Superior vena cava syndrome: A rare complication of percutaneous nephrolithotripsy laser lithotripsy

Ji Qing, Yang Jianjun, Jia Hongbin, Zhang Lidong
Department of Anesthesiology Jinling Hospital, School of Medicine, Nanjing University, Nanjing 210002, P.R. China

Abstract

Purpose: To describe a case of acute superior vena cava syndrome during percutaneous nephrolithotomy (PCNL), and to review the associated clinical features, management and complications.

Clinical features: A 34-year-old man, diagnosed as right renal calculi and nodal tachycardia, was admitted to receive percutaneous nephroscope laser lithotripsy. Shortly after stone disintegration, he suffered acute hypoxic and hypotension, and showed cyanoderma of face and chest skin, ocular proptosis, jugular filling and ventricular fibrillation. His vital signs subsequently became stable and cyanoderma faded. The patient was eventually discharged from the intensive care unit three days following the event.

Conclusion: Severe complications such as cardiac arrest could happen during PCNL. Close monitoring the vital signs is essential for early finding and quick response to rescue.

Key Words: Percutaneous nephrolithotripsy, superior vena cave syndrome, cardiopulmonary-cerebral resuscitation

INTRODUCTION

Percutaneous nephrolithotomy (PCNL) is a surgical technique with minimal invasive dissection, higher stone-free rates, shorter operation time and quicker post-surgery recovery.\(^1,2\) This procedure carries certain complications such as bleeding, urine leakage, renal parenchyma laceration, renal perforation, arteriovenous fistula and injury of nearby organs.\(^3\) However, severe complications were rarely encountered.\(^4\) Here we report an unusual case of superior vena cava syndrome (SVCS) that occurred in a patient during PCNL to remove right renal calculi.

CASE REPORT

A 34-year-old patient, 65 kg, was diagnosed as right renal calculi. He had American Society of Anesthesiologists (ASA) physical status II and had no previous medical history. The patient underwent PCNL in prone and bend position. Before anesthesia, BP was 120/79 mmHg and heart rate (HR) was 82 bpm. Intravenous general anesthesia was induced with midazolam 0.03 mg/kg, fentanyl 3 µg/kg, target-controlled infusion (TCI) with propofol 4 µg ml\(^{-1}\), and maintained with fentanyl 3 µg/kg and propofol 3–4 µg/ml. Neuromuscular block was produced with rocuronium 0.8 mg/kg and maintained with continuous infusion of vecuronium 0.05–0.06 mg/kg/h. After oral trachea intubation, the patient was mechanically ventilated to keep the end tidal carbon dioxide tension (PETCO\(_2\)) between 30 and 35 mmHg. A ureteric balloon catheter was placed at the height of the ureteropelvic junction (UPJ) following retrograde pyelography. The collecting system was punctured under ultrasound control, followed by a single-step dilatation with a 14F synthetic dilator. A 15F metal Amplatz sheath was inserted over the dilator. Stone disintegration was achieved with laser lithotripsy. The pressure of the water pump was kept at 150 mmH\(_2\)O.

In the first 80 min, hemodynamics was stable with BP 98–110/60–75 mmHg and HR 65–85 bpm. Then, a blood loss of about 250 ml occurred in the sight of surgical field.
Blood pressure immediately dropped to 50/42 mmHg from 100/63 mmHg with the HR 62 bpm. The arterial waveform diminished and the pulse pressure dropped to 5 mmHg. The possibility of the obstruction or mislocation of arterial canula was quickly cleared. Ephedrine 30 mg was intravenously injected in one minute.

In the next 2 minutes, PETCO\(_2\) decreased to 23 mmHg, systolic BP (SBP) decreased to 45 mmHg. The patient showed engorgement, ocular proptosis, jugular filling and dilated pupil of about 7 mm. Areas around head, neck, both upper extremities and 3–4 cm below nipples of thoracic region presented cyanoderma. The BP was about 40 mmHg. An arterial blood sample was obtained for blood gas analysis. The possibilities of breathing pipeline system blockage, one-lung ventilation or mislocation of endotracheal canula were excluded.

The patient was turned over to supine position. A central line was inserted and dopamine was injected intravenously. A minute later, SBP decreased to 35 mmHg. The surgery stopped and adrenaline of 0.5 mg was intravenously injected. The blood gas analysis showed that PaO\(_2\) at 274 mmHg, PaCO\(_2\) at 33 mmHg, and pH, base excess, Na\(^+\), K\(^+\), Ca\(^{++}\) and hematocrit were normal. Then adrenaline 2 mg was intravenously injected. SBP increased to 200 mmHg, HR 82 bpm. One minute later, ECG showed ventricular fibrillation. External cardiac massage and electric defibrillation were carried out immediately and the patient successfully cardioverted after the 2nd shock and 2 min of massage. BP was 80/35 mmHg and HR was 85 bpm. 5% sodium bicarbonate 150 ml was intravenously injected. Dopamine was administrated at the rate of 8 µg/kg/min simultaneously. Subsequently vital signs became stable. Cyanoderma presented in the pate, upper extremities and 3-4 cm below nipples of thoracic region almost vanished (See Figure 1 for summary).

The patient was admitted to ICU with mechanical ventilation. Measurements from second blood gas analysis were normal [Table 1]. Chest X-ray showed a high density patch located between the 7th and 8th rib of the right side chest. The general density of right side lung was higher than the left side. Chest X-ray the following day revealed the disappearance of the localized high-density patch. The density of the right lung remained higher than left side [Figure 2]. After three days, the patient was awake and oriented. His circulation was stable. The patient breathed spontaneously well and the respirator was detached. The patient was released the next day.

**DISCUSSION**

We reported a case of probable fatal superior vena cava syndrome (SVCS). The successful rescue was due to close monitoring, quick analysis of contributing factors and prompt treatment.

Dyspnea and symptoms of airway obstruction are often the complaints of conscious patients.\(^5,6\) However, SVCS may become indistinguishable during surgery because of the lack of utterable complaints and variable degrees of clinical presentation. Close monitoring of vital signals was essential to discover the development of SVCS at an early stage.

Many factors could lead to the development of SVCS. In our case, one potential reason was that injury to the right side pleural membrane caused pleural effusion and pressured the mediastinum. This was supported by the relatively high-density patch on the right side of chest found in chest X-ray. However,
Qing, et al: Superior vena cave syndrome during nephrostolithotomy

Figure 1b: The change of heart beat

Figure 1c: The clinical procedures taken at different time points

Figure 2: Chest X-ray results - (a) X-ray taken at 45 min after surgery. Notice the high-density patch located between right 7th and 8th ribs. The general density on the right side is also slightly higher than the left side; (b) X-ray taken at second day after surgery. Notice the higher density patch has already disappeared

it was unlikely. First, the amount of fluid in the chest cavity, judged from the density in the X-ray, was not much. That small amount of fluid was very unlikely to provide the enough pressure to compress the superior vena cava. Second, after changing the patient to supine position and external cardiac massage, the signs of SVCS quickly receded. It suggested a quick relief of pressure on the superior vena cava. Such a quick change of pressure is unlikely to be conducted by the fluid in a relatively big space, such as chest cavity. Considering these two points together, small amount of water and quick
pressure change, it is more likely the SVCS was caused by the water unexpectedly presented in the mediastinum, a very limited space with relatively stiff wall. With the water pump running, the rising water, although small, can cause the pressure against superior vena cava to rise fast, which eventually led to mediastinal tamponade and SVCS.[7,8,9] Anatomically, the retroperitoneal cavity connects with mediastinum at some location. When the kidney was punctuated, with the water pump running at 150 mmHg, it was possible that certain amount of water was pushed into the retroperitoneal cavity and found its way to mediastinal space.[10]

Once the potential reasons are recognized, treatments were straightforward to remove these factors by changing the patient position and external heart massage.

REFERENCES

1. Bird VG, Fallon B, Winfield HN. Practice patterns in the treatment of large renal stones. J Endourol 2003;17:355-63.
2. Lahme S, Bichler KH, Strohmaier WL, Götz T. Minimally invasive PCNL in patients with renal pelvic and calyceal stones. Eur Urol 2001;40:619-24.
3. Rozentsveig V, Neulander AZ, Roussabrov E, Schwartz A, Lismer L, Gurevich B. Anesthetic considerations during percutaneous nephrolithotomy. J Clin Anesth 2007;19:351-5.
4. Sacha K, Szewczyk W, Bar K. Massive haemorrhage presenting as a complication after percutaneous nephrolithotomy (PCNL). Int Urol Nephrol 1996;28:315-8.
5. Queen JR, Berlin J. Superior vena cava syndrome. J Emerg Med 2001;21:189-91.
6. Hemann R. Superior vena cava syndrome. Clin Excell Nurse Pract 2001;5:85-7.
7. Aslam PA, Eastridge CE, Hughes FA Jr. Mediastinal tamponade: report of a case. Am Surg 1970;36:248-50.
8. Gajre G, Dammas AS. Mediastinal tamponade following external jugular vein cannulation. Indian Pediatr 2001;38:1177-80.
9. Roddin M, Alexander D. Mediastinal tamponade. Anaesthesia 2000;55:705-6.
10. Kukreja RA, Desai MR, Sabnis RB, Patel SH. Fluid absorption during percutaneous nephrolithotomy: does it matter? J Endourol 2002;16:221-4.

Source of Support: Nil. Conflict of Interest: None.