Intra-Operatively Diagnosed Acute Ascending Aortic Dissection in a Patient Undergoing Emergency Exploratory Laparotomy for Suspected Bowel Ischemia

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Stem Case and Key Questions Content
A 62-year-old man presented to the Emergency Room (ER) after a brief syncopal episode with right sided weakness. He was given Tissue Plasminogen Activator (tPA) after a diagnosis of Acute Ischemic Stroke (AIS) with negative head and abdominal computerized axial tomographic scans. The patient was transferred to a tertiary care facility for further monitoring and management. After his arrival to the tertiary care facility, the patient developed severe coagulopathy, gastrointestinal bleeding, abdominal pain and hemodynamic instability. Approximately 18 hours after the initial presentation he was brought to the operating room (OR) for emergency exploration of the abdomen under general anesthesia. Upon placement of an ultra sound guided central line bilateral carotids were found to be dissected.

1. What is your differential diagnosis?
2. Is the tPA causing the severe coagulopathy?
3. Stroke Mimics receiving tPA, What are their outcomes compared to their stroke counterparts?
4. Why do patients with aortic dissections develop coagulopathy?
5. What further investigations would you perform while under anesthesia?
6. Whom would you like to consult next?

A Transesophageal Echo (TEE) was performed in the OR. A comprehensive exam revealed a dissection flap in the ascending aorta extending into the arch and descending aorta. The intimal entry tear appeared to be present at the Sino Tubular Junction (STJ). A dissection was
seen in the aortic arch, however the aortic arch was not completely visualized. An immediate cardiac surgery consult was obtained. The bowel was viable with pulses in superior mesenteric artery (SMA) and its branches. A diagnosis of acute ascending aortic dissection (AAAD) was made.

1. What are the most common risk factor for aortic dissections? What is a dissection?

2. What is the prognosis of untreated patients with AAAD?

3. What is the incidence of aortic dissections?

4. What are the consequences of delayed diagnosis in these patients?

5. What are the predictors of death in AAAD?

6. How can you limit the propagation of the dissection?

7. How often is the arch involved in AAAD dissections?

8. If the dissection starts in the arch not involving the ascending aorta, how is it managed?

9. How would you manage the hemodynamics in patients with AAAD?

10. What is the reason for the dynamic symptomatology in AAAD?

11. What should you look for in the TEE exam?

12. Pulse deficits, a new onset diastolic murmur in the aortic area are they of any significance?

13. What are the surgical goals in AAAD’s?

The patient was evaluated by the cardiac surgeon in the OR. Given the ongoing coagulopathy the plan was to proceed with an urgent surgery rather than an emergent surgery. The patient was given plasma products, platelets, and adequately resuscitated. The patient was kept intubated for computerized tomographic scan (CT SCAN).

1. What are acute aortic syndromes?
2. How are aortic dissections classified?

3. Is advanced age a contraindication for aortic reconstruction surgery?

4. Is medical management acceptable versus undergoing this high risk surgery?

5. What is the surgical mortality associated with AAAD repair?

6. What is the commonly used imaging test in the ER when a dissection is suspected?

7. What is the triple rule out protocol used in the ER?

8. Is a D-dimer test of any significance?

9. Are there any other biomarkers to look for in aortic dissections?

The patient was brought back to the OR 8 hours after the diagnosis of AAAD was made. The plan was to resect the entry tear, and re suspend the aortic valve, as well as a hemi or total arch replacement based on the operative findings involving the aortic arch and the great vessels and to do the distal anastomosis under hypothermic circulatory arrest (HCA). The surgeon also wants to cannulate the dissected aorta for arterial cannulation. He wants your help in getting the cannula into the true lumen as he has repeatedly cannulated the false lumen.

1. What monitoring would you use?

2. Are bilateral radial arterial lines required?

3. How would you maintain the hemodynamics? What are your goals and priorities?

4. How can you help the surgeon to place the aortic cannula in the true lumen?

5. What other sites are reasonable for arterial cannulation?

6. Is right axillary or subclavian arterial cannulation preferred over other sites?

7. If femoral cannulation is chosen which side would you prefer?
8. Is it ok to cannulate the carotid for arterial access? How about the left ventricular apex?

9. How would you monitor the integrity of the brain while on HCA?

Cardiopulmonary bypass (CPB) was achieved uneventfully. The aortic valve was re suspended. The intimal tear was resected and a proximal anastomosis was performed with the graft prosthesis while cooling the patient on CPB to 20°C and HCA was achieved. A plan for hemi arch replacement was made after inspecting the arch and the supra aortic vessels. Retrograde cerebral perfusion (RCP) was initiated via the right internal jugular vein. At 10 minutes into HCA the cerebral oximetry drops to 50% of baseline.

1. Is hypothermia of 20°C adequate?

2. What is the usual threshold for HCA time?

3. Does HCA at 20°C for greater than 30 minutes increase mortality?

3. What other options do we have to protect the brain?

The surgeon places a balloon tip catheter into the innominate and common carotid arteries. The perfusionist starts anterograde cerebral perfusion (ACP) at 10 ml/kg/minute at 18°C. The cerebral oximetry saturations return to normal. A hemi arch replacement was performed at the level of the lesser curvature of the arch. The patient was rewarmed and successfully weaned from CPB.

1. Is ACP better than RCP?

2. Does ACP increase the risk of stroke?

3. Do these cerebral perfusion techniques improve outcomes?

4. What about spinal cord protection in type AAAD while on HCA?

5. Is HCA necessary if only the ascending aorta has to be replaced?

6. What is a composite graft for a Bentall procedure?

After weaning from CPB the patient’s ventricular function is excellent and bleeding is controlled. However, TEE still shows a patent false lumen in the descending aorta (DA) with
intermittent blood flow.

1. How is the dissection extending into the DA managed?

2. What is an Elephant Trunk Procedure?

3. Frozen Elephant Trunk repair, is it an option?

4. Are there any hybrid options to cut short the HCA times?

5. Would an Endovascular approach been an option in this AAAD?

Patient returns after 6 months for an endovascular procedure on the DA. He states he has been having severe abdominal pain and diarrhea. He is scheduled for stenting of the SMA.

1. What is the long term survival after surgical repair of AAAD?

2. What is the most important goal in treating these patients?

3. How often should these patients be imaged after surgery?

4. What are the late surgical complications?

Model Discussion Content

AAAD is a life threatening emergency. Mortality is estimated to be 1-2% per hour after the onset of symptoms. Acute aortic syndrome includes aortic dissections, intramural hematomas and penetrating aortic ulcers. An intimal tear is usually present. The predisposing factors to cause the intimal tear are medial degeneration and cystic medial necrosis. Entry of blood into the media can create a false lumen in the aorta, resulting in blood flow through both the true and false lumens. Hypertension is the most common risk factor at approximately 75%. 20% have genetic disorders with altered connective tissues such as Marfans and Turner syndromes. The incidence of aortic dissections are 2.9 per 100,000 per year. An estimated 10,000 Americans die yearly secondary to aortic dissections. The American Heart Association and the American College of Cardiology published guidelines for the diagnosis and management of thoracic aortic diseases. It is important to have a high index of suspicion. The annual incidence of acute coronary syndromes is 440/100,000, with pulmonary embolism 69/100,000. Acute aortic dissections were 3 to 4/100,000. The high mortality in these patients stems from aortic rupture, cardiac tamponade, acute aortic valve insufficiency, severe bleeding, and involvement of the coronary arteries resulting in myocardial
ischemia or infarction. Physicians should have a high index of suspicion for this rare but catastrophic disease.

**History, clinical presentation and severity of the disease:**
Sudden onset of severe sharp chest or back pain was the single most common presenting symptom and is present in approximately 93% of patients. Neurological signs such as syncope(19%) and paraplegia were also noted. Patients with Pulse deficits are predictive of complications. Uncontrolled hypertension is associated with increased mortality. Advanced age increases the risk of dying from aortic rupture. Long term outcomes are poor in patients with persistent patent false lumen. As per the data from International Registry of Aortic Dissections (IRAD) in hospital mortality among medically managed patients was 57%, surgical mortality was 18%, with a significant decrease in surgical mortality over the last 17 years and 90% of patients presented to the hospital were surgically treated. Advanced age, female sex, abrupt onset of pain, pulse deficits, renal failure, hypotension, shock and cardiac tamponade were associated with higher mortality in AAAD patients. The dissections can extend anterograde or retrograde. Progression of dissection is attributed to the pulsatile nature of the blood flow. True lumen compression can result in end organ ischemia and malperfusion syndromes. The larger the entry tear the greater is the risk of compressing the true lumen.

**Classification of Aortic Dissections:** Debakey’s, and Stanford classifications are the most commonly used classification in clinical practice.

**Debakey classification**: The Debakey classification is based on the site of the intimal tear and extension of the dissection. 

- **Debakey 1**: Originating in the ascending aorta with distal extension to the DA
- **Debakey 2**: Originating in the ascending aorta and confined to it
- **Debakey 3**: Originates and involves the the DA only

**Stanford classification**: The Stanford classification is based on involvement of the ascending aorta.

- **Stanford A**: Dissection involving the ascending aorta proximal to the brachiocephalic artery.
- **Stanford B**: Dissection involving the DA. A type B dissection excludes the ascending aorta and arch.

Debakey and Stanford classifications are used in the decision making process between surgery and medical management.

Endovascular interventions (EI) are commonly performed for DA dissections. EI is still in the early stages when it comes to AAAD. Patients who deemed unfit for surgery have been managed with EI with some success. AAAD patients with DA involvement and malperfusion issues of the DA, are managed endovascularly.

A temporally based classification proposed by Booher and colleagues can be used to stratify survival rate using four distinct time domains . Hyper-acute: less than 24 hours

Acute: 2 to 7 days
Sub-acute: 8 to 30 days
Chronic: greater than 30 days. Mortality increases with time.

**Dissect:** It is a mnemonic based classification. Patients are divided into subsets according to anatomical involvement with relevance to endovascular management.\(^{14}\)

D-Duration of the disease
I- Intimal tear location of the dissected aorta
S- Size of the dissected aorta
SE- Segmental extent of aortic involvement
C- Clinical complications of the dissection
T- Thrombus within the aortic false lumen

**Diagnosis, Imaging techniques, and delays in diagnosis:**
Early diagnosis and prompt surgical treatment are associated with better survival in AAAD. Studies have shown 38% of aortic dissections are missed on initial presentation. In 28% of patients the diagnosis was established on postmortem examination.\(^{15}\) Only about 39% of patients were diagnosed within 24 hours after the onset of symptoms.\(^{16}\) The correlates for delayed recognition and treatment for type A dissections were female sex, atypical presentation, absent chest or back pain, absence of pulse deficits, hypotension and presentation to a non-tertiary care hospital.\(^{17}\) There are several imaging techniques available, however computerized axial tomographic arteriography (CTA) with and without contrast is the most commonly used. Contrast enhanced CT angiography is widely available and noninvasive.\(^{18}\) CT scan, magnetic resonance imaging (MRI) and transesophageal echocardiography (TEE) all these three imaging techniques are all reliable for ruling out or confirming thoracic aortic dissection.\(^{19}\) Benefits of transthoracic echo are portability, ease of rapid access in unstable patients. However the drawbacks include difficulty visualizing the ascending aorta. TEE imaging is invasive but with low risk of complications, and provides excellent images of the ascending aorta and aortic valve. Is been shown to correctly detect the site of the entry tears as well as the true and false lumens but imaging the arch can be problematic with interference by the left main bronchus. MRI provides excellent information, but is usually the test of choice for follow up. The classical feature seen in CT scan is dislodgement of intimal calcification. A one stop CT scan for chest pain is designed to differentiate acute coronary syndromes, pulmonary embolism, acute aortic dissections and it is called as `TRIPLE RULE OUT PROTOCOL`.\(^{20}\) In the German registry of acute aortic dissection Type A (GERAADA) the investigators found prior to surgery 80% patients had CT scans, 50% had echocardiography and less than 2% had MRI as the primary imaging technique. CT scan, use for type A dissections increased from 46 to 73% over the last 17 years, TEE as the first diagnostic study decreased from 50% to 23% over the same time period.\(^{6}\) The sensitivities for MRI, TEE and CT scans were approximately 95%. Diagnostic specificity of MRI is 97% and CT scan 87%.\(^{21}\) ECG may be normal or present with nonspecific changes or Ischemia commonly seen in the right coronary arterial territory. Chest X ray may show widening of the mediastinum.
**Biomarkers:** The absence of D-dimers has high negative predictive value. It can correlate with pulmonary embolism and aortic dissection. D-dimers may be useful in risk stratifying patients with suspected aortic dissection. Used to as a rule out marker for aortic dissection within 24 hours of onset of symptoms.22

**Management of Acute Aortic dissections:** Prompt diagnosis with CT scan, improved cerebral and myocardial protection strategies during aortic arch reconstruction and evolving endovascular techniques may result in less invasive surgeries.23 On hospital admission 50% of patients are hemodynamically unstable, 25% have neurological deficits, 20% have pericardial tamponade, 6% have already undergone CPR.24 The symptomatology in these patients can be dynamic. True lumen compression, and movements of the dissection flap cause waxing and waning symptoms by affecting the blood flow within both lumens.

**Goals in initial management:** Control of blood pressure is critical. This can help to prevent or significantly decrease extension of the dissection. Maintain a systolic blood pressure of 100 to 120, and heart rate of 60 to 80. Also avoid rapid increases in blood pressure over short time intervals.

**Anesthetic management of AAAD:** A thorough discussion and constant communication is paramount with all teams involved in the care of the patient regarding the surgical plans, outcome goals, and strategies for brain, heart and other end organ protection. Induction of anesthesia should ensure stable hemodynamics. Blood pressure control with beta blockers such as esmolol and labetalol are safe to use. Beta blockers by reducing the ventricular ejection force may help decrease the stress on the diseased aortic wall. External pacer defibrillator pads should be placed. Blood, plasma products, and platelets should be readily available. Maintaining adequate and stable hemodynamics is crucial. Large bore IV’s, central line, and foley catheter should be placed. The use of swan ganz catheter is always controversial. Temperature monitoring sites should include blood, nasopharyngeal, esophageal, rectum and or bladder. In addition to standard ASA monitors, bilateral radial arterial lines, TEE, near infra red spectroscopy, electroencephalography(EEG), sensory evoked potentials, transcranial doppler should be utilized for monitoring the integrity of the brain and spinal cord.25 Frequent analysis of arterial and mixed venous blood gases, glucose, and acid base status should be performed. Close monitoring, diligence, and team work during pre-extra corporeal circulation, CPB, HCA, rewarming, post CPB stabilization, hemostasis, and return of adequate ventricular function are extremely important. Have rapid infusion systems in the OR ready. Certain patients may require an intra aortic ballon pump, this could be problematic with a persistent and patent false lumen in the DA. Patients may need ventricular support ranging from ionotropes to ventricular assist devices. Cerebral, spinal cord and myocardial protection is paramount. The safety and positive outcomes for these high risk procedures requires preparation, expertise, and critical decision making skills. Knowing the options, having backup plans and exercising intimated judgement,
securing extra help and maintaining flexibility are key to assuring the safety of the patient.

**Surgical management:** AAAD is a life threatening surgical emergency. Surgical mortality has improved as per the IRAD registry from 25% 17 years ago to 18% currently. About 90% patients are treated surgically. Some patients are deemed unfit for surgery based on the severity of illness. 30 day mortality was 34.9% in 80 year old patients compared to 15.8% in their 70 year old counterparts.

**Goals of surgery:** Early surgery, reducing the risk of aortic rupture by closing or excising the entry tear, resecting the diseased ascending aorta and stabilization of the aorta. Improving end organ perfusion, preventing and relieving malperfusion states. Correcting of aortic insufficiency if present either by replacement or repair of the aortic valve.

**Surgical technique:** Surgical technique depends on many factors, such as the the site of intimal tear, extension of the dissection and competency of the aortic valve. The techniques involved include supracommissural replacement of the ascending aorta, valve sparing procedures with ascending aortic reconstruction, or a composite graft with the aortic valve and graft prosthesis. The intimal tear extends or reenters the arch in 70% cases. With proximal arch and hemi arch procedures the distal anastomosis is done in a parallel manner replacing the inner curvature of the arch. The tear is commonly at the inner curvature. With total arch replacements, reimplantation of the supraaortic vessels is done by implanting them on the graft. If the arch is severely destroyed the arch vessels are debranched and implanted separately. The distal anastomosis is done under HCA. Many institutions perform the proximal anastomosis while rewarming. For CPB the venous cannulation is usually done via the right atrium. The arterial cannulation site commonly right axillary/subclavian, however alternative cannulation sites are right femoral, dissected aorta, and carotids have also been used.

**Cerebral protection**

**Hypothermia:** The distal anastomosis is done under HCA. Hypothermia decreases metabolic requirements and offers cerebral protection by decreasing energy requirements for both electrophysiological and biological activities of the brain. The Griep School believes in profound hypothermia of 10 to 15°C, others prefer temperatures of 20°C is deemed safe. HCA of less than 30 minutes at 20°C is safe. HCA alone was used in 22.8% of patients in the GERAADA registry. Among these patients post-operative mortality was 15.9% when HCA times were 15 to 30 minutes. However, with HCA times greater than 30 minutes there was an increased mortality of 35.7%. During HCA the myocardium may be perfused intermittently via the coronary sinus with retrograde blood cardioplegia, this may interfere with visualization.

**Cerebral perfusion for cerebral protection when on HCA:** The brain is perfused with cold blood while on HCA. This can be administered as retrograde cerebral perfusion (RCP) via the Internal Jugular veins or can be given as anterograde cerebral perfusion (ACP) via the right axillary,
innominate, and left common carotid arteries. ACP can be given to both cerebral hemispheres via the innominate and the left common carotid arteries. With ACP there is an increased risk of stroke from air emboli and vascular debris. ACP extends the margin of safety. HCA times of 30 to 60 minutes are tolerated. RCP is given in only 2.2% patients, while 69.4% receive ACP. RCP may increase the safe interval for HCA, but not decrease the metabolic demands of the brain. RCP may help with clearing air in the cerebral vasculature. The optimal cerebral perfusion temperatures is not clear. Experimental studies support a temperature of 20°C. Clinical studies have not shown any disadvantages with moderate hypothermia of 25°C with HCA. The range of cerebral perfusion temperatures vary from below 15°C, to over 30°C. 15 to 20°C is the most commonly used range without significantly influencing the outcomes. EEG, sensory evoked potentials, jugular bulb venous saturation, transcranial doppler, near infrared spectroscopy are used to monitor cerebral protection.

Survival after surgery: 90% at 1 year, 72-77% at 5 years, 53-56% at 10 years. Late complications include dilatation of the aorta, especially with a patent false lumen thus increasing the risk of rupture. Control of hypertension is the most important intervention to avoid dilatation and rupture of the aorta. Frequent follow up with MRI is preferable, however the majority of patients undergo CT scan evaluation. Follow up imaging assessments should be performed at 3, 6, and 12 months and annually thereafter. The distal aorta is not repaired during these surgeries for type A dissection. The thoracic and abdominal portions of the aorta are involved in 40 and 30% of patients respectively with Type A dissections. The descending aorta is addressed at a later time with Elephant Trunk procedure. In the classic Elephant Trunk procedure after constructing the aortic arch, an extension of the arch graft, which can be used for repair of the DA at a later stage, is left in the DA. A Frozen Elephant Trunk procedure is a hybrid surgical technique where a reinforced stent graft is implanted into the descending aorta in an antegrade fashion. The ascending aorta and arch are repaired surgically.

Endovascular suitability of Type A dissection: The principal determinant for endo vascular repair is the location of the tear. It should be at least 20 mm away from the STJ. About 30 to 50% patients who undergo open repair may be candidates for endovascular repair. A graft close to the aortic valve may cause problems with aortic insufficiency or even coronary occlusion. The hemodynamics in the ascending aorta are not so conducive to deploy the graft. At this time open surgery remains the gold standard for ascending aortic dissections.

References:
17. Harris KM, Strauss CE, Eagle KA, Hirsch AT, Isselbacher EM, Tsai TT, Shiran H, Fattori R,


