Anesthetic Management in a Patient With Type A Aortic Dissection and Superior Vena Cava Syndrome

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Introduction: Induction of general anesthesia in patients with superior vena cava (SVC) syndrome may cause airway obstruction and cardiovascular collapse.

Case Presentation: Herein, we introduced a patient with the diagnosis of dissecting aneurysm of the ascending aorta who was candidate for emergency surgery. He also had symptoms of SVC syndrome. To maintain airway patency during anesthetic management, we decided to perform femoro-femoral cardiopulmonary bypass followed by general anesthesia and tracheal intubation.

Conclusions: Femoro-femoral bypass prior to initiation of sternotomy is a safe and easy method in patients with aortic dissection and SVC syndrome in whom earlier endotracheal intubation may not be feasible.

Keywords: Aneurysm; Aortic; Syndrome; Chest

1. Introduction

Anesthetic management for surgery in aortic dissection requires a smooth and deep induction so that prevention of any increase in blood pressure and heart rate during anesthesia is desired. Therefore, full muscle relaxation and inhibition of airway reflexes during tracheal intubation is the preferred way of anesthesia in these patients. Herein, we reported a patient with type A aortic dissection along with superior vena cava (SVC) syndrome who underwent emergency surgery.

2. Case Presentation

A 45-year old man was admitted to the emergency department of our hospital with chief complaint of chest pain. The pain was retrosternal and initiated three days previously, getting worse at the day of admission. Dyspnea as well as head and neck congestion have been superimposed gradually. His medical history included aortic valve replacement (in 1980) due to aortic insufficiency. He was receiving digoxin and warfarin therapy. His vital signs included a blood pressure of 110/70 mmHg, a heart rate of 92 beats per minute, a respiration of 24/minutes and oral temperature of 37.4°C. Edema was evident in his head and neck. The rest of physical examination was otherwise unremarkable. Electrocardiogram showed non-specific ST segment and T-wave changes and chest X-ray revealed mediastinal widening (Figure 1). Echocardiography demonstrated an aneurysm of the ascending aorta with a diameter of 7.3 cm along with a flap, suggestive of acute aortic dissection. There was also mild mitral regurgitation and left ventricular ejection fraction was 50%. Due to the emergency condition of patient, transesophageal echocardiography was not performed. Initial laboratory data were as follows; white blood cell count: 12400 /mm³, hemoglobin: 12.1 gr/dL, hematocrit: 31%, platelet count: 235000 /mm³, Na: 138 mEq/L, K: 4.3 mEq/L, blood urea nitrogen: 15 and creatinine: 1 mg/dL.

The patient was taken to the operating room with the diagnosis of dissecting aneurysm of the ascending aorta (DeBakey Type I and Stanford Type A) along with SVC syndrome. Due to the presence of superior vena cava syndrome, induction of anesthesia with its traditional and usual way was not considered from the beginning; the assessment of the airway was not performed. Even if the airway assessment had showed possibility of endotracheal intubation, sedative and muscle relaxant medications could not be administered to the patient.
Peripheral venous lines in both upper and lower extremities and arterial line through left radial artery were inserted. Slight sedation was made with 50 µg of fentanyl and 1 mg of midazolam, and under local anesthesia arterial and venous cannulas were inserted via femoral access to perform cardiopulmonary bypass (CPB). After ensuring the possibility of CPB, the patient underwent a complete femoro-femoral CPB using anesthetics, including etomidate 0.2 mg/kg, sufentanil 50 µg and cisatracurium 0.2 mg/kg. Then, a laryngoscopy and intubation was performed. Finally, the patient underwent a 7.5 hour surgery for repairing the aortic dissection. The times taken for CPB and aortic cross-clamp were 240 min and 50 min, respectively.

Patient underwent a successful Bentall operation. Post-operation intubation time was 8.5 hours and extubation was performed without difficulty. After eight days with an uneventful postoperative course, patient was discharged. The symptoms of SVC syndrome had been completely resolved at the time of discharge. However, patient returned after one month with a re-dissection. Unfortunately, the second operation was not successful and the patient died.

3. Discussion

Acute aortic dissection is a medical emergency and diagnostic and therapeutic evaluation of these patients should be performed simultaneously. The primary mortality varies between 3%; when the surgery is performed urgently and 20%; when preoperative evaluations take too long and performing diagnostic tests delay the surgery. Acute control of patients would be based on relieving pain and decreasing blood pressure with antihypertensive medications. Chest radiography is usually the initial imaging modality. Mediastinal widening is the most common abnormality seen in approximately 80% of patients. Double aortic knob sign, tracheal displacement to the right, pleural effusion due to leakage of the blood to the pericardial sac and pleural effusion (mostly on left) are other radiographic findings in type A aortic dissection. However, no signs of aortic dissection may be evident on chest X-ray. Computed tomography (CT) with intravenous contrast agents can be used in hemodynamically stable patients. The sensitivity and specificity of CT angiography for diagnosis of aortic dissection are 87-94% and 92-100%, respectively. Magnetic resonance imaging is an accurate tool for the diagnosis of aortic dissection with sensitivity and specificity of both more than 90%.

The anesthesia for surgery of the ascending aorta aneurysm, particularly dissecting type, requires very exact considerations. While preventing the myocardial depression by anesthetics, any sudden changes in blood pressure especially an increase, are very dangerous and by expansion of the dissection area and bleeding death may ensue. Therefore, during induction of anesthesia in these patients, any stimulation should be avoided and we need a deep and smooth induction.

The SVC syndrome is mainly a clinical diagnosis and is defined as a constellation of symptoms resulting from impairment in draining blood from the superior vena cava to the right atrium (1). Increased venous pressure leads to dilation of collateral veins in the thorax and neck leading to edema and cyanosis of the face, neck and upper chest, edema of the conjunctiva and evidence of increased intracranial pressure. Dyspnea is the most common symptom in 63% of patients. Other signs and symptoms suggesting the diagnosis of this syndrome include cough, edema of face, chest pain, dysphagia, orthopnea, edema of arms, head fullness, distorted vision, hoarseness, stridor, headache, nasal obstruction, nausea, pleural effusion and lightheadedness. The findings on physical examination are dilated thoracic and neck veins, face and upper extremities edema, and tachypnea, and seldom cyanosis, Horner syndrome, paralysis of vocal cords and symptoms of increase in intracranial pressure (1). Edema of the arms and face is usually prominent. Hoarseness may reflect edema of the vocal cords. Increased intracranial pressure is most likely due to increased cerebral venous pressure and usually manifests as nausea, seizure and decreased level of consciousness. A large number of underlying diseases can cause this syndrome. More than 80% of the cases result from malignant mediastinal tumors. In 75-80% of cases, the underlying disease is bronchogenic carcinoma that is usually of small cell carcinoma type (2). A non-malignant cause of this syndrome can be the thrombosis resulting from intracaval catheters of pacemaker wire (3). Furthermore, due to the anatomy of SVC, when the lymph nodes in this area or the ascending aorta are enlarged, this vein may be compressed resulting in a decrease in the blood flow and
hence complete obstruction may even occur. However, several rare etiologies have been reported. Fukui et al. reported a case of SVC syndrome caused by a chronic dissecting aortic aneurysm after aortic valve replacement (4). Pseudoaneurysms of the ascending aorta or aortic arch-traumatic, infectious or iatrogenic-have also been reported as causes of SVC syndrome (5-8). A rare case of SVC syndrome has been reported in a 8-year old girl as a consequence of long-term use of central venous catheters for hemodialysis (9).

Although SVC syndrome is a clinical diagnosis, imaging modalities can help in the establishment of predisposing factors. Anesthesia considerations in SVC syndrome due to aortic diseases are similar to SVC syndrome in mediastinal tumors. During induction of anesthesia, the airways obstruction is the most common and dangerous complication (10). An ongoing expansion of a thoracic aortic aneurysm can compress the surrounding structures including the esophagus, superior vena cava or innominate vein and large airways. Moreover, compression of the left main bronchus, left pulmonary artery, trachea or mainstem bronchus has been reported (11, 12). As a result, these patients should be carefully examined and evaluated before surgery. Computed tomography scan (CT) of the chest can demonstrate the precise location of the thoracic aortic aneurysm and its anatomical relation with adjacent structures (13). However, the airways may appear normal in initial CT scan, but may be obstructed after the induction of anesthesia. Moreover, severe hypoxia may occur in these patients due to pressure on the large veins while the airways are patent (14). Induction of general anesthesia tends to exacerbate extrinsic airway compression by decreasing lung volumes and relaxing bronchial smooth muscle. It is further exaggerated by neuromuscular blockade and positive pressure ventilation, which eliminate normal transpleural pressure gradients and subsequently cause narrowing of large-caliber airways (15). The presence of clinical symptoms is also important in the assessment of these patients as any history of dyspnea or cough while lying down can suggest the probability of airway obstruction during induction of anesthesia (10). In the management of anesthesia in symptomatic patients, premedication with sedatives should be avoided. Opioids and benzodiazepines can suppress the respiration depending on the dosage. On the other hand, benzodiazepines would cause muscular relaxation and increase the obstruction of airways by increasing the collapse of airways in large mediastinal tumors (14). Impaired venous drainage causes tongue swelling and laryngeal edema making intubation potentially difficult. All sizes of tracheal tubes and rigid bronchoscope should be available. The most important point to prevent airway obstruction and cardiopulmonary collapse is keeping the spontaneous respiration. Management of airways for a general anesthesia in these patients can be performed through different methods such as using inhalational agents, awake intubation or femoro-femoral cardiopulmonary bypass.

In severe life-threatening hypoxic cases due to obstruction of airway or pressure on pulmonary arteries as well as cases in which general anesthesia is unsafe such as symptomatic adults and children and asymptomatic adults whose minimum tracheobronchial diameter in CT is less than 50% of the normal rate, maintenance of oxygenation using femoro-femoral cardiopulmonary bypass gained a final success (10). Sendagupta et al. described a successful experience in the management of a patient with SVC syndrome due to a large anterior mediastinal mass by anaesthetizing the patient through a femoro-femoral cardiopulmonary bypass (16).

Standby CPB during induction of anesthesia is very dangerous, because after the occurrence of sudden collapse of the airway, there would not be sufficient time to prevent complications of cerebral hypoxia (10). In our case, considering concomitant presence of acute aortic dissection with SVC syndrome, as well as the special anesthetic considerations in each case, we encountered a big challenge in general anesthesia and management of airway. We did not have enough time to perform a chest CT imaging to precisely determine the anatomy of airways and also the degree of severity of SVC compression to decide for proper anesthetic management. Even if preoperative clinical assessment had not shown obvious extrathoracic airway narrowing, sedatives and muscle relaxant medications could not be administered to patient and proceeding to endotracheal intubation prior to establishment of sufficient arterial oxygenation through cardiopulmonary bypass could put our patient at potentially life-threatening risks, as described above, which were not acceptable with respect to his young age. We encountered an enlarging dissection of the ascending aortic aneurysm, which might also expand in retrograde direction to involve the aortic root and cause aortic valve rupture and thus any delay for further evaluation or workup could be lethal. Therefore, we decided to initiate femoro-femoral CPB immediately and after establishment of sufficient oxygenation, general anesthesia was inducted and tracheal intubation was undertaken.

In conclusion, we showed in the present case that femoro-femoral bypass prior to initiation of sternotomy is a safe and easy method in patients with aortic dissection and presentations of SVC syndrome in whom earlier endotracheal intubation may not be feasible.

References


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