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CASE REPORT

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Life threatening tension pneumothorax during cardiac surgery. A case report

A. Jain, D. Arora, R. Juneja, Y. Mehta, N. Trehan

Medanta Institute of Critical Care and Anesthesiology, Medanta The Medicity, Gurgaon, Haryana

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ABSTRACT

Tension pneumothorax is a life threatening condition that occurs when the intrapleural pressure exceeds atmospheric pressure. It requires prompt diagnosis and immediate treatment. Tension pneumothorax developing postoperatively after cardiac surgery is not uncommon but occurrence in the operating room during cardiac surgery is rare. We report a case of tension pneumothorax intraoperatively during off pump coronary artery bypass grafting.

Keywords: *pneumothorax, cardiac surgery, hypotension.*

INTRODUCTION

Tension pneumothorax occurs due to a one way communication between lung parenchyma and the pleural cavity leading to air entrapment in the pleural cavity with each inspiration with inability to release it during expiration. It requires prompt diagnosis and immediate treatment or it may lead to respiratory failure and cardiovascular collapse (1, 2). Tension pneumothorax developing postoperatively after cardiac surgery is not uncommon but occurrence intraoperatively during cardiac surgery is rare and not yet reported in the English literature. We report a case of tension pneumothorax occurring intraoperatively during off pump coronary artery bypass grafting (OPCAB).

Corresponding author:
Dr. Ashish Jain
DNB Anesthesia
Medanta- The Medicity
Sector 38, Gurgaon, Haryana, INDIA
e-mail: drashishjain07@rediffmail.com

CASE REPORT

A 62 years, 60 kg male with coronary artery disease, past history of smoking (60 pack-years), history of dyspnoea and chest pain on exertion NYHA grade II-III since one year was electively admitted for OPCAB. There was no history of chronic cough, recurrent respiratory tract infections, previous hospitalization, use of beta2 agonist or steroids, or history suggestive of chronic obstructive pulmonary disease (COPD), occupational lung disease or tuberculosis. General physical and systemic examination was within normal limits. Preoperative haematological investigations and pulmonary function test (PFT) were within normal limits. Chest radiograph revealed emphysematous changes. Transthoracic echocardiography showed no regional wall motion abnormality (RWMA) with normal valvular and left ventricular (LV) function.

Induction of general anesthesia was un-

eventful and anesthesia was induced with fentanyl sulphate, midazolam, thiopentone sodium and maintained with isoflurane and air oxygen mixture. Orotracheal intubation was easy, facilitated with vecuronium bromide and without airway trauma. Intermittent vecuronium bromide and fentanyl sulphate were used intravenously in standard doses. Pulmonary artery catheter introducer sheath and a triple lumen central venous catheter were inserted in the right internal jugular vein under ultrasound guidance in the first attempt without any complication. A pulmonary artery catheter was then floated through the sheath. During OPCAB the left pleura was opened whilst harvesting the left internal mammary artery while the right pleura remained intact. The left lung was observed to be hyperinflated without any evidence of bullae. The patient was mechanically ventilated on volume control ventilation mode under low flow anesthesia at 1.0 litre/minute with a tidal volume (TV) of 8 ml/kg, respiratory rate of 14/min and I:E ratio of 1:2.5 without application of PEEP, achieving a peak inspiratory pressure (PIP) of 18 cm of H_2O. During coronary artery grafting the tidal volume was decreased to 5 ml/kg and respiratory rate increased to 20/min because the left lung was obscuring the surgical field. Post induction arterial blood gas analysis (ABG) showed PaO_2 of 80 mm Hg on FiO_2 of 0.6 with rest of the values in normal range. Intraoperative endotracheal suctioning revealed excessive tracheobronchial secretions, simultaneously FiO_2 was raised to 1.0 and subsequent ABG remained within normal limits.

Major parts of the procedure remained uneventful. However at the time of grafting of proximal ends of saphenous vein to the aorta we observed a partial collapse of the left lung and the ventilator bellows not inflating fully. This raised a suspicion of a leak in the breathing circuit but we did not find any circuit leak. An attempt was made to auscultate the chest but was not successful because the patient was draped with sterile towels. The anesthesia machine was cross-checked by a biomedical engineer but no technical error was detected. The inspiratory gas flow was increased to 3.0 liter/minute resulting in adequate expansion of the left lung, restoration of tidal volume and full inflation of ventilator bellows. Bulging of right side pleura was not observed on inspiration.

During chest closure the heart rate (HR) increased to 130 beats/min with decrease in arterial blood pressure (ABP) to 80/60 mm Hg without any significant change in PIP, pulmonary artery pressure (PAP), 22/14 mm Hg and central venous pressure (CVP), 9 mm Hg. It was thought that the hemodynamic instability was due to the effect of chest closure and therefore was managed by administering fluid boluses and titrated increase in the dose of norepinephrine and epinephrine.

At the end of the procedure, when all drapes were removed and dressing had been applied on the surgical wound while the patient was still on mechanical ventilation, there was an increase in PIP to 40 cm H_2O. This was immediately followed by increase in HR to 150/min and a decrease in ABP to 60/40 mm Hg. We attempted to manage this episode as the previous one and transesophageal echocardiography (TEE) was called for. However there was no response to intravenous fluids and high doses of vasopressors. An increase in CVP to 14 mm Hg and PAP to 28/20 mm Hg with a decrease in cardiac index (CI) and cardiac output (CO) were observed. Auscultation of the chest revealed decreased breath sounds on the right side of the chest and crepitus was palpable over the neck and chest. This led to suspicion of subcutaneous emphysema and a diagnosis of tension pneumothorax was made. Immediately tube thoracostomy was performed on the right side and a gush

of air bubbles were observed in the under water seal drainage system followed by normalization of heart rate, blood pressure and pulmonary artery pressure. TEE performed subsequently to rule out any signs of myocardial ischemia revealed normal LV function, adequately filled LV, no RWMA and no valvular regurgitation.

The patient was then shifted to the postoperative intensive care unit and a chest radiograph obtained which revealed full lung expansion and mild subcutaneous emphysema. The trachea was extubated on the first postoperative day, chest tubes were removed on the third postoperative day without any evidence of pneumothorax on chest radiograph. Rest of the postoperative period was uneventful and the patient was discharged on the seventh postoperative day.

DISCUSSION

Tension pneumothorax is a life threatening condition and its occurrence intraoperatively should be promptly diagnosed and treated (3, 4). The most common causes are regional blocks (40 % of reported cases), airway instrumentation (19 %), barotrauma (16 %), and placement of central venous lines (7 %) (5). Patients with COPD are at increased risk (3). In our patient the cause of tension pneumothorax was thought to be rupture of an emphysematous bulla that might have been present on the right lung particularly since the chest radiograph showed emphysematous changes despite normal PFT. Moreover the patient was a chronic smoker and during surgery the lungs were observed to be hyperinflated.

A communication between lung parenchyma and pleural space may act as a one way valve allowing air to enter inside the pleural cavity during inspiration but preventing the air from escaping naturally during expiration. This results in an expanding pneumothorax that forces the lungs to collapse, increases intrathoracic pressure that causes decrease in venous return to the heart, decrease in stroke volume, cardiac output, cardiac index, blood pressure and tachycardia eventually leading to hemodynamic compromise (6). McLoud et al. (7) reported a rise in PADP consistent with the development of pneumothorax in 3 patients (2 on mechanical ventilation). Yu and Lee (8) reported an increase only in PADP with pneumothorax and they considered it could be due to the transmission of the intrapleural pressure to the pulmonary vasculature. Connolly (9) reported the first and only description of a patient with tension pneumothorax in whom all hemodynamic and ABG parameters were measured. The authors described the onset of hypoxemia, acidosis, increased CVP, PAP and decrease of CO, consistent with the development of pneumothorax.

Standard medical reference texts state that the immediate life-saving treatment for tension pneumothorax is needle decompression but there are case reports describing patients with tension pneumothorax managed successfully by chest tube drainage, without performing immediate needle decompression (10). Many experts would proceed directly to definitive treatment and bypass the step of needle decompression if the capability to perform tube thoracostomy is immediately available, and this is what we opted for.

Classical signs of pneumothorax may be masked during general anesthesia. In mechanically ventilated patients, the physician may suspect tension pneumothorax when there is an increase in pleural pressures necessitating an increase in peak airway pressure in order to deliver the same TV. Decreased expiratory volumes secondary to air leakage into the pleural space and increased end-expiratory pressure, even after discontinuation of PEEP, are two other

signs of tension pneumothorax in these patients. Increased PAP and decreased CO or CI are other parameters suggestive of tension pneumothorax (6-9). Hemodynamic instability, hypoxia and/or increased oxygen requirements occur within minutes during positive pressure ventilation in comparison to hours during spontaneous respiration (11). In our case there were many signs indicating tension pneumothorax, such as a decrease in expiratory TV followed by increase in PADP, decrease in CO and CI leading to hemodynamic instability and lastly subcutaneous emphysema. However, since most of the signs also indicate hypovolaemia and contractile deficit that may occur frequently during OPCAB, this may lead to delay in diagnosis and treatment of the condition.

We hypothesize that at the time of the first episode, start of a small pneumothorax resulted in reduction in minute volume without hemodynamic or airway pressure changes.

The pneumothorax slowly progressed resulting in the second episode which was associated with hemodynamic changes but since the chest was still partially open the hemodynamics could be stabilized by fluid administration and inotropic support. However after chest closure the tension pneumothorax caused rapid, profound hemodynamic changes in the now closed chest cavity. We would like to highlight that intraoperative tension pneumothorax may definitely manifest after chest closure in cardiac surgical procedures.

We conclude that the diagnosis of tension pneumothorax remains a challenge in mechanically ventilated patients under anesthesia. The presence of a cardiogenic shock-like picture, poor response to inotropes, increased inspiratory airway pressure, loss of tidal volume in a patient undergoing cardiac surgery may also be due to a tension pneumothorax.

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