

Asystolic Cardiac Arrest, Following Insufflation of Pleural Cavity in Robotic Assisted Thoracoscopic Thymectomy (RATT) - Case Report

Abstract

Severe bradycardia has been previously reported during capno mediastinum, but not asystolic cardiac arrest in robotic assisted thoracoscopic surgery with positive pressure insufflation. We describe the case of a 50 year old female undergoing robotic assisted thoracoscopic surgery for thymectomy, which developed asystolic cardiac arrest intraoperatively after initiating one lung ventilation and docking of the robot. Immediate deflation of the thoracic cavity and undocking of robot with administration of atropine and ephedrine helped her heart rate & blood pressure to revive. Greater awareness of this complication particularly in robotic surgeries which usually takes some time to undock will facilitate early diagnosis and encourage preventive measures. We here discuss the possible mechanisms of haemodynamic instability with positive pressure insufflation and propose certain preventive and early corrective measures, specifically when robot is already docked, so as to reduce the related complications.

Keywords: Anaesthesia; Cardiac arrest; Capno mediastinum; Robotic assisted thymectomy

Case Report

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Introduction

The introduction of positive pressures in the pleural cavity during thoracoscopy is associated with respiratory and hemodynamic perturbations. Most of them range from minimal clinically significant untoward effects to severe haemodynamic compromise but asystolic arrest has not been reported previously with robotic assisted thoracoscopic surgeries. We report a case of a patient who underwent right thoracoscopic thymectomy for myasthenia gravis with one lung ventilation and during the initial part of the procedure she developed hypotension and asystole.

Case Report

A 50 year old female, known case of myasthenia gravis was scheduled for thoracoscopic thymectomy. The patient had history of hypertension which was controlled with oral amlodipine 5 mg once daily doses. Her myasthenia gravis status was controlled on medications including anticholinesterases, azathioprine, and steroids. She had no residual shortness of breath or limitation of activity. She was suffering from significant gastro-esophageal reflux disease for which she was taking oral pantoprazole 40 mg once daily. She did not report any allergies. Her physical examination findings were within the normal range. Laboratory reports like haemograms, renal function tests, liver function tests, chest x-ray, electrocardiogram and echocardiogram were otherwise unremarkable except neurological examination confirmed a definite decremental pattern in repetitive peripheral nerve stimulation test and presence of IgG autoantibodies to acetylcholine receptors. Computed tomography of chest (CT scan) showed a homogenous opacity of well-defined mass in the anterior mediastinum obscuring the helium.

The medication for myasthenia gravis (anticholinesterases, azathioprine, steroids) and the associated disorders like reflux esophagitis (pantoprazole) continued till the morning of surgery. As the patient was on steroid therapy, intravenous hydrocortisone 100 mg was given to the patient pre-operatively. Sedative premedication was avoided. The standard monitors including electrocardiogram (ECG), pulse oxymetry (SpO₂), non-invasive blood pressure (NIBP) were attached to the patient once she was brought into the operating room. After securing a intravenous line in a peripheral vein, intravenous fentanyl was administered 1µg/kg. The right radial artery and right internal jugular vein were cannulated under local anaesthesia. Anaesthesia was induced with intravenous propofol 2 mg/kg and neuromuscular blockade was achieved by atracurium 0.05 mg/kg. The trachea was intubated with 35 French left sided double lumen end bronchial tube and correct position was confirmed by fibroptic bronchoscopy. Neuromuscular monitoring (ulnar nerve) and nasopharyngeal temperature were also monitored. Anaesthesia was maintained with isoflurane 1-1.2%, fentanyl (total intra-operative dose 350 µg), atracurium 25 mg and fraction of inspired oxygen (FiO₂) 0.6-1 %. For surgery, patient was positioned supine with 30 degree right up in order to facilitate easy docking of robot. Patient was stable all this period. Left sided one lung ventilation was initiated before putting the trocar into the thoracic cavity with tidal volume of 5-7 mL/kg, respiratory rate 15/min maintaining the intra-operative PaO₂ value (arterial oxygen) more than 100 mmHg. Incision was made only after initiation of one lung ventilation and followed by insertion of ports.

At the time of incision, heart rate was 88 beats/minute, blood pressure was 148/82 mmHg, oxygen saturation 99%. The mean airway pressure was noted as 28.3 mmHg. Patients right hemi

thorax was insufflated with carbon dioxide (carbomediatinum) and robot was successfully docked. After 20 minutes of docking time and 15 minutes of console time of operating surgeon, we noticed there was sudden decrease in heart rate from 86 beats/minute to 62 to 41/minute and finally reduced to 24/minute over a period of around 20 seconds. Simultaneously blood pressure dropped in the same sequential manner from 118/68 mmHg to 60/42 mmHg and to 22/10 mmHg in invasive blood pressure field monitor screen. There was also suddenly increase in mean airway pressure to 37 mmHg and decrease in saturation was from 98% to 72%, at the same time we, experienced some difficulty in bag ventilation also.

This was followed by momentary asystole. The surgeon and the assistant, in particular who was sitting by the side of robotic arm were alerted. We tried to revive heart rate with immediate three bolus doses of atropine of 0.6 mg each and ephedrine bolus dose of 30 mg. One lung ventilation was terminated and 100% oxygen was administered. The surgeons were immediately asked for release of insufflation pressure in order to bring the thoracic cavity pressure to atmospheric pressure. At the same time we noticed that the insufflators showing a pressure of 16mmHg which was neither the set pressure nor the required pressure for thoracoscopic surgery. Then all the robotic ports were removed and virtually the robot was undocked. Then video assisted thoracic surgery (VATS) camera was inserted to know what was happening inside the thoracic cavity. After visualization of thoracic cavity, the opposite pleura seemed to be under tension, as if tension pneumothorax was developed on the opposite side.

Surgeon attempted to open the opposite pleura and insert a chest drain. Patient's vital signs were yet to be completely stabilized as arterial pressure (mean) was less than 35-40 mmHg. Decision to go for an anterior thoracotomy was made as hemodynamics was unstable. Robot was undocked, chest opened and thymectomy was performed as open procedure. Her heart rate and blood pressure returned to normal. In absence of any further arrhythmia or hemodynamic abnormality the surgery was completed. Patient was extubated on table and sent to intensive care unit with clear sensorium, drain was removed the next day, and postoperative chest X-rays were normal. She was kept in the ward for two more days to medically stabilize myasthenia gravis and was discharged on fourth day from the hospital.

Discussion

Carbomediatinum, created by insufflation of the pleural cavity with carbon dioxide, helps surgeon by pushing the deflated lung furthermore, improves the surgical exposure, increases the intra pleural space by shifting the mediastinum away from the surgical field [1-4]. But now a day's most of literatures suggest, there may be risk of haemodynamic compromise takes the upper hand than the adequate exposure, in thoracoscopic surgeries [2-4].

For thoracoscopic surgeries, a pressure of maximum 8-10 mmHg with one lung ventilation is ideal to serve the purpose, with minimal haemodynamic changes. These changes are more marked in right sided carbomediatinum than the left side as it is a low pressure chamber in the cardiovascular system [3-13]. In review literature, the American association of Gynecology

reported an incidence of one in 2500 cases asystolic arrest during laparoscopy [7]. One more case of asystole during laparoscopic Cholecystectomy has been described [8]. One case of asystole following positive pressure insufflation of right pleural cavity during hilar lymphadenectomy is also described [5]. But till date asystole during the robotic assisted thoracic surgery has not been reported. Its management differs from other simple video assisted thoracoscopic surgeries in the sense that the main surgeon is away from the patient and assistant may sometimes be a trained nurse. So the actual reaction time from operating surgeon is sometimes delayed. It also takes time in undocking robot, if it is really required. So in case of a catastrophic event, the response by the surgeon is delayed when robot is used for the entire procedure [14].

There are several possible causes exist for cardio vascular changes in the laparoscopic thoracic surgeries. The physiological effects, created by positive pressure insufflations into the right pleural cavity may be equivalent to effects created by pneumo mediastinum or tension pneumothorax. In our case there might be sudden malfunctioning of the insufflators, which showed the pressure at the time of catastrophic event was 16mmHg which was neither the set pressure nor required pressure for this type of thoracoscopic surgery. It has been also described the possibility of tension pneumothorax during insufflations secondary to subclinical congenital diaphragmatic defect [9]. Cases of pneumothorax or increasing pneumo mediastinum generally presents with hypotension, increase in central venous pressure and decreased venous return. In our case tension pneumothorax that developed, was detected by video assisted camera (VATS) after undocking the robot. At times mechanical pressure by the surgeon on the heart during dissection may cause hypotension with arrhythmias which improves on releasing the pressure.

The exact cause of bradycardia and asystole are not properly established. Increased intrathoracic pressure sometimes could cause direct vagal stimulation, which may lead to bradycardia and asystole. Sometimes in an intact autonomic nervous system patient when experiences hypotension leading to activation of Bezold-Jarisch reflexes. The baroreceptors in aortic arches and carotid sinuses are sensed, which compensate by increasing cardiac contractility. But at the same time cardiac baroreceptors, then in turn sense this high intramural tension and induce significant parasympathetic discharge resulting in bradycardia and asystole [3].

Conclusion

We conclude, insufflation of the thoracic cavity should be reserved primarily in patients with unsatisfactory surgical exposure, even after use of one lung ventilation to deflate the lung.

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