Acute delayed onset hemodynamic and respiratory compromise due to thoracic translocation of hemourinoma after percutaneous nephrolithotomy☆

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ABSTRACT

Percutaneous nephrolithotomy (PCNL) despite its minimally invasive approach has an high complication rate, with the most common complications being extravasation of urine and perioperative bleeding requiring transfusion. While most of these complications are minor, many serious and life-threatening complications do occur. One such complication is the development of hemothorax or hydrothorax which usually develops in the early postoperative period with blood or urine passing from the surgical site through a newly established pleural-peritoneal fistula. Here we describe an unusual presentation and clinical management of delayed onset acute urohemothorax and hemodynamic collapse several days following PCNL.

Introduction

Percutaneous nephrolithotomy (PCNL) has become a common procedure for the removal of renal calculi due to the direct approach it provides, while causing less damage to surrounding tissues as compared to open surgery.1,2 Despite the minimally invasive approach, overall complication rates have been estimated at as high as 83% with some of the most common complications being extravasation of urine and perioperative bleeding requiring transfusion.1,2 While majority of these complications are minor, many serious and life-threatening complications do occur.1,3,4 We describe an unusual presentation and clinical management of a patient who developed delayed acute onset of urohemothorax and hemodynamic collapse following PCNL.

Case description

A 56-year-old male with cystinuria and chronic kidney disease (BUN 19 mg/dL, creatinine 1.8 mg/dL) presented for PCNL and holmium laser lithotripsy of left cysteine staghorn calculus. The procedure was performed with the patient under general anesthesia in the prone position. Percutaneous access via supracostal approach to the posterior aspect of the upper pole of the left kidney was established under endoscopic and fluoroscopic guidance. The procedure lasted approximately 6 h requiring extensive laser treatment and placement of an ureteral stent. Immediate postoperative chest radiograph revealed no evidence of pneumothorax, consolidation or pleural effusion. On postoperative day 1, patient developed tachycardia and persistent oxygen desaturation. A repeat chest x-ray showed no evidence of consolidation, pleural effusion, or pneumothorax at that time (Fig. 1). A ventilation/perfusion scan was obtained that reported high probability of pulmonary embolism and heparin infusion was initiated and titrated to a thromboplastin time goal of 60–90 seconds. On POD 3, the anticoagulation was discontinued secondary to the development of hemothorax and acute blood loss anemia (from 13.9 g/dl to 9.2g/dl). In addition, the patient developed worsening kidney injury and the serum creatinine increased to 6.1 mg/dl. A CT scan revealed significant blood collection in the left renal collecting system with moderate perinephric hematoma (Fig. 1), however there was no evidence of focal consolidation, pleural effusion, or pneumothorax at that time (Fig. 1). A ventilation/perfusion scan was obtained that reported high probability of pulmonary embolism and heparin infusion was initiated and titrated to a thromboplastin time goal of 60–90 seconds. On POD 3, the anticoagulation was discontinued secondary to the development of hemothorax and acute blood loss anemia (from 13.9 g/dl to 9.2g/dl). In addition, the patient developed worsening kidney injury and the serum creatinine increased to 6.1 mg/dl. A CT scan revealed significant blood collection in the left renal collecting system with moderate perinephric hematoma (Fig. 1), however there was no evidence of focal consolidation, pleural effusion, or pneumothorax (Fig. 2). Emergent transthoracic echocardiogram showed normal left and right ventricular size and
function. Patient was scheduled for emergent cystoscopy and ureteral stent exchange.

Immediately upon arrival to the preoperative unit, the patient suffered acute cardiopulmonary decompensation with tachycardia, tachypnea, and severe hypoxia. He was emergently intubated and placed on mechanical ventilation. Additionally, aggressive resuscitation with intravenous fluids and infusion of vasopressors was initiated. Emergent chest X-ray demonstrated massive left pleural effusion (Fig. 3). On left tube thoracostomy 1500 mL of blood-tinged serosanguinous fluid was drained. The patient was transferred to intensive care unit for further management. A total of eight units of packed red blood cells were administered and the patient was placed on continuous renal replacement therapy (CRRT). On POD 7, the patient was successfully weaned from mechanical ventilation. During his stay in the ICU a left perinephric drain and inferior vena cava filter were placed. Rest of his hospital stay was uneventful and unremarkable.

Discussion

Due to the complexity of the procedure, our patient likely sustained renal vasculature injury during the complex PCNL which was subsequently exacerbated in the setting of anticoagulation. In addition, it appears, an inadvertent subclinical injury to the parietal pleura during the initial percutaneous supracostal approach went unrecognized in the immediate intraoperative and postoperative period. The sudden severe life-threatening hemodynamic and respiratory compromise during transportation to preoperative area likely occurred due to acute translocation of abdominal contents into the left pleural cavity. This is clearly inferred, since the CT and X-ray chest obtained an hour prior to the catastrophic event show no evidence of fluid in the left pleural cavity. Despite the tendency for postoperative pleural effusion to develop early, there have been reports of PCNL-associated delayed onset hemo- or hydro-thorax. Large majority of such effusions are caused by diaphragmatic irritation and are considered to be sympathetic in nature. One such case involved the insidious development of pulmonary symptoms over the course of three days, one week after an uncomplicated PCNL.5

Primary risk factors, such as, operative times and ASA classification, cannot be easily controlled by the surgical team. In contrast, certain choices, such as the location of the percutaneous approach and the use of anticoagulation, can significantly increase the risk of serious complications and may create a clinical dilemma for the physician, who must weigh the risks and benefits of each therapy. For instance, anticoagulation has been shown to cause an absolute increase of 0.29 to the mean Clavien grade of PCNL complications.7

Potentially the highest risk factor for pleural injury and therefore subsequent development of hemo- or hydro-thorax is the chosen point of access. With a subcostal approach, the reported incidence of pleural injury has been reported at 0.5% compared to punctures above the 12th rib at 12%, and, those above the 11th rib at 23.1%. Therefore, a subcostal entry should be preferred whenever possible. Furthermore, fluoroscopic guidance and a puncture timed with end-expiration may also help decrease the risk.

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