A 35-year-old female with an unremarkable medical history who is four weeks’ post-partum presents with vague “chest pressure” for three days. Her husband initially noted her being slightly out of breath with exertion. She thought that she had strained a muscle carrying her baby around the house, but her symptoms did not improve over several days. She presented for evaluation. She denies dizziness, nausea, dysuria, headache, abdomen pain, fevers, chills, back pain, leg pain, or sweats. She is on no prescription medications but does take a daily prenatal multivitamin. Past medical history is significant for three live childbirths, no cesarean sections, and no stillbirths. She has no medication allergies. She does not smoke nor drink alcohol. Family history is noncontributory.

Physical examination is unremarkable with normal vital signs. Lungs are clear to auscultation. Cardiac exam revealed a regular, rate, and rhythm with no murmurs, rubs, or gallops. Abdomen is nontender, nondistended, and with good bowel sounds. Laboratory evaluation revealed a normal CBC, chemistries, ESR, TSH, and urinalysis.

EKG showed sinus rhythm at 64 with ST elevations across the precordial leads. Chest CT scan revealed a Stanford type A aortic dissection, and she was referred to a cardiothoracic surgeon and was taken to the OR.

Etiology and Pathophysiology

The pathophysiology of pregnancy-related aortic dissection appears to be multifactorial. Factors include hormonal alterations affecting the arterial lining, hemodynamic changes related to increased blood volume and cardiac output, and increased vascular resistance. The physical stress of labor and delivery may also be a contributing factor. Pregnancy-related changes occur in the arterial walls, which appear to be related to estrogen and progesterone surges, and these effects persist for weeks after delivery. Estrogen and progesterone receptors are known to increase during pregnancy which may also be a factor. Relaxin is also believed to play a part in the pathogenesis of pregnancy-related dissections. These affect ultimately lead to a reduction in the amount of acid mucopolysaccarides in smooth muscle fibers in the arterial wall, along with abnormalities in the transforming growth factor pathway.

Clinical Features

Aortic dissection can be a challenging diagnosis to make as symptoms can be nonspecific and overlap with other conditions. Most patients present with some combination of chest discomfort, shortness of breath, dizziness, back pain, nausea, abdominal pain, and diaphoresis. Symptoms can also radiate to the lower extremities making the diagnosis even more challenging. In some situations, the primary symptoms can resolve, and the patient can present with issues related to syncope or aortic valve regurgitation. Chest discomfort is the most common presenting symptom and is often described as a stabbing, tearing sensation. Pain in the chest is more often associated with ascending dissections, while back pain is more often associated with descending dissections. A pericardial effusion is a particularly concerning finding, which can potentially lead to tamponade and sudden death. Other complications can include acute ischemic events such as mesenteric ischemia, spinal cord ischemia, or renal artery ischemia. Ischemic events are more common with type A dissections. Coronary ischemia and myocardial infarction are other possible complications that carry a high mortality rate.

Diagnosis and Testing

The prompt and accurate diagnosis of aortic dissection requires careful history taking with a focus on risk factors and symptom characterization. Confirmatory diagnosis is usually made with CT imaging, MR imaging, or trans-esophageal echo. CT angiography is the most commonly used diagnostic study and has high sensitivity and specificity. Trans-esophageal echocardiography has a much lower sensitivity and specificity.
Aortic dissection can be classified based on several different classification systems. The Stanford classification divides dissections into types A and B where type A involves the ascending aorta and/or the aortic arch, while type B involves the descending aorta beyond the brachiocephalic vessels. This type of classification is helpful as ascending aortic dissections can involve the aortic valve so usually require surgery while descending aortic dissections can sometimes be managed medically. The DeBakey classification system classifies aortic dissections based on where the dissection originated and the extent of the tear.

**Treatment**

The management of aortic dissections depends on the location and severity of the dissection. In general, Stanford type A dissections require surgical management, while Stanford type B dissections are managed medically with optimal blood pressure control. Proper blood pressure control is paramount in the medical management and beta blockers such as Esmolol, Propranolol, or Labetalol are typically first-line therapy. Other classes of anti-hypertensive can also be used including calcium channel blockers. Nitroprusside is usually avoided in pregnancy because of risks to the fetus. Surgical risk for these patients can be increased by a variety of factors including age, coronary artery disease, renal function, presence of tamponade, congestive heart failure, and pulmonary status.

**Prognosis**

The prognosis of postpartum aortic dissection depends on many factors including the severity of the dissection, the location of the dissection, and how quickly the diagnosis is made. The overall mortality rate varies from 20-50% with risks occurring during the peri-operative and post-operative periods.

**Clinical Course and Follow-Up**

The patient was evaluated by a cardiothoracic surgeon and cardiologist and a decision made to proceed with surgery. The patient did well with surgery and was eventually discharged home five days later on a beta blocker.

**REFERENCES**


Submitted August 10, 2016