SPONTANEOUS AORTOESOPHAGEAL FISTULA AND RUPTURED AORTIC ANEURYSM – A CASE REPORT ON COMBINED AORTIC AND ESOPHAGEAL PROSTHESIS PALLIATIVE TREATMENT

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Abstract

Aortoesophageal fistulas are uncommon, dreadful vascular events, most frequently found in the setting of thoracic aorta aneurysms. Patients usually present with thoracic pain, dysphagia and sentinel hematemesis - the Chiari triad - followed by life threatening hematemesis. Emergent open surgery with debridement of necrotic tissue and in situ aortic graft repair is currently the best strategy. However, in patients which cannot withstand surgery, endovascular repair is currently gaining acceptance as a palliative treatment or as a bridge to surgery. We present a case of a 55-year-old female with a past of heavy alcohol abuse and a previously unknown massive aortic aneurysm, who presented to the emergency department complaining of acute dysphagia and epigastric pain. An abdominal ultrasound revealed left pleural effusion and suspected clots in the pleural space. A thoracic CTA was promptly done, where a spontaneous ruptured aortic aneurysm with aortoesophageal fistula was discovered. The team, fearing open surgery due to poor cardiac function, opted for a thoracic endovascular aortic repair. The aortoesophageal fistula dissected the esophageal wall in all of its thickness without rupture into the lumen. This was complicated with esophageal ischemia, aneurysmal sac infection and mediastinitis. Because the patient was in shock, in order to help control the infection, an esophageal prosthesis was placed, followed by proximal esophagostomy, distal esophageal closure and gastrostomy. Six months after initial presentation, the patient died at the emergency room, shortly after reentering with massive hematemesis and hypovolemic shock of undetermined origin.

CASE REPORT

Aortoesophageal fistulas are unusual thoracic emergencies, where a communication between the descending aorta and the esophagus is established. In more than 50% of cases they occur spontaneously in the setting of an aortic aneurysm and are mostly asymptomatic until presentation.[1] Other common causes are generally secondary to foreign body perforation of the esophagus, esophageal malignancy, iatrogenic perforation of the aorta during endovascular repair or perforation of the esophagus during endoscopic procedures.[1,2,3] Other less common causes include infections such as syphilitic or tuberculous aortitis, lung cancer, chemical ingestion, gastroesophageal reflux, trauma, and gunshot wound.[1,2,3]

These patients can present with the triad described by Chiari in 1914, which comprises mid thoracic pain, dysphagia and a sentinel hematemesis, followed by an interval period from hours to days before torrential hematemesis and frequent exsanguination.[1,4]

If hematemesis is controlled, the patients are still at risk for developing sepsis due to esophageal lesion or mediastinitis.[3] Hence, emergent repair is vital. Currently three groups of patients are defined: the low comorbidity patients with or without sepsis signs, the high surgical risk patients that show no septic signs and, in the third group, those who would not tolerate surgery and are with overt sepsis signs.[2,3,5,6] The first group is generally managed with open surgical replacement of the aortic defect/aneurysm, esophageal defect closure or debridement and bowel graft, and omental pedicle graft in both instances. This last step contributes for improved lymphatic drainage and infection prevention. The second group is preferably treated with endovascular aneurysm

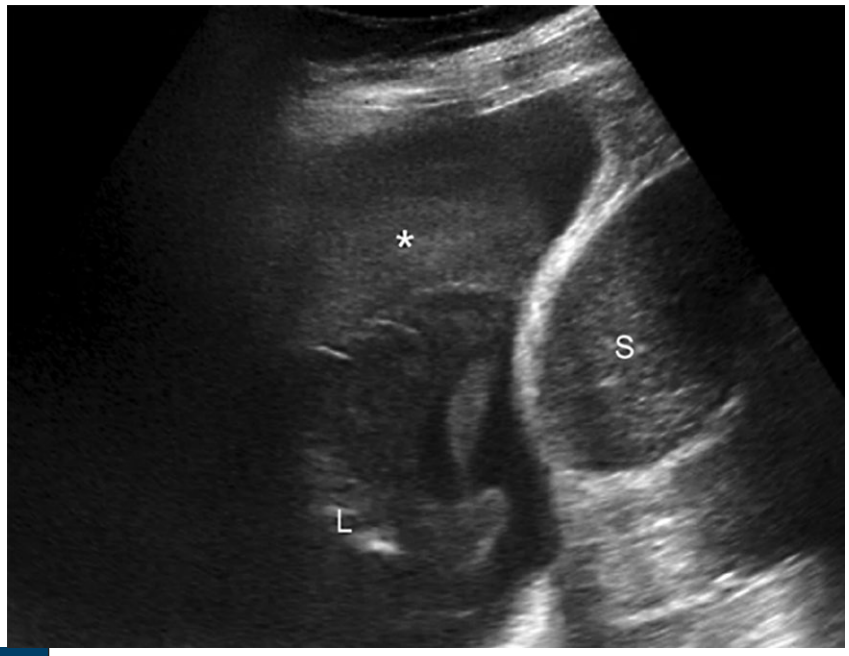

Figure 1

B-mode abdominal ultrasound over the left hypochondrium (coronal intercostal plane). In the pleural space, there is fluid and an echogenic collection (*), compatible with a clot. Notice the collapsed lung (L) and the diaphragm bordered by fluid and the Spleen (S).

repair (EVAR) as a bridge for open surgery. For the third group the treatment is considered palliative, with EVAR placement, esophageal endoscopic prosthesis if significant necrosis and lifelong antibiotics.[2,3,5,6]

We present a case of a 55-year-old female with a past of heavy alcohol abuse and a previously unknown massive aortic aneurysm, presented to the emergency department complaining of acute dysphagia and epigastric pain. On blood analysis there was no elevation of inflammatory markers and her hemogram was unremarkable besides mild anemia (10g Hgb/dL) with acute characteristics.

An abdominal ultrasound revealed left pleural effusion, with partial collapse of the left lung and low echogenicity amorphous material disperse in the pleural space (Figure1).

Suspecting the presence of clots in the pleural space, a thoracic computed tomography angiography was promptly done, where a primary aortoesophageal fistula and a ruptured aortic aneurysm with left hemothorax were discovered (Figure 2A). The aortoesophageal fistula dissected the esophageal wall without rupture into the lumen (Figure 2B).

Due to poor cardiac function, fearing open

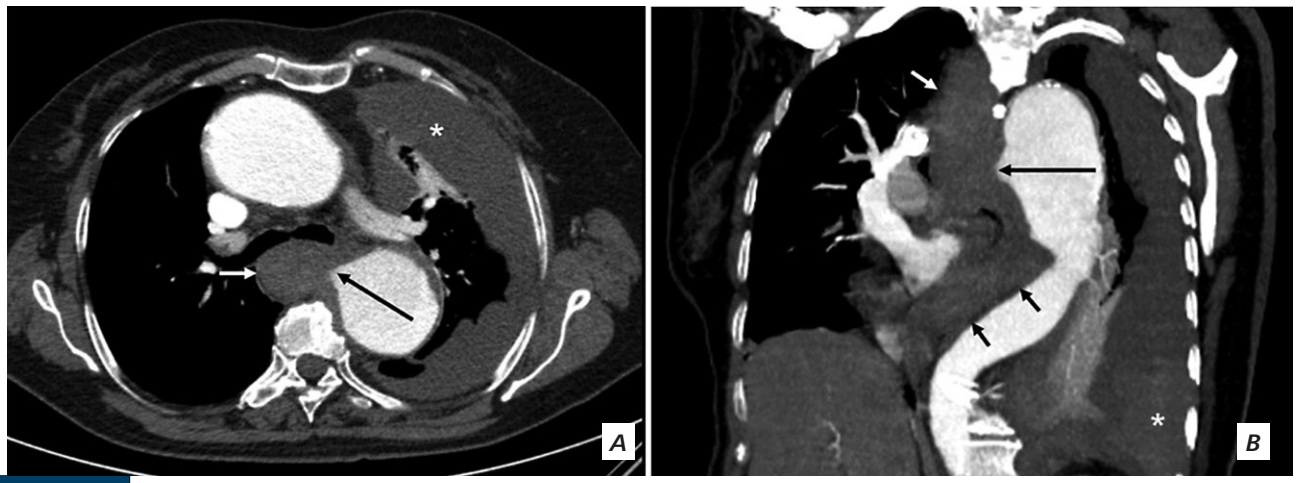

Figura 2

*Contrast enhanced thoracic CT (arterial phase) in the axial (**A**) and coronal oblique (**B**) planes, representing the aorto-esophageal fistula and hemothorax. The large arrows point to a "beak" shaped communication between the descending aorta and the diffusely thickened esophageal wall (small arrows). The esophagus density is homogeneous and the typical wall stratification is lost. Notice the high density pleural fluid (*) at a nondependent position, compatible with a clot secondary to hemothorax.*

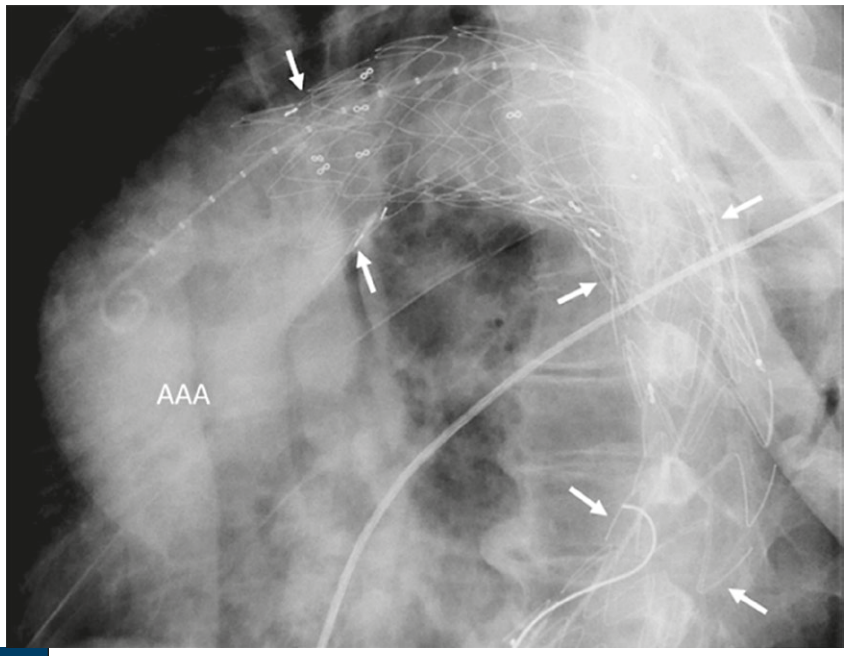

Figure 3 *Angiographic image demonstrating successful endovascular aneurysm repair (EVAR). The prosthesis can be easily identified by the high attenuation of the metallic mesh (between arrows). AAA – Ascending aorta aneurysm.*

surgery, the team opted for a thoracic endovascular aortic repair (Figure 3).

On the following days, esophageal ischemia, aneurismal sac infection and mediastinitis developed (Figure 4A). In order to help control the infection, an esophageal prosthesis was placed (Figure 4B), followed by proximal esophagostomy, distal esophageal closure and gastrostomy.

Six months after initial presentation, the patient reentered the emergency room with massive hematemesis and hypovolemic shock. Despite transfusional support, the patient deceased at the emergency room.

The cause of the bleeding was undetermined but aortoesophageal fistula relapse or esophageal variceal bleeding were suspected.

REFERENCES:

1. Hollander JE, Quick G. Aortoesophageal Fistula: A Comprehensive Review of the Literature. Am J Med. 1991 Sep;91(3): 279-87.
2. Akashi H, Kawamoto S, Saiki Y. Therapeutic strategy for treating aortoesophageal fistulas. Gen Thorac Cardiovasc Surg. 2014; 62:573–580

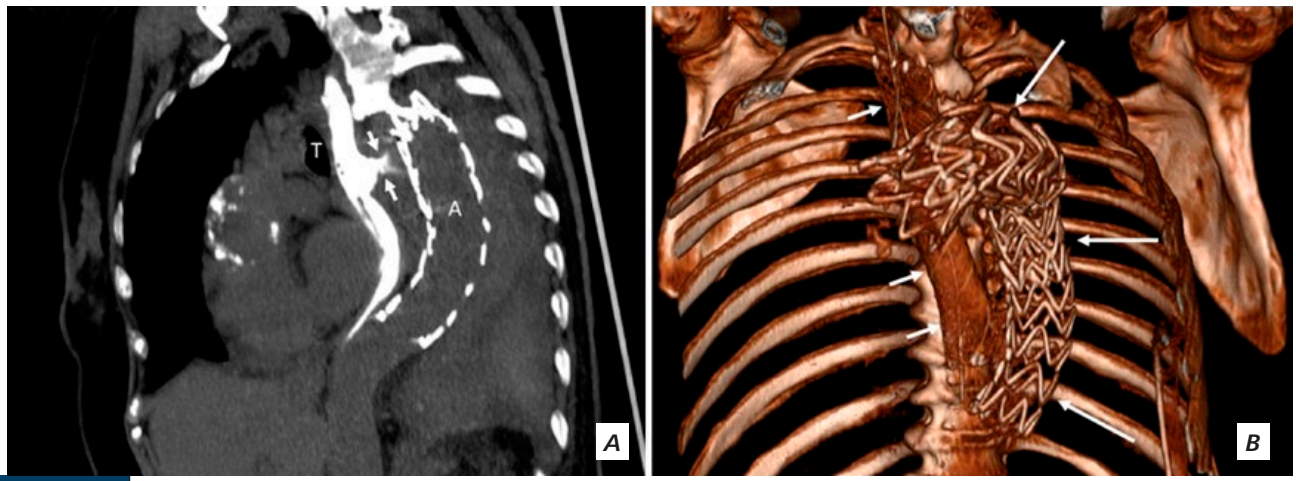

Figura 4 ***A*** *– Nonenhanced thoracic CT with a maximum intensity projection in the coronal oblique plane, after oral contrast, demonstrating a fistulous path from the esophagus to the aneurismal sac (between arrows). The treated aorta is also represented (A). (T) Represents the trachea at the level of the carina.* ***B*** *– Volume rendering technique (VRT) image of the thorax, after the placement of an esophageal prosthesis (short arrows), paralleling the aortic endovascular prosthesis (long arrows).*

3. Prokakis C, Koletsis E, Apostolakis E. Aortoesophageal fistulas due to thoracic aorta aneurysm: surgical versus endovascular repair. Is there a role for combined aortic management? Med Sci Monit. 2008; 14(4): RA48-54
4. Chiari H. Ueber Premdkorperverletzung des Oesopha. Ber Klin Wochenschr. 1914; 51: 7–9. 7.
5. Göbölös L, Miskolczi S, Pousios D, et al. Management options for aortooesophageal fistula: case histories and review of the literature. Perfusion. 2013 Jul;28(4):286-90.
6. Flores J, Shiiya N, Kunihara T, et al. Aortoesophageal fistula: alternatives of treatment case report and literature review. Ann Thorac Cardiovasc Surg 2004 Aug; 10(4): 241-6.