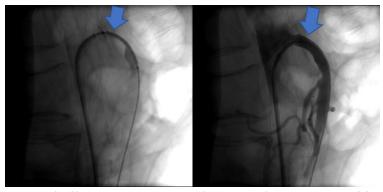


Figure 1. (Left) Mesenteric angiogram demonstrated a 90% stenosis of the SMA as shown by the blue arrow. (Right) Subsequent imaging following stent placement as shown by the blue arrow with improved distal flow.

DISCUSSION

In an older patient who presents to the emergency room with abdominal pain after oral intake, a broad differential should be maintained. The differential diagnoses include acute cholecystitis, acute mesenteric ischemia, chronic pancreatitis, chronic mesenteric ischemia, and peptic ulcer disease, among others. As seen in our patient, the only risk factors that she had prior to presentation were age and LAD coronary artery disease. As such, her pretest probability for chronic mesenteric ischemia being the etiology of patient's abdominal pain was low.

When CMI is considered on the differential, the diagnosis is based on symptoms and imaging. Computer tomography angiography (CTA) is the primary imaging modality in patients whose clinical suspicion of CMI is moderate to high.² On CTA, atherosclerotic plaque has a sensitivity of 100% and specificity of 95%. If CTA is unable to be obtained given renal insufficiency or contrast allergy, magnetic resonance angiography (MRA) can be performed.⁵ While MRA has both a high sensitivity (95%) and specificity (100%) in detecting mesenteric ischemia, it has a limited role in diagnosing distal stenosis as well as nonocclusive mesenteric ischemia. Additionally, its use may delay therapeutic options in acute settings because of the lengthier testing duration, making CTA the imaging modality of choice if able.⁶

Despite limited risk factors, our patient had a significant 90% stenosis of the SMA. The factor that makes our patient's presentation unique was that she had a renal transplant. Renal transplantation can accelerate cardiac atherosclerosis and the metabolic syndrome. Courivaud et al.⁷ found 32% of renal transplant patients met criteria for metabolic syndrome one year after transplant. Kasiske found atherosclerotic cardiovascular complications developed in 15.8% of patients during the post-transplant follow-up period.⁸ Our patient had coronary artery disease requiring stenting of her LAD seven years after her renal transplant, despite being in otherwise good health. Established data demonstrated the mortality benefit of statin therapy in renal transplant patients.⁹ The ALERT trial showed that twelve-year survival rates were higher (73%) in statin users versus non-statin users (64%) in renal transplant recipients. There is a

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paucity of data assessing the prevalence of renal transplantation and non-cardiac atherosclerosis as well as possible prevention of noncardiac atherosclerosis.

Once the diagnosis has been established by CTA, revascularization is indicated in patients to relieve symptoms.^{2,4,10-12} Previously, open surgical revascularization was the standard therapy. However, more recently, endovascular revascularization is less invasive and is now favored as the treatment of choice. 10-12 Cases of chronic mesenteric ischemic should be discussed with both vascular surgery and interventional radiology to discuss the rapeutic options and the best route for revascularization.

CONCLUSIONS

The prevalence of non-cardiac atherosclerotic conditions in renal transplant patients has not been well established in literature. As demonstrated by our case, renal transplantation should be considered a major risk factor for non-cardiac atherosclerotic disease. The prevalence of renal transplant and subsequent CMI should be assessed in larger clinical trials. In doing so, the focus can turn to prevention of non-cardiac atherosclerotic disease in this already vulnerable transplant patient population.

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