Concomitant Persistent Atelectasis following TEVAR Due to a Descending Aortic Aneurysm: Hybrid Endovascular Repair and ECMO Therapy

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INTRODUCTION

Many thoracic aortic aneurysms are discovered incidentally, and most develop without symptoms. Symptoms are usually due to sudden expansion of the aneurysm, which can cause a vague pain in the back, or sometimes a sharp pain that may denote the presence of impending rupture. Other symptoms are related to pressure on adjacent structures, such as pressure on the bronchus that can cause respiratory distress, or pressure on the laryngeal nerve causing vocal hoarseness. Pressure on the esophagus can cause difficulty in swallowing.

Currently, open surgery and thoracic endovascular aneurysm repair (TEVAR) are the choices of treatment for descending thoracic aneurysms (DTA). The decision to intervene on a DTA depends on its size, location, rate of growth and symptoms, and the overall medical condition of the patient. The indications for TEVAR should not differ from those for open surgery and typically include aneurysms larger than 6 cm in diameter. Saccular and symptomatic aneurysms are often repaired at a smaller size. It is also suggested that aneurysms with a growth rate more than 1 cm per year, or 0.5 cm in 6 months should be considered for early repair.

Despite the close proximity of the aorta and left main bronchus, atelectasis caused by thoracic aortic aneurysms is rare. We review the case report of a patient with concomitant persistent left pulmonary atelectasis causing acute respiratory distress due to complete compression of the left main bronchus after TEVAR of a descending thoracic aortic aneurysm.

CASE REPORT

A 70-year-old, hypertensive male patient with chronic obstructive lung disease was admitted to the emergency service because of back pain and dyspnea for 10 days. Plain chest X ray revealed a mass at the left superior lobe, subtotal atelectasis of the left lung, and a mediastinal shift to the left. With a preliminary diagnosis of pulmonary malignancy, thoracic CT revealed a ruptured 9 cm diameter saccular aneurysm of the descending thoracic aorta, distal to the origin of the left subclavian artery causing compression on the left main bronchus (Figure 1). The patient was transferred to the Cardiovascular Surgery ICU.

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The patient's vital and hemodynamic data were stable. Cardiac Doppler ultrasonographic findings were in physiological limits with a 50% ejection fraction. Serum laboratory findings revealed hemoglobin of 9.7 g/dL, hematocrit 38%, blood urea nitrogen (BUN) 27 mg/dL, and creatinine 1.0 mg/dL. Vital signs showed heart rate of 110 (sinus tachycardia) bpm, blood pressure 138/84 mmHg in both arms, and arterial blood gases in room media in normal limits. Hemodynamic and laboratory data did not suggest any bleeding at the moment. Considering the overall medical condition of the patient, emergency TEVAR was planned. The patient was transferred to the catheter lab urgently, and TEVAR was performed under general anesthesia. The procedure was performed successfully without any complication (Figure 2).

Figure 1. Ruptured 9 cm diameter saccular aneurysm of the descending thoracic aorta, distal to the origin of the left subclavian artery causing compression on the left main bronchus: (a) sagittal view; (b) axial view; (c) coronal view.

Figure 2. TEVAR performed successfully without any endoleak.

Arterial Blood Gas Analysis

Two hours later following TEVAR at the Cardiovascular Surgery ICU, despite mechanical support with a FIO₂ 100% and PEEP of 22 cm H₂O, deep hypoxemia (PO₂ 51 mmHg, PCO₂ 45 mmHg) developed, progressively worsening as respiratory distress developed. Chest X ray revealed total atelectasis of the left lung. While immediate bronchoscopy was carried out, the patient deteriorated. The patient was immediately connected to veno-venous (V-V, right jugular and femoral vein cannulation) ECMO. Repeat bronchoscopy revealed an open left main bronchus, but obstruction of the distal airway a few centimeters ahead due to mucous plugs. The bronchoscope could not be pushed forward in to the viscous plugs and secretion. The plugs and viscous secretion were washed up with intermittent approaches. At postoperative day 15 the patient was weaned from ECMO, and was extubated 48 hours later. Arterial blood gas analysis of the patient following V-V ECMO pulmonary assist therapy is shown in the Table. Following a recovery period of one week the patient was discharged from the hospital. At present, the patient has had an event-free follow up period of 13 months. The radiographic scenes are shown in Figure 3.

DISCUSSION

Sixty percent of thoracic aortic aneurysms (TAA) are located at the ascending, 40% at the descending, 10% at the arcus, and 10% at the thoracoabdominal aorta. It has been reported that the natural growth rate of the TAA is approximately 0.1 cm per year [Isselbacher 2005], and rupture probability in aneurysms that are larger than 7 cm in diameter is 43%. Generally it is accepted that TAAs larger than 6 cm in diameter should be considered for early repair [Jonker 2010]. It has been estimated that the incidental mortality rate of rupture of the TAA in the first 6 hours is 50%, and 25% in the next 24 hours [Barbato 2006]. The main goal of TEVAR in TAAs is to reduce the pressure increase in the aneurysmal sac/tube, thus reducing the rupture rate and mortal hemorrhage [Dias 2004]. Currently, open surgery and TEVAR are the choices of treatment for DTA. The decision to intervene on a DTA depends on its size, location, rate of growth and symptoms, and the overall medical condition of the patient. The indications for TEVAR should not differ from those for open surgery.

Recently, in a nationwide risk adjusted study on 923 patients, it has been reported that TEVAR has a significant advantage in mortality rates over open surgery in ruptured DTA (rDTA) (23.4% versus 28.6%). It has been emphasized that multivariate analysis revealed that the size or extent of the medical center was an independent factor in predicting the outcome of the procedures. In another meta-analysis similar hospital mortality rates have been reported (19% versus 33%). The overall major complications (acute myocardial infarction, stroke, paraplegia) in the endovascular group have also been reported to be significantly less than in the open surgery group. In the TEVAR group, a three-year symptom free survival rate of 71% was reported [Gopaldas 2011; Jonker 2010]. The overall medical condition of the patient in this case, and the comorbidities which were present, led us to endovascular repair of the rDTA.

The rise in CRP, leukocytosis, and fever following the endovascular repair may have been an inflammatory response due to the aortic stent. Similarly, the occurrence of pleural effusions, periaortic changes, and despite no endovascular leak the increase in the diameter of the aneurysm may also have been due to this response; which resides without any specific treatment. It has been estimated that in approximately 73% of the endovascular repairs pleural effusions occur and in 33% of cases periaortic changes occur respectively. It has been demonstrated tomographically that in the first ten days of endovascular repair the aortic aneurysm diameter increases in 32% of patients, while in half of patients any change in the aneurysm diameter has not been detected. In another subgroup of 16% of patients, a decrease in the diameter of the aneurysm was detected [Schoder 2003; Sakai 1999; Ishida 2007].

Figure 3. (a) Chest X ray before TEVAR; (b) 90 minutes after TEVAR; (c) discharge day.

Recently, the effect of stent grafts on aneurysm shrinkage in 1450 endovascular abdominal aortic repairs without endovascular leak has been reported. According to the investigators, effective intrasaccular pressure and diameter reductions occur in a mean of postoperative 13 months [Cieri 2012]. Currently, there is no general consensus on intrasaccular pressure changes and vascular remodeling after endovascular repair of abdominal aortic aneurysms. There are various results which have been reported by different clinical studies [Kwon 2011].

In our case, we expected a relief on the compressive force on the left main bronchus following TEVAR, but instead deep hypoxia and cardiopulmonary collapse due to atelectasis developed. Because of the sudden deterioration of the patient and the excessive compression on the left main airway, an effective bronchoscopic intervention was not possible. This could be due to a bulging because of an early periaortic reaction resulting in an increase of intrasaccular pressure and/or diameter.

Mechanisms of atelectasis in the perioperative period are studied and understood quite well. Atelectasis following general anesthesia occurs in 90% of patients, and in 15-20% it happens before the operation has started, and can last for many postoperative days. Advanced chronic obstructive lung disease and present perioperative atelectasis could be the main reason for the abruptly progressing respiratory distress. The persistent atelectasis following the postoperative second day suggests to us such a mechanism. Despite the close proximity of the aorta and left main bronchus, atelectasis caused by compression of the thoracic aortic aneurysms is rare. The treatment of pulmonary atelectasis from compression of the left main bronchus by an aortic aneurysm with endobronchial stenting is still a topic of controversy. Silicon, metal, or hybrid endobronchial stents are available at the market but the long term follow-up results are still controversial [Yap 2009; Hedenstierna 2010].

Extracorporeal membrane oxygenation (ECMO) allows prolonged extracorporeal circulation in the intensive care unit (ICU). ECMO uses classic cardiopulmonary bypass technology to support circulation. It provides continuous, non-pulsatile cardiac output and extracorporeal oxygenation. Veno-venous ECMO (VV ECMO) provides respiratory support, while veno-arterial ECMO (VA ECMO) provides cardiorespiratory support to patients with severe but potentially reversible cardiac or respiratory deterioration refractory to standard therapeutic modalities. However, recent technological advances in the ECMO circuit have led to a reduction in the rate of technical issues and complications. Moreover, improved understanding of the benefits of ECMO has emerged from its widespread use as rescue therapy for patients with ARDS and refractory hypoxemia. When the cardiac function is preserved, VV-ECMO is used to improve gas exchange. Traditionally, VV-ECMO required the insertion of at least two cannulae in large veins (jugular, femoral, or both), sometimes more to facilitate drainage and flow and therefore oxygenation [Lafçı 2013; Schmid 2002; Martinez 2012].

Until recently, emergency open surgical repair of rupture of DTA was the preferred method of treatment. But caution

should be exercised in open surgery in a group of patients for whom transthoracic graft replacement transthoracic aorta portends high mortality and morbidity. Advances in endovascular techniques have enabled nonsurgical treatment of intrathoracic aortic aneurysms. Comorbidities like bleeding complications of rupture or dissection, renal failure, chronic obstructive lung disease, ischemic heart disease, and advanced age are predictors of bad news. Currently experience with endovascular treatment is encouraging. ECMO can be used in adult patients with ARDS or cardiogenic shock who fail to respond to conventional therapies. In our patient, despite aggressive mechanical support and bronchoscopic intervention, progressing hypoxia and circulatory collapse could not be overcome. We believe that, although the evidence in favor of ECMO for ARDS treatment is not strong enough to make a general recommendation, it should be considered when other therapies fail. A hybrid therapy mode of endovascular aneurysm repair and ECMO could be applied to patients, especially those with multiple comorbidities.

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