Acute Aortic Syndrome

INTRODUCTION

Acute aortic syndrome (AAS) represents a spectrum of life-threatening conditions with similar clinical presentation and the need for urgent management. It includes classic acute aortic dissection (CAAD), intramural hematoma (IMH), and penetrating aortic ulcer (PAU). Although not included in the original definition of AAS, traumatic aortic rupture (TAR) and aortic aneurysm rupture have also been considered to be part of the AAS spectrum.

AAS is characterized by disruption of the media layer of the aorta and typically presents with acute chest pain. The term “acute aortic syndrome” was first coined in 2001 by the Spanish cardiologists Vilacosta and San Román, who described AAS as a spectrum of interlinked lesions with the intent to increase awareness and to speed up diagnosis and appropriate treatment (Figure 32.1).

Although the incidence of AAS is lower than that of acute coronary syndrome (ACS), AAS carries a higher mortality, and is therefore a critical component of the differential diagnosis of chest pain in the CCU. Overall incidence of AAS is 2 to 4 cases per 100,000 individuals. Because AAS is rare, the International Registry of Acute Aortic Dissection (IRAD) was created in 1996 as a way to combine data acquired from multiple top institutions in Europe, North America, and Asia. The 2010 intersocietal guidelines for the diagnosis and management of patients with thoracic aortic disease proposed a standard approach to the diagnosis and treatment of AAS.

Although clinical history and physical examination are important, imaging is essential in the diagnosis of AAS. Transesophageal echocardiography (TEE), computed tomography (CT), and magnetic resonance imaging (MRI) are the preferred imaging modalities and angiography is rarely needed.

CLASSIFICATION OF ACUTE AORTIC SYNDROMES

Historically, CAAD was the first recognized form of AAS. The classification schemes used for the classic aortic dissection were subsequently extended to include IMH and PAU.

AASs are classified on the basis of the location and extent of involvement of the aorta. Two systems have been proposed, the DeBakey and the Stanford systems (Figure 32.2). The DeBakey system, which was proposed in 1965 by the Lebanese-American surgeon Michael Ellis DeBakey, divided aortic dissection into three types based on the anatomic location. Type I originates in the ascending aorta and propagates beyond the aortic arch, type II is limited to the ascending aorta only, and type III is limited to the descending thoracic aorta. The Stanford classification appears to have wider acceptance and is now used for all three AAS types: CAAD, IMH, and PAU.

INTRAMURAL HEMATOMA

IMH is defined by crescentic or circumferential thickening of the media layer of the aortic wall. IMH is likely due to a ruptured vasa vasorum resulting in intramural bleeding but without a detectable intimal tear. It was first described in 1920 by the German pathologist Ernst Kruckenberg, who is also well known for his description of the so-called Kruckenberg tumors (transperitoneal ovarian metastases from stomach and colon cancers). On TEE, CT, or MRI, IMH is typically visualized as a crescentic or concentric thickening of the aortic wall > 5 mm (Figure 32.3).
The natural history of IMH often includes progression to CAAD, which accounts for its high morbidity and mortality.

**Etiology and Pathophysiology**

IMH may account for up to 6% to 30% of all AAS, with a higher reported prevalence among the Korean and Japanese populations as compared with Western subjects. It is unclear whether this is a true discrepancy in prevalence versus a reflection of differing classification, evaluation, or treatment practices. Often, IMH is diagnosed as such even though very small intimal tears indicative of limited aortic dissection may be present but missed by modern imaging modalities. This may overestimate the true prevalence of IMH as opposed to CAAD.

The characteristic feature of IMH is its location in the portion of the media closer to the adventitia, as opposed to CAAD which is typically located in the media closer to the intima. Although the most cited hypothesis of the pathophysiologic mechanism of IMH is rupture of the vasa vasorum, there is very little corroborating clinical or experimental evidence. Owing to the low incidence of IMH and the close association with CAAD, a definitive etiology still remains unclear.

**Clinical Manifestations**

According to the IRAD experience, IMH typically presents with the symptoms of severe chest and back pain, similar to CAAD. However, IMH is less likely to present with manifestations of severe aortic regurgitation and pulse deficits. IMH is rarely stable. It may either progress to CAAD or regress spontaneously, and therefore serial imaging is crucial. Stanford type B lesions in the descending aorta are more common than type A lesions in the ascending aorta (60% vs 35% of all IMH, respectively). Cardiogenic shock may be present in 14% of patients, more typically with type A IMH. Pericardial effusion and tamponade may also be present, which are also more common in type A IMH. When compared with CAAD, type A IMH has a significantly higher risk of rupture (26% vs 8%, respectively). A widened mediastinum may be present on chest X-ray; however, this is neither sensitive nor specific to IMH.

**Diagnosis**

As with all types of AAS, rapid diagnosis is paramount in IMH. TEE, CT, and MRI are the preferred diagnostic tools. CT is often...
Intramural Hematoma in Pregnancy

Although there are no specific guidelines in pregnancy for patients with IMH, pregnancy is considered a risk factor for the development of aortic pathology, especially in Marfan syndrome. As with other forms of AAS, expedited delivery via cesarean section is considered reasonable for pregnant patients with acute IMH, if possible.

CLASSIC ACUTE AORTIC DISSECTION

CAAD is the most common form of AAS. It occurs in approximately 75% of all AAS. The overall incidence of CAAD is low, estimated at 0.5 to 4.0 cases per 100,000 per year, and is thought to affect men more than women in a 2:1 ratio.

Risk factors for CAAD include connective tissue disorders such as Marfan (fibrillin gene), Loeys-Dietz (transforming growth factor β receptor 1 and 2 genes), Ehlers-Danlos type 4 (collagen gene), and Turner syndrome (X monosomy), as well as the aortopathy associated with bicuspid aortic valve (NOTCH1 gene). In addition, hypertension is a significant risk factor and is more prevalent among older patients. Last, aortic instrumentation or surgery, as well as cardiac catheterization, are rare but reported causes of aortic dissection.

CAAD was first described in 1555 by Andreas Vesalius (1514–1564) who reported traumatic abdominal aortic aneurysm in a man who fell off a horse. Intimal tear, the hallmark of CAAD, was first described by Daniel Sennert (1572–1637), a German anatomist and published in 1650 posthumously. A very famous description of CAAD was by the British royal physician Frank Nichols (1699–1778) who provided the first unmistakable account of CAAD (deemed a “Transverse fissure of the aortic trunk”) in his autopsy of King George II, who died in 1760 while straining in the lavatory. Successful surgical repair of descending aortic dissection was not reported until 1955, by Michael DeBakey (1908–2008) and his colleagues, and ascending dissection until 1962 by Frank Spencer and Hu Blake.

Etiology and Pathophysiology

CAAD is characterized by an intimal tear, which leads to abnormal blood flow from the aortic lumen into the media (Figure 32.5). Consequently, there is a longitudinal separation of the media layers by the blood flow, which tears an intimal medial flap from the remainder of the aortic wall (Figure 32.6A–C). This flap separates the abnormal false lumen from the true aortic lumen. Intimal tears typically occur at the locations within the aorta...
Section IV • Aortic, Pericardial, and Valvular Disease in the CCU

Clinical Manifestations

The typical symptom of acute aortic dissection is “aortic pain” similar to other forms of AAS. Acute, severe, tearing chest pain is the hallmark symptom of CAAD. Pain limited to the chest is typical of type A CAAD, and pain in the back is more often the symptom of type B CAAD. One study found older patients are less likely to abrupt onset of pain as compared with younger patients.15

Pulse deficit, present in up to 33% of patients according to the IRAD study, reflects impaired or absent blood flow to peripheral vessels. This is manifested by weak carotid, brachial, or femoral pulses on physical examination.

Other physical examination findings of CAAD include diastolic murmur or aortic regurgitation, hypotension related to either tamponade or aortic rupture, focal neurologic deficits reflecting propagation of the dissection toward involvement of carotid or cerebral arteries, and syncope.

Electrocardiogram (ECG) may be useful in distinguishing the chest pain of AAS from ACSs; unlike ACS, uncomplicated CAAD does not present with ischemic ECG changes. However, if the aortic dissection leads to coronary ischemia through involvement of coronary ostia (type A), the ECG will be less helpful with differentiation of symptoms.

Chest X-ray (CXR) imaging occasionally shows widening of the mediastinum, a nonspecific finding seen with other syndromes such as mediastinal hematoma. Other CXR findings are double aortic knob (40% of patients), tracheal displacement to the right, and enlargement of the cardiac silhouette.

Serum biomarkers such as D-dimer are often elevated in CAAD, but this is a nonspecific finding. In contrast, a normal D-dimer level may help exclude the diagnosis of CAAD. Investigational biomarkers such as elastin degradation products, calponin, fibrinogen, fibrillin, and smooth muscle myosin heavy chain are currently being evaluated.

Diagnosis

As with other forms of AAS, the 2010 intersocietal guidelines for the diagnosis and management of patients with thoracic aortic disease provide a useful decision tool to help guide diagnostic and management strategies for CAAD with a special emphasis on a combination of clinical risk assessment and rapid imaging.

CT with intravenous iodinated contrast is often the diagnostic modality of choice for CAAD because of its superb spatial resolution, rapid acquisition times, widespread availability, and its ability to diagnose other causes of acute chest pain such as trauma and pulmonary embolism. The reported sensitivity of CT for CAAD is 87% to 94% and specificity is 92% to 100%. CT features of CAAD are intimal tear, dissection flap with a true and false lumen, dilatation of the aorta, and pericardial effusion.

TEE is especially useful in the diagnosis of CAAD when a CT with contrast cannot be performed, such as in hemodynamically unstable patients or in patients in whom the risk of iodinated intravenous contrast is high such as renal insufficiency or severe allergy. The reported sensitivity of TEE is 98% and specificity is 63% to 95%. Findings on TEE are a dissection flap separating the true and false lumen, site of intimal tear represented by flow from the true lumen into the false lumen on color Doppler (Figure 32.7). Spectral Doppler may help corroborate the diagnosis by demonstrating “to and fro” flow into and out of the false lumen.

Complications such as aortic regurgitation and pericardial tamponade can occur; and, over time, chronic changes such as false lumen thrombosis and aneurysm are common.
The true lumen is identified by its expansion with systole and contraction in diastole. The true lumen is often smaller than the false lumen. In early stages, the false lumen may be echo free or may contain spontaneous echo contrast (also known as “smoke”) due to stasis of blood flow. In later, more chronic stages, the false lumen may be partly or completely obliterated by thrombus formation.

Complications of CAAD may be seen on echocardiography such as aortic regurgitation, pericardial effusion/tamponade, and wall motion abnormalities indicative of ischemia if there is coronary ostial involvement.

It is important not to confuse the intimomedial flap of CAAD with either artifacts or surrounding vascular structures. Linear reverberation artifacts in the ascending aorta should not be mistaken for type A aortic dissection. Typically, reverberation artifacts are located twice as deep as the anterior aortic wall. In addition, a dilated azygos vein adjacent to the descending thoracic aorta may give an illusion of a type B dissection. Color or spectral Doppler imaging in both instances may help distinguish true aortic dissection from its masqueraders (Figure 32.8).

Although on transthoracic echocardiography (TTE) aortic dissection can occasionally be seen, TTE should only be used as a screening tool owing to lack of sufficient sensitivity and specificity.

MRI and aortography also may reveal aortic dissection; however, they are reserved for specific situations. MRI may be used when the patient cannot receive iodinated contrast for CT nor undergo TEE. Aortography is of limited use and is typically performed during invasive endovascular therapeutic procedures.

Management and Prognosis

Type A CAAD is a true medical emergency, requiring immediate surgical repair because the mortality increases by the hour. Approximately 90% of medically managed patients with type A CAAD die within 3 months of presentation. On the other hand, the prognosis is more favorable for patients with type B CAAD in whom medical management is often preferred over surgical repair because surgically managed patients have been shown to have higher mortality compared with those on medical therapy alone. Medical therapy generally consists of tight blood pressure control and β-blockade. Surgical management of type A CAAD typically consists of excision of the intimal tear if possible and obliteration of entry into the false lumen, as well as implantation of a graft to replace the ascending aorta. Surgical therapy for type B is more complicated because of the presence of many spinal artery branches, and therefore has a risk of paraplegia. Nevertheless, surgical therapy of type B dissection is often

![FIGURE 32.7](#) Classic acute aortic dissection: dissection flap on TEE. Two-dimensional (2D) TEE of the ascending thoracic aorta in short axis (A) and long axis (B) demonstrating CAAD. In this case, the dissection flap is circumferential with a 360° separation of the true and false lumens. CAAD, classic acute aortic dissection; FL, false lumen; TEE, transesophageal echocardiography; TL, true lumen.

![FIGURE 32.8](#) Reverberation artifact masquerading as type A dissection on TEE. Two-dimensional (2D) TEE of the ascending thoracic aorta in a long-axis view (A) and a short-axis view (B). Red arrows point to linear reverberation artifact. Note that the reverberation artifact is located twice as deep (2x) as the anterior aortic wall, characteristic of reverberation artifacts. TEE, transesophageal echocardiography.
necessary when there is aortic branch ischemia and end-organ damage. Endovascular graft therapy to treat type B CAAD has shown promise (Figure 32.9). It is important to identify risk factors for higher mortality in type A CAAD such as advanced age, prior cardiac surgery, hypotension or shock, pulse deficit, cardiac tamponade, and ischemic ECG changes.

**SECTION IV • Aortic, Pericardial, and Valvular Disease in the CCU**

**Penetrating Aortic Ulcer**

PAU represents the process by which an atherosclerotic plaque erodes and penetrates through the elastic lamina into the media layer of the aorta, causing ulceration (Figure 32.10). PAU may further erode through the adventitia leading to either focal (pseudoaneurysm) or complete aortic rupture (Figure 32.11). Thrombus occasionally forms within PAU. In addition, PAU may lead to either IMH or aortic dissection, which is why PAU is characterized as an AAS.

**Etiology and Pathophysiology**

PAU accounts for 2% to 11% of all AASs. It was first described in 1986 by Anthony Stanson and colleagues. Patients with PAU typically are older (>70 years old) and have risk factors for atherosclerosis including hypertension, smoking, and hyperlipidemia. The natural history of PAU is not well described. PAU may cause remodeling of the aortic wall and aneurysm formation, contained rupture through the aortic wall and attendant pseudoaneurysm formation, complete aortic rupture with mediastinal or pleural hemorrhage, or progression to IMH and CAAD.

**Classic Acute Aortic Dissection in Pregnancy**

The 2010 intersocietal guidelines for the diagnosis and management of patients with thoracic aortic disease recommends expedited fetal delivery via cesarean section for patients with CAAD during pregnancy given the high mortality of the disease (class IIa recommendation). The diagnostic imaging modality of choice is MRI without gadolinium to avoid exposing the mother and fetus to ionizing radiation. TEE is an option and is considered safe in pregnancy; however, caution must be used when providing procedural sedation because the medications typically administered (midazolam and fentanyl) may be teratogenic, especially in the first trimester. In these cases, topical anesthesia with viscous lidocaine is crucial. There have been reports recommending monitoring fetal HR and uterine tone during TEE.

**FIGURE 32.9** Endovascular graft repair. 3D CT. Three-dimensional (3D) reconstruction of contrast-enhanced chest CT in a sagittal view (A) and coronal view with surrounding structures removed (B) demonstrating an endovascular stent graft located between the junction of the aortic arch and descending thoracic aorta, extending to the distal descending thoracic aorta. CT, computed tomography.

**FIGURE 32.10** Penetrating aortic ulcer: TEE. Two-dimensional (2D) TEE of the descending thoracic aorta in the midesophageal short-axis view (A) and long-axis view (B). Yellow arrows point to demonstrating severe atherosclerotic plaque and PAU; yellow dashed arrows point to an area with developing IMH. IMH, intramural hematoma; PAU, penetrating aortic ulcer; TEE, transesophageal echocardiography.
Chapter 32 • Acute Aortic Syndrome

TRAUTOMATIC AORTIC RUPTURE

Although TAR is not considered to be a part of the original AAS triad, it is a life-threatening aortic emergency with only a 15% to 20% survival. TAR is typically caused by deceleration injuries sustained in motor vehicle accidents (MVAs) and falls greater than 3 m. It is the second leading cause of death after blunt trauma, occurring in approximately 1.5% to 1.9% cases.

Etiology and Pathophysiology

The most common site of injury in TAR is at the aortic isthmus, immediately distal to the left subclavian artery at the site of the ductus arteriosus. This location is considered to be the most vulnerable to torsional and shear forces because it is thought to be a transition zone between the semi-mobile aortic arch and the fixed descending thoracic aorta. Other possible sites of injury are the transverse arch, ascending aorta, and descending aorta proximal to the diaphragm. Typically, the intima and medial layers rupture first, followed by rupture of the adventitia after an unpredictable interval of time.

Clinical Manifestations

Symptoms of PAU are similar to that of other AASs. The pain associated with PAU is variable, and dependent on the location of the ulceration. Type A PAU typically presents with chest pain and type B PAU is more likely to present with back pain. Unlike IMH or CAAD, there have been reports of PAU as an incidental finding in asymptomatic patients.

Diagnosis

The diagnosis of PAU is primarily made by CT, TEE, and MRI. Aortography is not typically used for PAU because of lack of direct visualization of the aortic wall. All three techniques are able to image atherosclerotic changes, ulceration, and complications such as pseudoaneurysm, rupture, and mediastinal and pleural hemorrhage. Identification of an ulcer crater distinguishes PAU from IMH with intraparietal. PAU lesions are typically focal as opposed to those of CAAD and IMH, which are more extensive.

Management and Prognosis

The natural history of PAU is poorly understood. On one hand, PAU is considered to be a surgical emergency with risk similar to or worse than other forms of AAS. On the other, reports have described the progression of PAU as slow, with low prevalence of life-threatening complications. There is therefore equipoise regarding the optimal medical versus surgical treatment strategies. Nevertheless, surgical management of PAU with aortic grafting is considered appropriate in the presence of aortic rupture, persistent or recurrent pain, hemodynamic instability, or rapidly expanding aortic diameter.

Penetrating Aortic Ulcer in Pregnancy

Because PAU is a disease that primarily affects older people (>70 years of age), it is highly unlikely that it will occur during pregnancy. There is therefore no available guideline to direct optimal management.

FIGURE 32.11 Penetrating aortic ulcer with rupture/pseudoaneurysm, CT. CT with intravenous iodinated contrast of the chest. Arrows point to PAU with aortic rupture and pseudoaneurysm of anterior portion of the proximal descending thoracic aorta. CT, computed tomography; PsA, pseudoaneurysm.
more specific in differentiating ductus arteriosus diverticula from TAR. Another very useful advantage of TEE is its portability, with the ability to be performed at the bedside of hemodynamically unstable patients, a common scenario in TAR. The main limitation of TEE is an apparent “blind spot” at the distal ascending aorta and proximal aortic arch caused by bronchial shadowing. Aortography, the former gold standard, may be performed; however, it is invasive and can result in worsening of the aortic rupture in as many as 10% of patients and is therefore not the preferred diagnostic modality.

Management and Prognosis
Emergent surgical therapy is the standard of care for TAR. As with AAS, medical therapy consists of very close blood pressure and HR control. Hemodynamically unstable patients should be operated on immediately. Surgical options comprise open repair with prosthetic grafts, and endovascularly delivered fabric-covered stents. Endovascular repair has been shown to have decreased overall mortality compared with surgical repair, and is recommended when possible. The overall survival of TAR is approximately 10% to 18%. Survival to emergency room care greatly improves the odds of long-term survival, and survival to surgical therapy improves the odds even more, to approximately 70% to 90%.29

Traumatic Aortic Rupture in Pregnancy
Although there are no specific guidelines for the management of TAR in pregnancy, expedited delivery via caesarian section with emergent aortic surgery is a reasonable therapeutic approach given the high mortality both to the mother and fetus.

REFERENCES
Patient and Family Information for:  
ACUTE AORTIC SYNDROME

GENERAL CONCEPTS OF ACUTE AORTIC SYNDROME

WHAT IS THE ILLNESS?

AAS refers to four related diseases of the large vessel that leaves the heart, called the aorta. These are CAAD, IMH, PAU, and TAR. These conditions involve damage to the wall of the aorta and require prompt care because they are associated with a high chance of dying unless treated rapidly.

HOW WILL THE PATIENT BE TREATED?

Once the diagnosis of AAS is established by CT, TEE, or MRI, the disease is typically treated with medications that lower blood pressure and HR. The doctor will determine the type of AAS (type A or type B) based on the location of involvement in the aorta. A cardiothoracic surgeon may be consulted, who will assess the need for surgery. Surgery is often needed as soon as possible.

WHAT IF THE PATIENT IS PREGNANT OR THINKING OF BECOMING PREGNANT?

Given the high mortality of AAS and the frequent need for emergency cardiac surgery, the doctor may recommend expedited delivery. If at risk of AAS because of genetic conditions that may affect the aorta, the patient should consult the doctor to assess the risk if she is thinking about becoming pregnant.

INTRAMURAL HEMATOMA

WHAT IS THE ILLNESS?

IMH is described as bleeding into the wall of the aorta due to breakage of the internal blood vessels of the aorta. Symptoms of IMH are sudden severe chest or back pain. IMH is best diagnosed by imaging the aorta using CT, TEE, or MRI. On experiencing symptoms suggestive of IMH, the patient or a family member should seek medical care immediately because the risk of dying from this condition increases by the hour.

HOW WILL THE PATIENT BE TREATED?

Once the diagnosis of IMH is established, medications will be given to reduce the blood pressure and HR. A cardiothoracic surgeon may be consulted, who will assess the need for surgery. Surgery will often involve either replacement of the diseased portions of the aorta or placement of special type of stent within the aorta that will help contain the bleeding and prevent the aorta from bursting.

WHAT IF THE PATIENT IS PREGNANT OR THINKING OF BECOMING PREGNANT?

IMH carries a high risk of mortality, and often requires emergency surgery. The doctor will tailor the medications for IMH to include only those with minimal risk to the baby. If the pregnant patient or family member requires emergency surgery, expedited delivery is prudent. Rapid consultation with an obstetrician is crucial. If the person has a condition that puts her at risk for IMH such as Marfan syndrome or other genetic disorders of the aorta, consult the doctor to assess the risk if thinking about becoming pregnant.

CLASSIC ACUTE AORTIC DISSECTION

WHAT IS THE ILLNESS?

CAAD is the most common type of AAS. It is caused by a tear of the inner layer of the aorta, called the intima. This tear can then propagate, leading to separation of the layers of the aorta. There are hereditary disorders such as Marfan syndrome and bicuspid aortic valve that may put the person or a family member at risk of CAAD because of weakening of the aortic wall.

Symptoms typically experienced are severe “tearing” chest or back pain that occurs at rest. If the person or a family member experiences such symptoms, seek medical care immediately.

CAAD will be diagnosed using CT, TEE, or MRI, which are widely available and can be performed and interpreted rapidly.

HOW WILL THE PATIENT BE TREATED?

As with other types of AAS, the doctor will prescribe medications that lower blood pressure and HR. A cardiothoracic surgeon may be consulted, who will assess the need for surgery. Surgery is often needed as soon as possible.

WHAT IF THE PATIENT IS PREGNANT OR THINKING OF BECOMING PREGNANT?

As with other types of AAS, CAAD is often a surgical emergency. As such, consultation with an obstetrician and expedited delivery may be recommended. If the patient or a family member has a disorder that involves the aorta, consult the obstetrician before deciding to conceive.

PENATRATING AORTIC ULCER

WHAT IS THE ILLNESS?

PAU is caused when a very severe plaque breaks through the aorta, causing a hole, or ulceration. Risk factors for PAU include advanced age, high blood pressure, high cholesterol, and smoking. Symptoms include chest and back pain, although some patients may have no symptoms.
Along with other forms of AAS, PAU is diagnosed by CT, TEE, or MRI.

**HOW WILL THE PATIENT BE TREATED?**
The doctor may recommend close monitoring with imaging studies, medications, or surgery.

**WHAT IF THE PATIENT IS PREGNANT OR THINKING OF BECOMING PREGNANT?**
PAU is typically a disease that affects older people (more than 70 years of age). It is highly unlikely that it will occur during pregnancy.

**TRAUMATIC AORTIC RUPTURE**

**WHAT IS THE ILLNESS?**
TAR describes tearing of the aorta after a chest injury. It most commonly occurs after MVAs and bad falls.

TAR is a very dangerous condition and requires prompt medical attention. A mark across the skin of the chest due to a seat belt often is present when TAR is caused by an MVA. TAR is diagnosed using CT and TEE.

**HOW WILL THE PATIENT BE TREATED?**
Emergency surgery is the standard of care for TAR. The doctor may prescribe medications to lower HR and blood pressure if necessary; however, a cardiothoracic surgeon may be consulted as soon as possible. Treatment typically requires open heart surgery.

**WHAT IF THE PATIENT IS PREGNANT OR THINKING OF BECOMING PREGNANT?**
Because TAR is a surgical emergency, consultation with an obstetrician for early delivery is crucial. The surgeon may recommend delivery by cesarean section at the time of surgery to repair the broken aorta.
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