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

Ziae Totonchi¹; Nader Givtaj²; Mozghan Sakhaei¹; Afshin Foroutan¹; Mitra Chitsazan³*; Mandana Chitsazan⁴; Hamidreza Pouraliakbar⁵

¹Department of Cardiac Anesthesiology, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran
²Department of Cardiac Surgery, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran
³Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran
⁴Shahid Beheshti University of Medical Sciences, Tehran, IR Iran
⁵Department of Radiology, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran

*Corresponding author: Mitra Chitsazan, Rajaie Cardiovascular Medical and Research Center, Vali-Asr St., Niayesh Blvd, Tehran, IR Iran. Tel: +98-9122210385, E-mail: mitra.chitsazan66@yahoo.com

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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. Postoperation 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 diagnostically 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 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 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 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 upper chest, edema of the conjunctiva and evidence of increased intracranial pressure.
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Moreover, severe hypoxia may occur in these patients
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due to pressure on the large veins while the airs 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, benzodi
azepines would cause muscular relaxation and increase
the obstruction of airways by increasing the collapse
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t 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 per
formed through different methods such as using inha
lational agents, awake intubation or femoro-femoral
cardiopulmonary bypass.

In severe life-threatening hypoxic cases due to obstruc
tion 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 oxy
genation using femoro-femoral cardiopulmonary bypass
achieved a final success (10). Sandagupta 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 dan
gerous, because after the occurrence of sudden collapse
of the airway, there would not be sufficient time to pre
vent complications of cerebral hypoxia (10).

In our case, considering concomitant presence of acute
aortic dissection with SVC syndrome, as well as the spe
cial anesthetic considerations in each case, we encoun
tered a big challenge in general anesthesia and manage
ment 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 com
pression 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 oxygena
tion through cardiopulmonary bypass could put our patient
potentially life-threatening risks, as described above,
which were not acceptable with respect to his young age.
We encountered an enlarging dissection of the ascend
ing aortic aneurysm, which might also expand in retro
grade 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 initi
ate femoro-femoral CPB immediately and after establish
ment of sufficient oxygenation, general anesthesia was
inducted and tracheal intubation was undertaken.

In conclusion, we showed in the present case that fem
oro-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 en
dotracheal intubation may not be feasible.

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