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Case Report

A critical adverse event during minimally invasive oesophagectomy

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KEYWORDS

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Abstract Thoracic minimally invasive procedures may produce certain life threatening complications perioperatively. Vigilent monitoring, prompt diagnosis and timely intervention are very important to reduce morbidity and mortality in such surgeries.

1. Introduction

Minimally invasive oesophagectomy is gaining popularity. We present a life threatening complication during one such procedure which was managed successful.

2. Case report

A 60-year-old female with distal one third oesophageal cancer was admitted for thoracoscopic + laparoscopic oesophagectomy. She had no significant medical or allergic history. Physical and laboratory examination did not reveal any significant abnormality. She was optimized preoperatively with bronchodilators, chest physiotherapy, hydration and incentive spirometry.

Left radial artery cannulation and right internal jugular venous catheterization were done under local anaesthesia. General anesthesia was induced with Thiopentone and Fentanyl. Vecuronium was given for muscle relaxation. She was intubated orally with 7 mm ID single lumen armored endotracheal tube and bilateral equal air entry was confirmed. Balanced anaesthesia was maintained with air, oxygen isoflurane vecuronium and fentanyl. The patient was placed in prone position with due precautions. Capnothorax was established to displace the right lung by insufflation of carbon dioxide through a Verrez needle using an electronic pressure controlled carbon-dioxide insufflator at a rate of 2 L/min creating a pressure less than 10 mm of Hg.

Thoracoscopic mobilization of the oesophagus was uneventful and took 80 min. An underwater sealed chest drain with continuous suction was inserted through one of the access ports. The rest of the ports were closed. Electrocardiogram, pulse oximetry, intra arterial blood pressure and central venous pressure were maintained within normal limits throughout the procedure. The fresh gas flow and minute ventilation were adjusted to maintain an ETCO2 between 37 and 39 mm of Hg.

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The patient was then turned supine after clamping the chest tube. On turning supine peripheral pulses were not felt, arterial waveform was absent and cardiac sounds were not audible. Standard basic life support measures were started immediately. ECG showed coarse ventricular fibrillation. Patient was defibrillated with 150 J using a biphasic defibrillator and CPR continued with injections of Adrenaline repeated every 3 min. Following this sinus rhythm appeared for a short period with recurrent pulseless ventricular tachycardia. Patient was again defibrillated with 150 J and a bolus injection of 40 U of vasopressin was given. CPR continued for almost 20 min. Patient regained spontaneous respiration with adequate tidal volume. Sinus rhythm was established with an intra arterial blood pressure of 68/38 mm of Hg and SPO2 of 100%. Dopamine infusion was started at 7 mcg/kg/mt to maintain a mean arterial pressure of 80 mm of Hg. The central venous pressure remained unchanged during the event but the ETCO2 and the peak airway pressure were increased.

Chest X-ray showed no pneumothorax. ABG revealed partially compensated non-respiratory acidosis. On restoration of a stable haemodynamic state, patient was shifted to ICU. The rest of the surgical procedure was deferred.

3. Discussion

The common causes of cardiovascular collapse during laparoscopic surgery include dysrhythmia, vasovagal reactions, blood loss, cardiac tamponade, carbon dioxide embolism, pneumothorax and pneumomediastinum.

The timing of the event in our patient coincided with the clamping of the chest tube and changing of the patient position from prone to supine position. During the change of position carbon dioxide must have escaped from the pleural space into the posterior mediastinum elevating intrathoracic pressure resulting in a cardiac tamponade like situation with impairment of venous return and hemodynamic compromise [1,2].

Theoretically the presence of a thoracostomy tube should have decompressed the right thorax. but a size 28 French gauge tube, even if ideally positioned and patent, may not have been able to cope with the volume of carbon dioxide in the thorax or there might had been an encysted pocket of carbondioxide not drained by the thoracostomy tube. The continuous CPR and subsequent release of the thoracostomy tube clamp might have driven out the trapped gas releasing the tamponade effect.

Carbon dioxide embolism which occurs if blood vessels are opened, would be expected to cause fall in end tidal CO_2 which was not seen. Stimulation of the vagus nerve in the oesophageal hiatus could cause hypotension but this episode occurred at a time when the surgical instruments had been removed from the hiatus which makes vagal stimulation unlikely.

There are reports of cardiovascular collapse in laparoscopic Nissen fundoplication [3]. Pneumothorax has been reported to complicate laparoscopic cholecystectomy [4] because of peritoneo-pleural communications.

In our case we did not use a double lumen tube which seems to be a more common practice. Palanivelu describes thoracoscopic part of esophagectomy done in prone position with single lumen endotracheal tube, pneumothorax being maintained with low pressure insufflation of 6 mm Hg [5].

Anticipation of such complications and complete evacuation of the pneumoperitoneum and pneumothorax are emphasized to minimize morbidity and mortality. If carbon dioxide insufflation is necessary, it is important to keep the pressure low (between 2 and 7 mm Hg).

This case report illustrates a critical adverse event that occurred in the context of a relatively new surgical procedure and highlights the need for eternal vigilance.

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