Leaking Thoracic Aneurysm. Does this patient need surgery or ECMO?

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CASE PRESENTATION:

A 35-year-old man presents with a closed right femur fracture after a motor vehicle collision. The fracture requires open reduction and internal fixation. Chest Computed Tomography (CT) reveals tear of the proximal descending thoracic aorta. Laboratory findings show hemoglobin of 9.3 g/dl, hematocrit 37%, blood urea nitrogen (BUN) 24mg/dl, and creatinine 1.0 mg/dl. Vital signs: heart rate (HR) of 100 (sinus tachycardia) bpm and his blood pressure (BP) is 138/84 mmHg in both arms
1. Definition of aortic dissection.
2. How are dissecting aortic aneurysms classified?
3. Is surgical repair indicated on this patient? What is the primary goal on this patient?

Case continues...

Patient is hemodynamically stable. The decision was made to proceed with non-operative management.

Two days later, he develops progressively worsening respiratory distress, accompanied by mental status changes. He is endotracheally intubated. Massive aspiration occurred during the intubation. Soon thereafter patient becomes unstable with acute drop in blood pressure. Bilateral chest tubes placed, draining RT 600 mls and Lt 400 mls of blood.

4. Differential diagnosis?

Surgical team makes the diagnosis of a ruptured aortic dissection and make arrangements for STAT transfer to OR. Cardiac anesthesia called to the bedside. Despite a FiO2 of 1.0 and positive end-expiratory pressure (PEEP) of 20cmH2O, his PaO2 was 50 mmHg. CXRay shows big infiltrate in the right lung compatible with aspiration process. BP 110/70 and HR: 92bpm, still on Esmolol/Nipride infusions and no more blood in the chest tube. The CT surgeon insists in transferring the patient to OR. Let’s reassess the situation!
This patient is dying from hypoxemia and will not tolerate lung isolation for thoracotomy. Initial respiratory failure was due to fat embolism syndrome compounded with aspiration during intubation. Vitals sings are stable on Nipride and Esmolol infusion, no more blood in the chest tubes. Do not repair what is not broken!!!!!!

6. What are the possible treatment options for our patient?
7. What is ECMO?
8. What is the role of ECMO in the management of this patient? Discuss indications.
9. Discuss ECMO Modalities. Is V-A ECMO appropriate? What about V-V approach?

**Case Continue.....**

The decision was made to take the patient to the OR and place him on veno-arterial (V-A) ECMO (femoral vein->right atrium-femoral artery) at 5 L/m. A pulsatile arterial trace was noted. A blood gas sampled from the right radial artery shows a PaO2 of 45 mmHg, and an arterial blood gas drawn from the ECMO inflow cannula reveals a PaO2 of 500 mmHg.

10. What is the MOST likely explanation for these? Findings?

**Discussion:**

In the United States, there are 40 000 motor vehicle death annually, and around of 8000 of the victims had traumatic rupture of the aorta. (TRA)
The definition of aortic dissection is a disruption of the media layer of the aorta with bleeding within and along the wall of the aorta resulting in separation of the layers of the aorta. In the majority of the patients (90%), an intimal disruption is present that results in tracking of the blood in a dissection plane within the media.

There are several aortic dissection classification systems. The DeBakey classification system classifies aortic dissection by their site of origin and distal extension.
Type I: Dissection begins in the ascending aorta valve and extends throughout the aorta down to the common iliac arteries.
Type II: Dissection is limited to the ascending aorta, most common in patients with Marfan syndrome.
Type IIIa: Dissection begins distal to the left subclavia artery and ends in the descending thoracic aorta.
Type IIIb: Dissection begins distal the left subclavia artery and extend into the abdominal aorta. This type rarely requires surgical intervention.

In contrast, the Stanford classification system classifies aortic dissection into two types based on their clinical course and surgical significance:
Type A: Dissection originates in the ascending aorta and includes DeBakey’s type I and type II dissections.
Type B: Dissection originates in the descending aorta and is equivalent to DeBakey’s type III dissection.
Once the diagnosis of acute aortic dissection is obtained, initial management is directed at limiting propagation of the false lumen by reducing the force of left ventricular contraction without compromising perfusion, thus reducing shear forces and preventing further extension of the dissection or possible rupture. Beta-blockers (e.g. esmolol, metoprolol) and labetalol (beta- and alpha-blocker) can be used. If further reduction in BP is required, nicardipine, sodium nitroprusside, glyceryl trinitrate, or hydralazine are appropriate. Beta-blockers should be given before vasodilators, as the reflex catecholamine release due to vasodilatation may increase left ventricular contraction and the concomitant increase in dP/dT. If the patient has a low Glasgow coma scale (GCS <8) or profound hemodynamic instability, intubation and ventilation are indicated.

In acute type B aortic dissections, surgical intervention is only indicated if there is persistent or recurrent intractable pain, aneurysm expansion, peripheral ischemic complications, and rupture. This is because surgical repair has no proven superiority over non-surgical treatment in stable type B dissection patients.

Despite progress in both surgical and intensive care techniques, the outcome of conventional surgical management of acute rupture of the descending thoracic aorta (DTA) is still disappointing. In patients with traumatic aortic rupture, postoperative mortality ranges 8–20% and depends mainly on the severity of associated trauma (pulmonary contusion, head injury, bone fractures, and solid abdominal organ rupture). When aortic repair is performed with cross-clamping alone, the mean rate of postoperative paraplegia is 7%. Use of circulatory assistance can lower the incidence of spinal cord ischemia to 3%, but the need for systemic heparinization increases the risk of fatal
hemorrhage, especially in patients presenting cerebral or pulmonary contusion. The key factor in open repair has been to keep the total aorta cross-clamp time as short a period as possible, preferably less than 45 minutes. Although endovascular stent grafting has not been prospectively studied for this clinical scenario, FDA approved devices are being used “off label”. The problem with endovascular grafting for this type of TRA is the lack of sufficiently small prostheses for use in young patients.

Surgery involving the descending or thoracoabdominal aorta requires a left lateral thoracotomy. One-lung ventilation with a double-lumen endotracheal tube is standard procedure in such cases. A right radial arterial pressure line is essential as the left subclavian artery may be clamped. Femoral arterial pressure is also monitored to ensure adequate perfusion of the lower body.

Our patient initially developed hypoxemia from a fat embolism syndrome. He had a long bone fracture, developed hypoxemia and mental status changes, accompanied by a petechial rash over his upper body. Aspiration at the time of the intubation made things worse.

Many treatment option and strategies are available to treat severe respiratory failure, such as: Modes of ventilation that may improve oxygenation like APRV/inverse ratio ventilation/high frequency ventilation, selective lung ventilation, prone ventilation, Nitric Oxide, hyperbaric oxygen, liquid ventilation and artificial extracorporeal gas exchange such as extracorporeal membrane oxygenation (ECMO) and intravenous oxygenation (IVOX) or extracorporeal CO2 removal (ECCO2R). ECMO was chosen for our patient due to the severity of the hypoxemia.
Extracorporeal membrane oxygenation (ECMO) is an intervention intended to support a patient during reversible life-threatening pulmonary or cardiac failure or both. Heart failure is an indication to place the patient in veno-arterial (V-A) ECMO to provide for oxygenation and adequate perfusion to the body. Cannulas can be placed centrally, usually with the chest open, or peripherally via femoral vessels. Respiratory failure is the indication for Veno-venous (V-V) ECMO, like the patient in the vignette. Cannulas in this configuration are placed via the femoral and/or internal jugular veins (Fig. 1).
In the presence of pure respiratory failure the patient should have been placed on VV ECMO. He was placed on V-A ECMO, and on this situation, there is the risk of ejecting desaturated blood that has passed through the sick lungs. Despite high ECMO flows (maximizing drainage); blood from this patient’s right atrium continues to pass through the diseased lung and eventually is ejected systemically via the healthy left ventricle (Fig. 2).
Figure 2. Veno-Arterial ECMO. Competitive flow between native heart ejection and ECMO arterial flow. Note that the blood coming from the heart is poorly oxygenated and potentially perfusing the arch vessels.

This blood is poorly oxygenated and eventually mixes with fully oxygenated blood delivered from the femoral arterial cannula. Blood sampled in the right arm reflects the low PaO₂ coming from the lung. One clue to this competitive flow phenomenon is that the arterial line has a pulsatile trace. When this happens, clinicians should sample blood from the right radial artery to evaluate potential
desaturation of the ejected blood. The major risk is perfusion of the brain with deoxygenated blood.

The solution to this problem is to maximize venous drainage by placing another venous cannula to diminish pulmonary blood flow with its associated shunt or to switch to V-V ECMO, which should have been the proper indication. Figure 3 and 4

Figure 3. Additional venous drainage line.
Sweep flow regulates CO₂ elimination through the system. The oxygenator sweep flow is the flow of gases passing through the membrane and is the functional equivalent of the minute ventilation in the native lungs. Sweep flows are inversely proportional to the PaCO₂. The PaO₂ of the blood coming out of the oxygenator is regulated by an oxygen blender that determines the FiO₂. Femoral cannulas perfuse oxygenated blood to the upper body in a retrograde pattern as long as the heart is not ejecting a significant amount of blood. In patients with cardiac failure, the weak heart is
unable to eject any significant amount of blood. Hence it will not compete with the retrograde femoral cannula flow and the oxygenation of the upper and lower body are the same. The case in the vignette had pure respiratory failure (with a good heart) and should have been placed on V-V ECMO from the beginning.

References: