Incidental finding of severe hyperkalemia in a patient with end-stage renal disease during video-assisted lung lobectomy: A case report

ABSTRACT
Patients with end-stage renal disease are at risk of developing hyperkalemia and acidosis, both of which have disastrous sequelae during elective video-assisted thoracic surgery for lung cancer. Herein, we present a case where severe hyperkalemia and combined acidosis were incidentally found in a 68-year-old man with the end-stage renal disease after establishing one-lung ventilation during video-assisted lobectomy. There was no significant instability of vital signs, abnormality of perioperative electrocardiography, or malignant arrhythmia. Therefore, we arranged for related management promptly, and the surgery was relatively smooth. This incidental intraoperative hyperkalemia was thought to have resulted from one-lung ventilation and hypercarbia and/or metabolic acidosis. More frequent arterial blood gas analysis and aggressive blood potassium control during video-assisted thoracic surgery should be considered for patients with end-stage renal disease.

Key words: Acidosis, end-stage renal disease, hyperkalemia, one-lung ventilation, video-assisted thoracic surgery

Introduction
According to the United States Cancer Statistics, the annual number of newly diagnosed lung cancer cases has increased in the past 15 years. This may reflect the prevalence of older adults in the general population and the availability of advanced radiological screening modalities such as chest multi-detector computed tomography. Moreover, older patients have more comorbidities, including poor renal function, and can develop postoperative complications and unexpected outcomes.[1]

VATS, one of the treatments for lung cancer, often entails intraoperative one-lung ventilation (OLV), which can induce respiratory acidosis due to hypoventilation. Patients with end-stage renal disease (ESRD) may experience hyperkalemia and metabolic acidosis during VATS, resulting in malignant arrhythmia or other fatal complications. However, anesthesiologists focus on monitoring respiratory conditions and vital signs and miss acidosis and relative conditions, which leads to catastrophic postoperative complications, including malignant arrhythmia and cardiac arrest.

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Case Report

A 68-year-old man was diagnosed with pulmonary adenocarcinoma and scheduled for video-assisted right lower lung lobectomy and mediastinal lymph node dissection. He weighed 66 kg, and his height was 167.5 cm. He had multiple comorbidities, including hypertension, chronic obstructive lung disease, and ESRD (stage 5 chronic kidney disease [CKD]), all of which were medically managed. Preoperative renal replacement therapy was not indicated. Preoperative laboratory investigations are summarized in Table 1. Preoperative electrocardiography (ECG) showed a sinus rhythm and several atrial premature complexes, and chest radiography revealed a 1.5 cm nodule in the right lower lung.

Induction and intubation were performed uneventfully. A 37-Fr double-lumen endotracheal tube was placed at a distance of 31 cm from the incisor. The surgery was performed with the patient in the left lateral position. The surgery duration was 220 min under OLV; the tidal volume was approximately 4.55 mL/kg, and the end-tidal CO$_2$ level was approximately 31–40 mmHg. Intraoperative ECG (lead II) showed non-significant elevation of the T wave, similar to that observed in the preoperative ECG [Figure 1]. We administered 1,500 mL of intravenous fluid during surgery. The urine output was 400 mL, and the estimated blood loss was approximately 50 mL.

The arterial blood gas (ABG) analysis immediately after shifting from one-lung to two-lung ventilation showed severe hyperkalemia and metabolic acidosis [Table 2]. Pseudohyperkalemia was suspected. Re-evaluation of potassium level revealed persistent hyperkalemia (7.33 mmol/L); hence, we arranged related management including intravenous glucose solution, regular insulin, and calcium chloride.

After the treatment, the patient’s potassium level dropped to 5.67 mmol/L, while the pH and bicarbonate level increased to 7.283 and 19.1 mmol/L, respectively [Table 2]. He was transferred to the intensive care unit for postoperative recovery. During his stay in the intensive care unit, his potassium levels were relatively unremarkable. Exubination was performed uneventfully on postoperative day 2, and he was transferred to the regular ward the next day.

Discussion

Hyperkalemia is a potentially life-threatening condition wherein serum potassium levels exceed 5.5 mmol/L.[2] The possible causes of perioperative hyperkalemia can be categorized into (1) reduced urinary potassium excretion, (2) increased potassium load, and (3) altered potassium distribution.[3] These causes include but are not limited to severe acidosis and propofol infusion syndrome.[4–5]

In the present case, hyperkalemia may have resulted from ventilator-induced acute renal failure due to CKD, severe acid-base disturbance, OLV-induced hypoventilation, or severe acidosis. Severe acidosis can enhance the transcellular shift of potassium into the bloodstream to maintain electroneutrality, thereby, elevating serum potassium levels.[6] Conversely, hyperkalemia can cause metabolic acidosis via its multiple effects on renal ammonia metabolism,[7] leading to the development of a vicious cycle between metabolic acidosis and hyperkalemia. Other

| Parameter                        | Result   |
|----------------------------------|----------|
| Hemoglobin                       | 10.7 g/dL|
| Hematocrit                       | 32.3%    |
| Na$^+$                            | 139 mmol/L|
| K$^+$                             | 5.1 mmol/L|
| BUN                               | 77 mg/dL |
| Creatinine                       | 6.62 mg/dL|

BUN: blood urea nitrogen

| Parameter                     | After OLV | Rechecked | After Treatment |
|-------------------------------|-----------|-----------|----------------|
| Plo2 (%)                      | 60        | 60        | 60             |
| pH                             | 7.194     | 7.172     | 7.283          |
| pCO2 (mmHg)                   | 44.2      | 48        | 40.1           |
| pO2 (mmHg)                    | 218.6     | 245.2     | 313.6          |
| Hematocrit (%)                | 30        | 30        | 27             |
| Na$^+$ (mmol/L)               | 141.4     | 141.8     | 143.3          |
| K$^+$ (mmol/L)                | 7.61      | 7.33      | 5.67           |
| CI (mmol/L)                   | 118.3     | 119.0     | 118.2          |
| Lactate (mmol/L)              | 0.8       | 0.6       | 1.1            |
| HCO3$^-$ (mEq/L)              | 17.2      | 17.7      | 19.1           |

OLV: one-lung ventilation

Figure 1: Intraoperative electrocardiogram (ECG) in the lead II revealed mild elevation of the T wave (a), as is apparent in the preoperative ECG (b)

Table 2: Intraoperative parameters and arterial blood gas analysis

Table 1: Results of hematologic laboratory tests 2 days before surgery

| Parameter         | Result  |
|-------------------|---------|
| Hemoglobin        | 10.7 g/dL |
| Hematocrit        | 32.3%    |
| Na$^+$            | 139 mmol/L |
| K$^+$             | 5.1 mmol/L |
| BUN               | 77 mg/dL |
| Creatinine        | 6.62 mg/dL |

BUN: blood urea nitrogen

*OLV*: one-lung ventilation
CO$_2$ insufflation and surgical stimulation during VATS may also lead to hyperkalemia. According to an animal study, CO$_2$ insufflation time and plasma level were positively correlated during laparoscopic surgery.[8] Theoretically, systemic acids induced by CO$_2$ absorption may cause hyperkalemia, although there is scant literature about this phenomenon.[9] Surgical stimulation influences the serum potassium level in two phases. In the first phase, hyperkalemia may occur due to potassium release from hepatocytes after stress-induced alpha-1 adrenergic stimulation. In the second phase, hypokalemia may occur due to muscle cell uptake after stimulation of beta-2 receptors.[9]

Our patient’s preoperative estimated glomerular filtration rate was below 10 mL/min/1.73 m$^2$ and may have hampered the renal excretion of potassium.[10]

The effects of OLV on potassium balance during thoracic surgery remain highly debated. Theoretically, acute hypoxic pulmonary vasoconstriction in the non-ventilated lung can reduce the activity of the voltage-gated potassium channels controlling the membrane potential, causing the currents into the extracellular space to decrease and the serum potassium level to increase.[10]

This case highlights the possibility of respiratory and metabolic acidoses during VATS in patients with CKD and consequent severe hyperkalemia. Differential diagnoses of metabolic disturbances must always be considered to avoid misdiagnosis solely based on the association of OLV with hypoxia and hypercarbia. Although preoperative laboratory findings were within the normal ranges in our patient, we acknowledge that it might have been safer to monitor his potassium levels during OLV to prevent acute hyperkalemia. Perioperative ABG analysis or transcutaneous CO$_2$ monitoring may facilitate the timely diagnosis or prevention of silent, yet fatal, hyperkalemia in patients with ESRD.

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Ethics approval and informed consent
The study was approved by the Institutional Review Board of the authors’ affiliated institution informed consent is fully accomplished before initiation of the study.

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Conflicts of interest
There are no conflicts of interest.

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