Abdominal hypertension characterised by severe haemodynamic changes as a complication of percutaneous nephrolithotomy

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SUMMARY

Percutaneous nephrolithotomy (PCNL) was performed on a patient in a prone position. During the first hour of intervention, some unwanted haemodynamic complications arose, which included a decrease in systemic blood pressure, an increase in heart rate and an increase in peak inspiratory pressures. Owing to worsening of those unexplained haemodynamic abnormalities, surgery was terminated. As the patient turned from prone to supine position, abdominal distention was noticed. Later, 3 litres of fluid were removed from the abdomen via peritoneal lavage. Upon fluid removal, a dramatic haemodynamic improvement was observed. The problem was thought to have been caused by a technical error in placing the dilator inside the abdomen, which allowed the irrigation fluid to accumulate inside the abdominal cavity. The aim of this presentation is to raise awareness of intra-abdominal hypertension when the aforementioned haemodynamic abnormalities are detected during PCNL. Early detection and treatment may prevent morbidity and mortality in similar cases.

BACKGROUND

Minimally invasive techniques have become more and more popular, leading to the replacement of open surgical intervention with percutaneous nephrolithotomy (PCNL) as the preferred treatment method for nephrolithiasis. PCNL has become a common treatment method due to its high success rate, minimal blood loss and short recovery time with minimal morbidity.¹ However, there are some complications associated with this procedure, such as, bleeding, massive absorption of irrigation fluid, fever, infection, colon perforation and pneumothorax.^{2–6}

Abdominal hypertension is defined as intra-abdominal pressure of 12 mm Hg or more. If abdominal hypertension is not recognised, it may result in abdominal compartment syndrome (ACS). ACS is a decrease in blood perfusion to the tissues due to high pressure and resulting loss of circulation. ACS may cause organ dysfunction due to lack of blood flow.⁷

We would like to present a case in which serious haemodynamic complications developed due to abdominal hypertension-ACS during PCNL.

CASE PRESENTATION

A 30-year-old man underwent percutaneous nephrolithotomy under general anaesthesia. The patient was classified as ASA I according to the American Society of Anesthesiologists's classification. The patient did

not have any problems on preoperative evaluation. Preoperative laboratory findings were as follows: haemoglobin 14 g/dl, haematocrit 41%, glucose 100 mg/dl, urea 30 mg/dl, creatinine 0.8 mg/dl, sodium 140 mEq/l and potassium 4.2 mEq/l.

The patient was administered 0.5 mg of atropine and 2.5 mg of midazolam intramuscularly 30 min prior to the operation. The patient was monitored by ECG, and through non-invasive blood pressure and peripheral oxygen saturation (SpO₂) measurements in the operating room. Haemodynamic parameters were as follows: blood pressure 130/80 mm Hg, heart rate (HR) 80 beats/min, and SpO₂ 98%. Anaesthesia induction was performed by injection of 160 mg of propofol, 7 mg of vecuronium and 200 mcg of fentanyl. Anaesthesia maintenance was provided by 6–7% desflurane inside 50% O₂. Fentanyl and vecuronium were replaced as needed. Respiration was maintained via mechanical ventilation keeping ET-CO₂ between 30–40 mm Hg. Peak inspiratory pressure was 20 mm Hg after intubation.

The left ureteric catheter was placed while the patient was in the lithotomic position. Later, the patient was switched to a prone position, and surgery was initiated. Haemodynamic parameters remained stable during the initial hour of the surgery. However, after the first hour, the patient's HR started to gradually increase and his blood pressure decreased slowly. Blood loss was assumed to be the cause of the blood-pressure drop. Therefore, in response, the patient's complete blood count (CBC) was obtained and 500 cc of colloid fluid was administered. Haemoglobin concentration was measured at 11 g/dl, and haematocrit at 34%. Towards the end of the surgery, the patient's HR increased to 130 beats/min and systolic blood pressure dropped to 80 mm Hg. Peak inspiratory pressures rose to 30 mm Hg. However, SpO₂ stayed normal. Spasms or secretions inside the airways or lungs were ruled out by auscultation and endotracheal aspiration. The surgeons were informed of the haemodynamic changes during the second hour of surgery and intervention was terminated. After surgery, the patient achieved spontaneous ventilation with a high respiratory rate but low tidal volume. The patient was administered 2 mg of neostigmine and 0.5 mg of atropine intravenously to reverse the effects of the paralytics.

After termination of surgery, the patient was turned from the prone to the supine position. Abdominal distention and tension were observed via inspection and palpation. Peritoneal lavage was performed via an intraperitoneal catheter, and fluid was drained via passive drainage. Shortly after passive drainage, the haemodynamic parameters

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improved-HR returned to 90 beats/min, blood pressure was 110/80 mm Hg and SpO₂ was 99%. The patient was extubated soon after respiration became more regular. Arterial blood gas did not show any electrolyte abnormality (pH 7.34, PCO₂ 38.1 mm Hg, PO₂ 168 mm Hg, Na 143 mEq/l, K 3.61 mEq/l, HCO₃ 21 mmol/l, BE-3). The patient was transferred to the recovery room with an HR of 98 beats/min, a blood pressure of 130/80 mm Hg and SpO₂ of 98%. The patient experienced no further problems and was discharged 2 days after the operation.

DISCUSSION

ACS is a clinical condition in which abdominal distension and increased intra-abdominal pressure, respiratory insufficiency, increased central venous pressure and decreased urine output are observed. After decompression, all indications disappear and patients recover in full. ACS develops after abdominal trauma, especially after massive intra-abdominal bleeding or intestinal oedema, intestinal obstruction, pregnancy or ascites. Intra-abdominal hypertension precedes ACS. Therefore, ACS is preventable given timely and appropriate intervention.^{7,8}

Abdominal hypertension or ACS may develop due to iatrogenic aetiologies, such as laparoscopy or massive fluid resuscitation.⁸ The goal of our report was to present a case with abdominal hypertension as a complication secondary to massive intra-abdominal fluid accumulation and following erroneous placement or displacement of a dilator.

In our case, we initially considered the increased HR and low blood pressure, observed during the second hour of our surgery, to be a bleeding complication of PCNL.⁹ It is hard to estimate the amount of blood lost, during PCNL surgery, due to bloodshed on the surgical dressings and on the floor; thus, we used the patient's CBC to estimate the blood loss. We replaced the volume lost via colloid infusion. Haemoglobin concentration was only decreased from 14 to 11, which would not explain the significant clinical change. Therefore, transfusion was not considered, and volume loss was replaced using colloids.

The increase in the peak inspiratory pressures may have been due to dislocation of the endotracheal tube towards the bronchi, secretions, bronchospasm and pneumothorax, which can happen during this type of procedures.¹⁰ However, the patient had normal breathing sounds, no secretions upon aspiration and normal SpO₂.

After termination of surgery, the patient was turned from prone to supine position. Abdominal distention and tension were noticed via inspection and palpation, causing us to consider intra-abdominal hypertension. Three litres of fluid were drained via peritoneal lavage. The surgical team reported difficulty in placing the dilator during pelvicalyceal intervention which, they stated, may have caused fluid leakage inside the intra-abdominal cavity. The accumulated fluid inside the intra-abdominal cavity resulted in increased intra-abdominal pressure, decreased venous return, decreased cardiac output and increased intrathoracic pressure, all of which cause unwanted haemodynamic consequences. In addition, increased intra-abdominal pressure may decrease functional residual capacity and may adversely affect respiration by elevating the diaphragm.¹¹ Urine output is also expected to

drop along with haemodynamic and respiratory compromise. During PCNL, a Foley catheter is not introduced and the pelvicalyceal system is irrigated intensely; thus, we were not able to control urine output. Koroğlu *et al*¹² established that there were no clinically significant changes in fluid-electrolyte balance when 0.9% NaCl was used in PCNL. Electrolyte imbalance was not observed, because it was used 0.9% NaCl during PCNL.

In our case, intra-abdominal hypertension was not considered until the patient was turned from prone to supine position. The aim of this presentation is to raise awareness of intra-abdominal hypertension when the aforementioned haemodynamic abnormalities are detected during PCNL cases. Early detection and timely treatment may prevent morbidity and mortality in similar cases.

Learning points

- Intra-abdominal hypertension should be considered when the aforementioned haemodynamic abnormalities are detected during percutaneous nephrolithotomy cases.
- When intra-abdominal hypertension is suspected, timely treatment may prevent morbidity and mortality.

Competing interests None.

Patient consent Obtained.

Provenance and peer review None.

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