Case Report

Prolonged renal failure post-percutaneous mechanical thrombectomy

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Abstract

Percutaneous mechanical thrombectomy (PMT) has been gaining acceptance as a preferred approach for the treatment of acute deep venous thrombosis (DVT). In addition to treating acute DVT and decreasing the risk of pulmonary embolism, it has been reported that direct extraction of the thrombus decreases the risk of post-thrombotic syndrome (PTS), the economic impact of managing which is reported to account for 75% of the total cost of management of DVT patients. PMT combines localized thrombolysis with mechanical thrombectomy. Recently, there have been some reports of reversible acute kidney injury (AKI) occurring post-PMT. The pathophysiology of AKI in such cases is due to hemoglobinuria-associated acute tubular necrosis. Therefore, the overall prognosis of AKI post-PMT has been reported to be good. We report here a case of AKI post-PMT for an extensive DVT of the lower extremity whereby the patient continues to require HD even 5 months after the procedure. The patient had normal renal function prior to the procedure and evidence of hemoglobinuria at the time of diagnosis of AKI. Our case illustrates that patients with a large thrombus load may develop severe AKI post-PMT thus requiring hemodialysis for an extended period of time. Limiting the length of time that the mechanical thrombectomy is performed and quantifying the amount of effluent obtained would appear to be a prudent practice to reduce the risks of renal failure. However, no specific guidelines exist as for the limits of hemolysed exudates to be collected.

Keywords: mechanical thrombectomy; deep venous thrombosis; acute kidney injury

Introduction

Percutaneous mechanical thrombectomy (PMT) has been primarily used for arterial thrombus but more recently it has been gaining acceptance as a preferred approach for the treatment of acute deep venous thrombosis (DVT). The procedure basically combines localized thrombolysis (with local thrombolytic infusion) and mechanical thrombectomy (guide wire directed catheter vibrations of 500–3000 rpm). To prevent any embolism and systemic dispersion of the thrombolytic agent, proximal and distal balloons are positioned outside the thrombus. After mechanical activation for 5–15 min, the proximal balloon is deflated and the fragmented thrombus is aspirated under the cover of an inflated distal balloon. Cases of reversible acute kidney injury (AKI) post-PMT have been described. We report here a case of prolonged renal failure in a patient who underwent PMT.

Case presentation

Our patient was a 48-year-old male with past history of well-controlled hypertension. He presented to the emergency department (ED) with a 1-week history of painful swelling of the left leg. On examination the patient’s heart rate was 82 beats/min; blood pressure 136/80 mmHg and he was saturating 100% on room air. Doppler examination of the extremity revealed an extensive DVT in the left lower extremity, for which he underwent PMT. Preprocedure, the laboratory evaluation was essentially normal including a blood urea nitrogen (BUN) of 15 mg/dL (reference range 7–18 mg/dL) and a serum creatinine of 0.85 mg/dL (reference range 0.7–1.4 mg/dL). The patient was hydrated with 0.9% saline throughout the procedure and there was no record of any hypotensive episode during the procedure. The immediate postprocedure period was also uncomplicated with documentation of good urine output and the patient was thus discharged a day after. He was discharged on subcutaneous low-molecular weight heparin and oral anticoagulation (warfarin) with advice to follow up as an outpatient. Six days later, he presented to the ED with complaints of generalized weakness and decreased urine output. He denied shortness of breath, chest pain, abdominal pain, fever or chills. He denied any recent nonsteroidal inflammatory drug use. The laboratory evaluation was essentially normal including a blood urea nitrogen (BUN) of 15 mg/dL (reference range 7–18 mg/dL) and a serum creatinine of 0.85 mg/dL (reference range 0.7–1.4 mg/dL). The patient was hydrated with 0.9% saline throughout the procedure and there was no record of any hypotensive episode during the procedure. The immediate postprocedure period was also uncomplicated with documentation of good urine output and the patient was thus discharged a day after. He was discharged on subcutaneous low-molecular weight heparin and oral anticoagulation (warfarin) with advice to follow up as an outpatient. Six days later, he presented to the ED with complaints of generalized weakness and decreased urine output. He denied shortness of breath, chest pain, abdominal pain, fever or chills. He denied any recent nonsteroidal inflammatory drug use. Physical examination was unremarkable. Laboratory analysis now revealed a BUN of 194 mg/dL (reference range 7–18 mg/dL), creatinine of 36 mg/dL (reference range 0.7–1.4 mg/dL), potassium of 7.9 mEq/L (reference range 3.5–5.1 mEq/L), bicarbonate of 15 mmol/L (20–29 mmol /L) and calcium of 7.9 mg/dL (8.4–10.2 mg/dL). The urine sample was red and urine analysis (urine dipstix) was strongly positive for blood but microscopy showed only 2–4 red blood cells/
high-power field (hpf). The serum creatinine kinase (CPK) level was normal—50 ng/mL (38–120 ng/mL). Based on the random urine sodium of 30 mg/dL and random urine creatinine of 90 mg/dL, the calculated Fractional Excretion of Sodium (FENa) was 8.5. Renal ultrasound revealed normal sized kidneys with normal cortical texture and no hydrenephrosis.

The patient thus had an AKI with evidence of acute tubular necrosis (ATN). A red-colored urine in the setting of only 2–4 RBC’s/hpf on urine analysis and a normal serum CPK level were all suggestive of hemoglobinuria as the underlying cause of ATN.

In view of marked hyperkalemia, the patient was started on emergent hemodialysis (HD). Intravenous hydration (with bicarbonate) was continued after the first session of HD; however, no recovery in renal function was noted. He continues to be HD dependent, even 5 months after his discharge from the hospital.

Discussion

DVT results in significant patient morbidity and mortality [1]. Anticoagulation therapy does not prevent post-thrombotic syndrome (PTS), the most common late complication of DVT which is characterized by chronic leg heaviness/aching, edema and chronic trophic skin changes-associated venous ulceration [1]. The economic impact of managing PTS and the corresponding complications account for 75% of total cost of management of DVT patients [1]. In addition to treating acute DVT and decreasing the risk of pulmonary embolism, it has been reported that direct extraction of the thrombus decreases the risk of PTS [1]. PMT basically combines localized thrombolysis with mechanical thrombectomy. The system consists of three components: a single-use catheter with proximal and distal balloons with drug (thrombolytic agent) infusion holes between the two balloons and a single-use pump and a pump drive unit. The proximal and distal balloons are positioned outside the thrombus thus preventing systemic dispersion of the thrombolytic agent. A thrombolytic dispersion wire is placed in the catheter which when activated by an electrical pump causes the catheter to vibrate at 500–3000 rpm. Mechanical action of vibration along with localized dispersion of a thrombolytic agent within the thrombus causes rapid resolution of the thrombus. After mechanical activation for 5–15 min, a proximal balloon is deflated and the thrombus is aspirated under the cover of the inflated distal balloon which will prevent any systemic embolization. The combination of a localized thrombolytic agent with mechanical lysis increases the efficacy of the procedure.

AKI is a known complication of endovascular procedures [2]. It may range from the commonly encountered dye-associated AKI post-cardiac catheterization to the more uncommon but severe atheroembolic episodes following procedures such as coronary artery bypass grafting [2]. PMT may be complicated by pancreatitis, retroperitoneal hematoma, hemolysis, reocclusion, partial removal of thrombus and reversible AKI [1, 3, 4]. Our patient, however, underwent a venous endovascular procedure and therefore an atheroembolic episode is unlikely. A thromboembolic episode is a possibility but there was no evidence of systemic clot embolization. Furthermore, there was no evidence of renal infarcts on kidney imaging or frank hematuria. Our patient refused kidney biopsy, but the presence of a red-colored urine in the setting of only 2–4 RBC’s/hpf on urine analysis and a normal serum CPK level, with a FENa of 8.5 were all suggestive of hemoglobinuria associated AKI. While the exact mechanism of hemoglobinuria causing renal failure is unclear, it is thought to be a nephrotoxic effect of heme proteins which cause cast formation and obstruction, resulting in ATN in the proximal tubular cells of the kidney [5]. Because the underlying pathogenesis is ATN, the overall prognosis of AKI post-PMT has been reported to be good, except that our patient continues to be on HD even 5 months after undergoing the procedure. Although not associated with PMT, there have been reports of delayed renal recovery (even 6–12 months) after an episode of severe ATN-associated AKI in the surviving intensive unit care patient population [6]. As was evidenced by his preprocedure BUN and serum creatinine levels, the patient had no evidence of chronic renal insufficiency. Based on the laboratory analysis, other rare causes of kidney injury were also ruled out [7].

In conclusion, our case illustrates that patients risk development of severe AKI from hemoglobinuria post-PMT, requiring HD for an extended period of time. This should be included in the list of potential complications associated with PMT. Patients with a large thrombus load (lower extremity veins) are likely to be at the highest risk of AKI. In addition to maintaining adequate intravenous hydration during and after the procedure, limiting the length of time that the mechanical thrombectomy is performed, and more importantly quantifying the amount of effluent obtained, would appear to be a prudent practice to reduce the risks of renal failure. However, no specific guidelines exist as to the limits of hemolyzed exudates to be collected. It would also be worthwhile to point out that the procedure should not be done on an outpatient basis, especially if the clot burden is large.

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