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Acute Renal Failure After Uterine Artery Embolization

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Abstract

Renal failure is a potential complication of any endovascular procedure using iodinated contrast, including uterine artery embolization (UAE). In this report we present a case of acute renal failure (ARF) following UAE performed as a treatment for uterine fibroids. The likely causes of ARF in this patient are explored and the possible etiologies of renal failure in patients undergoing UAE are reviewed.

Key words: Acute renal failure—Uterine artery embolization—Uterine fibroids

Uterine artery embolization (UAE) is a procedure that is gaining acceptance as an alternative treatment for symptomatic uterine fibroids [1–3]. Although the complication rate for this procedure is relatively low, there are unique complications seen during UAE that are not seen during surgical treatments for fibroids [4]. Renal failure is a complication that can occur after any endovascular procedure including UAE that is not often seen following hysterectomy or myomectomy. A case of acute renal failure (ARF) after UAE is described. The management of this patient and a review of the possible causes of ARF are discussed.

Case Report

A 26-year-old woman with a past medical history significant for severe dysfunctional uterine bleeding and anemia secondary to uterine fibroids presented to the hospital with 6 days of severe vaginal bleeding. Over the past 4 years the patient had been hospitalized twice for severe vaginal bleeding requiring transfusion with multiple units of packed red blood cells (PRBC). During the 4 years she had been on various forms of hormonal therapy in an attempt to control her dysfunctional uterine bleeding without any success. On this admission her hemoglobin was 4.3 g/dl. MRI performed during this admission revealed a very large single fundal fibroid measuring 15.7 × 10.3 × 13 cm.

An extensive discussion regarding her treatment options ensued. Because of her young age and desire for future fertility, a uterus-sparing treatment option was strongly desired. Medical therapy had failed adequately to control the bleeding in this patient. Myomectomy was discussed as an option. Because of the large size of the fibroid, it was determined that if myomectomy was attempted, there was an approximately 30% chance a hysterectomy would need to be performed. This chance of hysterectomy was not acceptable to the patient and therefore it was decided to perform a UAE.

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After receiving 4 units of PRBC, her preprocedural hemoglobin was 7.6 g/dl. Her preprocedural creatinine (Cr) measured 0.7 mg/dl. A bilateral uterine artery embolization was performed using Contour polyvinyl alcohol (PVA) particles (355–500 μm) (Boston Scientific, Natick, MA) with 2 vials of PVA delivered to each artery. A total of 100 cm^3 of Omnipaque 300 mg I/ml (Amersham, Princeton, NJ) contrast was used. An aortic injection was performed after the embolization to look for ovarian arteries feeding the fibroid. No ovarian arteries were visualized. The renal arteries were not selected and no significant catheter manipulation was done near the renal arteries.

Pain control, while the patient was in the hospital, consisted of preprocedural Toradol (Hoffmann-La Roche, Nutley, NJ) 30 mg IV, pre- and postprocedural morphine, postprocedural Motrin (McNeil Pharmaceuticals, Ft. Washington, PA) 800 mg every 8 hours and Percocet (Endo Pharmaceuticals, Chadds Ford, PA). Her pain was well controlled and her hemoglobin was stable. The patient was discharged to home the following day. She was given a prescription for Motrin 800 mg (to be taken every 8 hours for 3 days, then as needed for pain) and Percocet 5/325 mg (1 or 2 tablets to be taken as needed for pain every 4–6 hr).

Four days after her UAE she presented to clinic with complaints of anorexia, nausea/vomiting and mild abdominal pain, which had been present since the UAE. Serum creatinine drawn at the time of the clinic visit was 2.2 mg/dl. This was a concerning rise in creatinine, but the decision was made to manage her as an outpatient with close-interval follow-up. The patient was sent home under close observation with instructions to maintain aggressive hydration. She was seen in clinic 3 days later. Repeat creatinine taken at this time measured 4.5 mg/dl. The patient had taken Motrin 800 mg every 8 hours since she was released from the hospital. Immediately following her clinic visit, the patient was admitted to the hospital for management of renal failure. Her hemoglobin on admission was noted to be 6.3 g/dl. She was transfused with 2 units PRBC and mounted an increase in hemoglobin to 8.6 g/dl. She was also hydrated with normal saline. Her urine analysis was essentially normal and did not indicate eosinophiluria or have any white blood cell casts. In addition, there was no elevation of the peripheral eosinophil count. A renal ultrasound was performed which ruled out an obstructive cause of renal failure. Perfusion to the kidneys was noted to be normal on Doppler ultrasound.

The patient responded to persistent hydration with steadily decreasing serum creatinine levels (Fig. 1). On the day of discharge, 4 days after her admission, the patient's creatinine measured 2.9 mg/dl. She also had improvement of her nausea, vomiting and abdominal pain, and her hemoglobin was stable. The patient was followed closely as an outpatient and her creatinine continued to fall. On postoperative day 31 her creatinine measured 1.0 mg/dl. One hundred and eighty days after the UAE her Cr was 0.8 mg/dl.

Discussion

Renal failure can result from many different insults to the kidneys. This patient's differential diagnosis included renal vascular causes

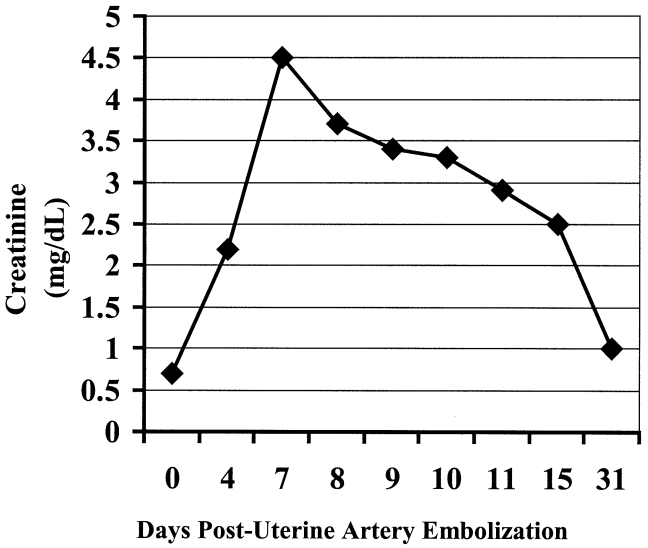

Fig. 1. The progression of serum creatinine levels (mg/dl) during the first month after uterine artery embolization.

as well as pre-renal, renal and post-renal causes of renal failure. It was initially hypothesized that the ARF may have been due to a post-renal obstructive cause. However, the patient was noted to be non-oliguric and reported no difficulty in voiding, and a renal ultrasound showed no evidence of obstruction bilaterally. The renal vascular causes are thromboembolic occlusion of the renal artery or vein. There was no indication during the UAE procedure for risk of either event. In addition, in the setting of normal kidneys prior to UAE, it is unlikely a unilateral renal artery occlusion would cause ARF. To further alleviate such concerns a Doppler ultrasound of the kidneys demonstrated normal blood flow to the renal parenchyma and patent proximal renal arteries.

Two main factors in this case pointed toward a pre-renal cause of acute renal failure. The first is that the patient had clinical evidence of decreased effective arterial blood volume (EABV) with the presence of orthostatic changes in vital signs. Her decreased EABV was secondary to her vomiting and vaginal bleeding. The second factor is the patient's nonsteroidal anti-inflammatory drug (NSAID) use, specifically Motrin 800 mg every 8 hours for the previous 5 days. It has been shown that especially in the setting of renal hypoperfusion, inhibition of renal prostaglandin synthesis by NSAIDs can result in ARF. The mechanism for this is that the vasodilatory prostaglandins cause afferent arteriolar dilation to maintain glomerular blood flow in the setting of hypoperfusion. However, NSAID use prevents the increase in prostaglandin production resulting in decreased glomerular blood flow and pre-renal ARF. Furthermore, NSAIDs may also cause an acute interstitial nephritis resulting in ARF even without esosinophiluria or increased peripheral eosinophil count [5, 6].

Another possible cause of this patient's ARF is contrast nephropathy. This is less likely, due to the normal preprocedural renal function and the relatively low volume of iodinated contrast used. The iodinated contrast could have been an added risk factor for ARF along with decreased effective arterial volume and concurrent NSAID use [7]. Lastly, the release of myoglobin from the ischemic degradation of the large fibroid could possibly have played a role in the ARF. This was thought to be unlikely because of the normal urine analysis [8].

Although the most likely cause of the ARF in this case is a decreased EABV causing pre-renal ARF worsened by NSAID use, iodinated contrast use also likely played a role. All three of these factors are under the physician control during UAE. It is important to recognize renal failure as a possible complication of UAE. ARF is likely underdiagnosed in UAE patients because postprocedural creatinine levels are rarely obtained. Limiting contrast use, encouraging hydration and, in patients with decreased EABV, limiting and/or monitoring NSAID use would help to decrease the risk of ARF in patients undergoing UAE.

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