outline

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Aortic Dissection

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Rapid Access

Approach to the Critical Patient

- **When aortic dissection (AD) is suspected, immediate computed tomography (CT) angiography of the aorta is indicated unless there is a compelling contraindication.**
- Consider early emergency surgical consultation or transfer to a facility with an appropriate vascular specialist.
- **CIRCULATION**
  - **Hemodynamic goals:**
    - Heart rate 60 beats/min.
    - Systolic blood pressure (SBP) <120 mm Hg.

- Consider an arterial line for accurate titration of antihypertensive agents.
- In the event of blood pressure discrepancy among extremities, dose the antihypertensive medication based on the extremity with the higher SBP.

- Upon confirmation of dissection, initiate pharmacologic measures to reduce the pulse pressure:
  - 2-drug strategy: First, β-blockade; second, antihypertensive agent
    - β-blockade
      - **Esmolol IV** is preferred if rapidly available for its titratability given as 500 µg/kg bolus over 1 min followed by 50 µg/kg/min continuous infusion; may titrate up by 25-50 µg/kg/min every 5-15 min to a max of 300 µg/kg/min.
    - Antihypertensive agent
      - **Nicardipine IV** 5 mg/h; may titrate up by 2.5 mg/h every 5-15 min to a max of 15 mg/h, **or**
      - **Clevidipine IV** initial dose 1-2 mg/h IV infusion, titrate up by doubling the dose at 90-s intervals initially; once approaching target blood pressure, increase by less than double dose and lengthen dose interval to 5-10 min, to a max of 32 mg/h, **or**
      - **Sodium nitroprusside** IV 0.3 µg/kg/min, titrate up by 0.5 µg/kg/min every 5 min to a maximum of 10 µg/kg/min for a max duration of 10 min, **or**
      - **Fenoldopam** IV 0.01-0.03 µg/kg/min, titrate up by 0.05-0.1 µg/kg/min every 15 min to a max of 1.6 µg/kg/min.
  - 1-drug strategy
    - **Labetalol IV** 10-20 mg bolus over 2 min, then 20-80 mg bolus every 10-15 min to a max of 300 mg, **or** initiate labetalol continuous infusion at 0.5-2 mg/min, titrate up by 0.5 mg/min every 10 min to a max of 10 mg/min.
      - Note: Labetalol IV has 7 times more beta blocker activity than alpha blocker activity; therefore, labetalol is often inadequate for blood pressure control.

- **If sudden cardiac arrest occurs, consider point-of-care ultrasound to evaluate for pericardial effusion with tamponade and/or hemothorax.**
  - The temporary return of spontaneous circulation may occur following successful pericardiocentesis.

EM:RAP Links

- **Aortic Dissection: Who do I work up?** emrap-video

Key Concepts

- The **aortic wall** figure is composed of 3 contiguous tissue layers in sequence from the vessel lumen moving outward: intima, media, adventitia.
- Aortic dissection (AD) represents a failure of the aortic intima, leading to propagation of dissection at the aortic media due to the patient's pulse pressure. The passage of blood into this space can extend the tear and create a false lumen, ultimately compromising perfusion at branch vessels or causing rupture.
- AD can take any of the following 3 basic morphologies:
  - **Typical AD** - Splitting of 2 layers as described above.
  - **Penetrating aortic ulcer** - Atherosclerotic ulceration of the aortic wall through the elastic lamina (which separates the intima and media) and into the media, permits the entry of blood between the layers, potentially leading to hematoma or eventual aortic wall rupture.
  - **Intramural hematoma** - Crescentic layer of blood forms spontaneously in the aortic wall, without frank separation of layers or an intimal flap. Prognosis and location mimic dissection and, while controversial, most recommend similar management to dissection based on anatomic location.

PITFALLS ◆

- AD is rare and difficult to diagnose due to the non-specific presentation of symptoms.
  - In as many as one-third of patients, AD is not diagnosed upon initial presentation.
  - The presentation of AD can mimic a variety of other conditions including acute coronary syndrome, pulmonary embolism, cerebrovascular accident, refractory shock, hypertensive emergency, abdominal catastrophe, or acute limb ischemia.
  - Delay in diagnosis or failure to consider AD contributes to its high mortality.
- **Mortality from AD occurs via 4 pathways:**
  - Acute cardiac tamponade - Intrapericardial rupture of the ascending AD.
  - Acute aortic insufficiency - Unseating of the aortic valve can produce immediate cardiogenic shock, in contrast with chronic aortic insufficiency, which is hemodynamically tolerated to a greater extent.
  - Aortic free rupture - Usually into the left pleural space.
  - End-organ ischemia - Any organ can be affected.

- AD is classically categorized by the **Stanford system** figure :
  - Type A dissection - involves the ascending aorta.
  - Type B dissection - involves the descending aorta only, effectively distal to the left subclavian artery.
- An alternative classification is the **DeBakey system** figure :
  - Type I dissection - originates in the ascending aorta, through the aortic arch, and continuing into the descending or abdominal aorta.
  - Type II dissection - originates in the ascending aorta but does not extend into the descending aorta.
  - Type IIIa dissection - originates in the descending aorta but does not extend into the abdominal aorta.
  - Type IIIb dissection - originates in the descending aorta and continues into the abdominal aorta.
- **CT angiography of the aorta is the preferred diagnostic study in the ED** but does not formally evaluate for the presence or absence of valve competence (ie, aortic valve insufficiency).
- **Bedside echocardiography** emrap-video is an important diagnostic tool in the unstable patient.

PITFALLS ◆

- ED treatment consists of reducing the pulse pressure-related shear force ($\Delta p/\Delta t$) through pharmacologic rate control followed by blood pressure control along with emergent surgical consultation.
  - If appropriate surgical services are not locally available, emergent interfacility transfer is indicated.
- Definitive management is dependent on AD classification.
  - Ascending dissection requires urgent surgery in most cases (Stanford Type A and DeBakey Types I and II).
  - Descending dissection is usually treated medically (Stanford Type B and DeBakey Types IIIa and IIIb). However, if the descending dissection compromises a major branch vessel (eg, superior mesenteric artery, renal artery, iliac artery), then surgery is typically required.
- Outcomes for patients with acute aortic disorders are heavily dependent on timely diagnosis and intervention, whether surgical or medical.

Diagnosis

- The most common presenting symptom of AD is pain.
  - Pain is classically described as sudden, severe, and of maximal intensity at onset. However, this description is not universal; some patients may also present with a more gradual onset of mild pain that is similar in description to musculoskeletal ailments of the thorax, back, or groin.
  - Pain originates and is maximal under the sternum in ascending dissection. The pain from descending dissection often originates between the shoulder blades or the upper thoracic back and may migrate distally toward the abdomen and legs.
  - Chest pain associated with AD is more commonly described as sharp and not the classic description of tearing or ripping. Tearing or migrating pain is estimated to be present in only 30%-40% of cases.※
  - Pain above and below the diaphragm, or chest or abdominal pain with neurologic signs or symptoms such as syncope, should increase suspicion for AD.

PITFALLS ◆

- As many as 10% of patients present without any pain at all. Cases of painless AD have been reported, although they are usually accompanied by other findings and are more likely to present with neurologic symptoms; thus, although painless dissection may occur, a truly *asymptomatic* dissection likely does not exist.

- **Important patient history clues suggestive of increased risk for AD include:**
  - Family history of aortic aneurysm, AD, bicuspid aortic valve, or sudden cardiac death
  - Personal history of bicuspid aortic valve
  - Connective tissue disease (eg, Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz disease).
  - Uncontrolled hypertension
  - Cocaine abuse
  - Pregnancy
  - Thoracic trauma
  - Aortic coarctation
  - Iatrogenic (eg, cardiac catheterization, aortic valve replacement).

Physical Examination

PITFALLS ◆

- **Physical examination may be completely normal upon presentation.**
  - Examination findings classically associated with thoracic AD are present in less than one-third of all cases.

- **~40% of patients with a dissection will demonstrate an aortic insufficiency/regurgitant murmur**, reflecting unseating of the aortic valve by an ascending AD. This murmur can be quite subtle and difficult to detect.
- Patients can present in shock, either hemorrhagic shock from ruptured AD and subsequent exsanguination or obstructive shock secondary to cardiac tamponade associated with aortic rupture into the pericardium.
- Syncope or prolonged unconsciousness can be the initial presentation of patients with cardiac tamponade. Jugular vein distention, pulsus paradoxus, muffled heart sounds, and grayish discoloration of the face may be seen in these patients.
- Pulse and blood pressure considerations:
  - Pulse deficits or focal neurological deficits significantly increase the likelihood of AD in the appropriate clinical setting.
  - 30% of patients with a Stanford Type A dissection and 15% with a Type B dissection will demonstrate a pulse deficit.
  - There may be absent or diminished pulses in the carotid, brachial, and/or femoral arteries.
  - A difference in blood pressure between the two arms of at least 20 mm Hg suggests involvement of aortic branch arteries.
- Death, syncope, or hemiplegia may occur after occlusion of one or both carotid arteries. AD may present as stroke-like symptoms in 4.7% of cases.※
- Anuria may be present secondary to disturbed renal perfusion.
- Paraplegia or quadriplegia may be present secondary to occlusion of the vessels feeding the anterior spinal arteries.
- The physical stigmata seen in connective tissue disease may be observed. See: **Deep Dive - Physical Examination** for detailed discussion of such physical examination findings).

PEARLS

- The presence of the following positive findings was found to increase the probability of AD:※
  - Tearing or ripping pain
  - Migrating pain

- Sudden-onset chest pain
- Focal neurological deficits
- Pulse deficit

- The presence of the following negative findings was found to decrease the probability of AD:※
  - Absence of sudden-onset chest pain
  - Pain not described as tearing or ripping
  - Chest X-ray (CXR) with a normal appearing aorta and normal mediastinum
- Most clinical findings associated with AD are insensitive in isolation. However, a combination of findings increases the accuracy of the clinical assessment for AD.

Laboratory Evaluation

- Routine hematologic investigations are likely of limited utility in establishing the diagnosis of AD. However, such investigations may demonstrate acute organ dysfunction (eg, renal failure, hepatitis secondary to ischemia, or lactic acidosis associated with hypoperfusion) which may be helpful in establishing the severity of the patient's dissection.
- A **type and screen with crossmatch** is essential for operative management and should be collected in the ED.

PITFALLS ◆

- Caution: Do not rely on the D-dimer assay alone to exclude a diagnosis of acute AD due to the limited evidence of its accuracy (American College of Emergency Physicians Clinical Guidelines, Level C).
- As many as 18% of AD patients exhibit an elevated troponin level. Chest pain with an elevated troponin does not reliably exclude AD.

Imaging Tests

- The classically described CXR abnormalities associated with AD are not reliably present; furthermore, such abnormalities are non-specific and inadequate for either ruling in or ruling out AD.
- **CT angiography** slideshow **is the imaging modality of choice for the diagnosis of AD**, given its speed, availability, high sensitivity and specificity, and the wealth of information provided by CT angiograms.

- CT angiography of the aorta is reported to have a sensitivity of nearly 100% and a specificity of 100% for acute AD.
- American College of Emergency Physicians (ACEP) Clinical Guidelines support the use of CT angiography of the aorta as equivalent in diagnostic accuracy to transesophageal echocardiogram (TEE) or magnetic resonance angiography (MRA) (ACEP Clinical Guidelines, Level B recommendation).

- **Transthoracic echocardiogram** image is insufficient for the definitive diagnosis of thoracic AD due to its inherent insensitivity secondary to technical limitations in the ability to visualize the aortic arch and descending aorta.
  - ACEP Clinical Guidelines recommend not relying on an abnormal bedside transthoracic echocardiogram for the definitive diagnosis of thoracic AD (ACEP Clinical Guidelines, Level B). However, the guidelines do provide a consensus recommendation that if an AD is identified on limited bedside ultrasound, then surgical consultation and/or transfer to a higher level of care capable of definitively managing AD should occur (ACEP Clinical Guidelines, Level C).
- TEE can be considered to establish the diagnosis of AD. However, TEE requires specialized equipment, clinicians, and sedation, which may prevent a timely diagnostic study. Thus, TEE is not recommended as an initial imaging study in the ED. The sensitivity of TEE is between 86% and 100% with a specificity of 90% to 100%.
- MRA of the aorta has a high sensitivity (95%-100%) and high specificity (94%-98%) for thoracic AD. However, MRA is a specialized exam with limited availability, as well as longer image acquisition time, which could delay diagnosis.

Treatment

- Ensure that the patient is nothing-by-mouth, then obtain large-bore IV access (consider arterial line placement).
- Provide adequate analgesia with opiates, if the patient's hemodynamic status allows.

PITFALLS ◆

- **If the patient is hypertensive and/or tachycardic, rapidly reduce heart rate to 60 beats per min with β-blockade, then target SBP <120 mm Hg by using an antihypertensive agent:**
  - 2-drug strategy: First, β-blockade; second, antihypertensive agent.
    - β-blockade
      - **Esmolol IV** is preferred if rapidly available for its titratability given as 500 µg/kg bolus over 1 min followed by 50 µg/kg/min continuous infusion; may titrate up by 25-50 µg/kg/min every 5-15 min to a max of 300 µg/kg/min **then**

- Antihypertensive agent
  - **Nicardipine IV** 5 mg/h; may titrate up by 2.5 mg/h every 5-15 min to a max of 15 mg/h, **or**
  - **Clevidipine IV** initial dose 1-2 mg/h IV infusion, titrate up by doubling the dose at 90-s intervals initially; once approaching target blood pressure, increase by less than double dose and lengthen dose interval to 5-10 min, to a max of 32 mg/h, **or**
  - **Sodium nitroprusside IV** 0.3 μg/kg/min, titrate up by 0.5 μg/kg/min every 5 min to a maximum of 10 μg/kg/min for a max duration of 10 min, **or**
  - **Fenoldopam IV** 0.01-0.03 μg/kg/min, titrate up by 0.05-0.1 μg/kg/min every 15 min to a max of 1.6 μg/kg/min.
- 1-drug strategy:
  - Labetalol IV 10-20 mg bolus over 2 min, then 20-80 mg bolus every 10 to 15 min to a max of 300 mg, **or** initiate labetalol continuous infusion at 0.5-2 mg/min, titrate up by 0.5 mg/min every 10 min to a max of 10 mg/min.
  - If the use of a β-blockade is contraindicated secondary to bronchospasm, then consider a calcium channel blocker.
    - Diltiazem IV 0.25 mg/kg bolus over 2 min, then initiate a continuous infusion at 5 mg/h, titrating up by 5 mg/h every 30 min to a max of 15 mg/h.

- **Immediate surgical consultation should occur for all ADs, regardless of location.**
  - Stanford Type A AD, or Type B with evidence of limb or end-organ ischemia, requires immediate operative intervention.
  - Type B AD may be managed medically in the absence of end-organ ischemia or limb ischemia.

Disposition

- A patient with acute AD is typically not discharged home unless the goals of care are inconsistent with operative treatment or medical management.
- Anticipate patient transfer to the operating room with cardiothoracic or vascular surgery, or transfer the patient to a facility with access to these subspecialties.
- All patients with acute AD who are receiving IV medications require ICU admission.
- If transfer is required for management, the most expeditious means is encouraged (ie, air versus ground) based on availability, distance, local travel issues, weather, and so on.

Charting Pearls

PEARLS

- AD is a time-dependent disease. Note the key time intervals and interventions:
  - Time from the onset of symptoms
  - Time to order, perform, receive, and interpret imaging
  - Time to order and initiate blood pressure control
  - Time to receive surgical or radiology consultation
  - Time to request emergency transfer; time to initiate emergency transfer

Deep Dive

Background

Epidemiology

- AD is estimated to occur in 2.9 to 3.5 per 100,000 patients per y.※
- The mean age of onset for AD is 63 y of age.※
- Two-thirds of ADs occur in males.※
- In individuals less than 40 y of age, 59% of ADs are associated with either Marfan syndrome or bicuspid aortic valve.※
- **Risk factors associated with AD include:**※
  - Hypertension (76%)
  - Atherosclerosis (27%)
  - Aortic aneurysm (16%)
  - Previous cardiac surgery (16%)
  - Marfan syndrome (5%)
  - Iatrogenic injury (4%)
  - Cocaine use (1.8%)
  - In women of childbearing age, pregnancy imparts a 4-fold increased risk of AD compared with non-pregnant females, with an estimated risk of 5.5 per 1,000,000 versus 1.7 per 1,000,000.※
  - Similar to larger registry data, individuals with hypertension and/or connective tissue diseases are more likely to develop an acute AD.※

- AD more frequently occurs between 6 a.m. and 12 p.m., peaking at 9 a.m., and during the winter months, with a peak in January.※
- Mortality considerations:
  - The in-hospital overall mortality for Stanford Type A dissections is estimated to be 22% and for Stanford Type B dissections is 14%.※
  - AD mortality is estimated to increase by 1% to 2% per h following the onset of symptoms.※
  - Mortality is higher for patients presenting with painless acute AD. These patients are also more likely to present with neurologic symptoms.※
  - The factors that increase in-hospital mortality include shock, refractory hypertension, recurrent pain, mesenteric malperfusion, pericardial tamponade, and periaortic hematoma. Other factors include an age greater than 70 y, migrating pain, ischemic changes on ECG, and the presence of pulse deficit.※

Pathophysiology

- The aorta is histologically composed of 3 predominant layers: the tunica intima (innermost), tunica media, and tunica adventitia (outermost).
  - The tunica media gives the aorta its elasticity.
  - Normal aging leads to medial degeneration of the aorta, with hypertension accelerating this process.
- AD typically occurs when the integrity of the tunica intima is disrupted, allowing pulsatile blood to dissect through the tunica media, leading to the creation of a false lumen.
- The pulsatile aortic flow propagates the dissection, typically in an antegrade fashion, from the intimal tear.
- Fenestrations between the true lumen and false lumen can occur downstream from the initial intimal defect, typically near the ostia of vessels branching off the aorta, maintaining false lumen patency.※
- Areas of repeated mechanical stress associated with the cardiac cycle as well as higher hydrodynamic forces, such as the ascending aorta and first portion of the descending thoracic aorta, are at increased risk of AD.※
- Two widely accepted **classification systems** figure exist for ADs:
  - Stanford System - Classified by the proximal site of the dissection alone:※
    - Type A dissections involve the ascending aorta.
    - Type B dissections involve the descending aorta, effectively distal to the left subclavian artery.

- DeBakey System - Classified by both the proximal site of the dissection as well as the distal extent of the dissection:※
  - Type I Dissection - originates in the ascending aorta, through the aortic arch, continuing into the descending or abdominal aorta.
  - Type II Dissection - originates in the ascending aorta, does not extend into the descending aorta.
  - Type IIIa Dissection - originates in the descending aorta, does not extend into the abdominal aorta.
  - Type IIIb Dissection - originates in the descending aorta, extends into the abdominal aorta.
- 67% of ADs are Stanford Type A with 33% being Stanford Type B.※
- Congenital anomalies of the aortic valve increase the probability of AD:※
  - Bicuspid aortic valves are found in approximately 1% to 2% of the general population; however, bicuspid aortic valves are found in approximately 9% to 13% of patients with AD.
  - A bicuspid aortic valve potentially reorients the normal laminar flow toward the wall of the aorta, producing local injury and making the wall more susceptible to ascending dissection.
- Mortality from AD is typically associated with the complications associated with dissection formation:※
  - **Acute cardiac tamponade** (intrapericardial rupture of ascending AD).
  - **Acute aortic insufficiency** (unseating of aortic valve can produce immediate cardiogenic shock as opposed to better-tolerated chronic aortic insufficiency).
  - **Aortic free rupture** (usually into the left pleural space).
  - **End-organ ischemia** (any organ can be affected).
- Cardiac tamponade occurs in 18.7% of patients with AD and is an extremely poor prognostic indicator.
  - In-hospital mortality associated with tamponade secondary to AD is 54% versus 24.6% for those without tamponade.※
- Acute aortic insufficiency occurs in as many as 32% of patients and is more common in patients with Stanford Type A dissections.※
- Free rupture of the aorta is reported to occur in approximately 18% of ADs and can lead to rapid exsanguination and death.※

- End-organ ischemia/malperfusion syndromes occur in roughly one-third of patients with AD and are driven by two major processes:※
  - A branch vessel off the aorta remains fed by the true lumen, although narrowing of the ostium secondary to the hydrostatic pressure of the false lumen leads to impaired flow and thrombosis of the branch vessel.
  - A branch vessel off the aorta is fed by the false lumen, while the dissection extends into the branch vessel, leading to impaired perfusion.
- Hypoperfusion of the mesentery secondary to AD is relatively rare, yet imparts significant mortality risk to the patient.
  - Mesenteric ischemia is found in approximately 3.7% of patients with a Stanford Type A dissection, with an overall in-hospital mortality of 63.2% versus 23.8% in those without mesenteric ischemia.※
  - Mesenteric ischemia is found in approximately 7.1% of patients with a Stanford Type B dissection with an overall in-hospital mortality of 30.8% versus 9.1% in those without mesenteric ischemia.※
- Myocardial infarction is rare but reported in as many as 3% of cases of AD, typically presenting as an inferior or posterior myocardial infarction secondary to involvement of the right coronary artery.※
- Acute limb ischemia is commonly reported with AD, with one study reporting rates of approximately 2.5% for upper limb ischemia and 11.7% for lower limb ischemia.※
- Approximately 6% of individuals presenting with a Stanford Type A AD present with clinical evidence of stroke.※
- A distinct subtype of aortic pathology relevant to AD is the formation of an intramural hematoma without an identifiable dissection flap. Intramural hematomas can reabsorb, progress to aneurysm formation, or lead to dissection. Intramural hematomas in the ascending aorta have mortality rates that mimic ascending AD.※

Diagnostic Considerations

Clinical Presentation

- AD is a challenging diagnosis and is missed upon initial presentation in as many as one-third of patients. AD is most often mistaken for an acute coronary syndrome.※
- The presentation of AD can mimic several other conditions including pulmonary embolism, cerebrovascular accident, refractory shock, hypertensive emergency, abdominal catastrophe, or acute limb ischemia.
- The most common presenting symptom of AD is pain.※

- The pain associated with AD is classically described as sudden in onset and severe. Registry studies report that 95.5% of individuals with AD report pain, with 84.8% of all individuals with AD describing an abrupt onset of pain; 90.6% of the individuals studied reported their pain as “severe or worst ever.” Only 50.6% of the individuals described a “tearing or ripping pain,” with 64.4% reporting a sharp pain.※
- This description of pain is not universal, because patients may also present with a more gradual onset of mild pain that is similar in description to musculoskeletal ailments of the thorax, back, or groin.※
- The pain is often said to be preceded by either acute physical exertion or an acute emotional event.

- Cases of painless AD have been reported, with registry studies suggesting that 5% to 10% of patients present without any pain at all.※ Typically, these presentations of AD are confounded by neurologic symptoms, including altered mentation.※ Thus, a true “painless” AD is questioned and likely does not occur.
- The location of the pain may suggest the location of the dissection.※
  - In ascending dissection, the pain is thought to originate and be maximal at the anterior chest just under the sternum.
  - In descending dissection, the pain is thought to originate between the shoulder blades in the upper thoracic back, and may migrate distally toward the abdomen and legs.
  - The pain typically changes as the dissection extends or evolves.
- It is important to consider AD for any pain above and below the diaphragm as well as chest or abdominal pain with neurologic signs or symptoms, including syncope.
- **Important patient history clues suggestive of increased risk for AD include:**
  - Family history of aortic aneurysm, AD, bicuspid aortic valve, or sudden cardiac death
  - Personal history of bicuspid aortic valve
  - Connective tissue disease (eg, Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz disease)
  - Uncontrolled hypertension
  - Cocaine abuse
  - Pregnancy
  - Thoracic trauma
  - Aortic coarctation
  - Iatrogenic (eg, cardiac catheterization, aortic valve replacement).

Physical Examination

- The physical examination may be completely normal upon presentation. **The exam findings classically associated with thoracic AD are present in less than one-third of all cases.**※
- Despite a large number of case studies in AD, the role of clinical examination has yet to be prospectively evaluated in an independent and blinded study.
- Cardiovascular Findings:
  - Approximately 40% of patients with a dissection demonstrate an aortic insufficiency/regurgitant murmur, reflecting unseating of the aortic valve by an ascending AD.※
  - Patients may present in shock, suggesting hemorrhage from a ruptured AD leading to internal bleeding or intrapericardial rupture leading to cardiac tamponade. Jugular vein disorder, pulsus paradoxus, muffled heart sounds, and grayish discoloration of the face may be seen in a patient with cardiac tamponade.※
  - Blood pressure at presentation is variable with 49% of individuals being hypertensive (SBP ≥150 mm Hg), 34.6% normotensive, 8.0% hypotensive (<100 mm Hg), and 8.4% in frank shock (SBP ≤80 mm Hg).※
  - Either syncope or prolonged unconsciousness can be the initial presentation of patients with cardiac tamponade.
  - 30% of patients with a Stanford Type A dissection and 15% with a Type B dissection demonstrate a pulse deficit.※ There may be absent or diminished pulses in the carotid, brachial, and femoral arteries. A difference in blood pressure between the two arms of at least 20 mm Hg may be found when there is involvement of aortic branch arteries.※ However, the finding of unequal blood pressures is non-specific.
  - Pulse deficits (positive likelihood ratio, 5.7; 95% confidence interval, 1.4-23) or focal neurological deficits (positive likelihood ratio, 6.6-33) significantly increase the likelihood of AD in the appropriate clinical setting.
  - Anuria may be present secondary to impaired renal perfusion.
- Neurologic Findings:
  - Death, syncope, or hemiplegia may occur after occlusion of one or both carotid arteries. AD may present as stroke-like symptoms in 4.7% of cases.※
  - Paraplegia or quadriplegia can result after the occlusion of vessels feeding the anterior spinal arteries.
- Physical stigmata concerning for underlying connective tissue disease, increasing the likelihood of AD include

- Tall, thin body, long digits, suggestive of Marfan and non-Marfanoid connective tissue disease.
- Pectus excavatum or carinatum.
- Thumb-palm sign - Thumb stretches beyond the medial border of the flat palm of the hand, indicating excessively long bones and lax joints.
- Steinberg sign (thumb sign) - Tip of the thumb is visible medial to the little finger when the hand is clasped in a clenched fist.
- Hypermobility or “double jointedness,” suggestive of lax connective tissue.

- Available registry data allows the sensitivity of specific clinical history, physical examination, and chest radiography findings to be estimated, although likely overestimating the accuracy of these diagnostic methods through selection bias of more obvious cases. A small number of studies estimate the specificity of components of the clinical exam but remain limited by the lack of independence between the selection of patients for study and clinical findings.※
  - The presence of the following positive findings was found to increase the probability of AD (positive likelihood ratio; 95% confidence limits):※
    - Tearing or ripping pain (10.8; 5.2-22.0)
    - Migrating pain (7.6; 3.6-16.0)
    - Sudden-onset chest pain (2.6; 2.0-3.5)
    - Focal neurological deficits (33; 6.6-33.0)
    - Pulse deficit (5.7; 1.4-23.0)
  - The presence of the following negative findings was found to decrease the probability of AD (negative likelihood ratio; 95% confidence limits):※
    - Absence of sudden-onset chest pain (0.3; 0.2-0.4)
    - Pain not described as tearing or ripping (0.4; 0.3-0.5)
    - CXR without large aorta or wide mediastinum (0.13; 0.02-1.00)
  - Most of the clinical findings associated with AD are insensitive in isolation. However, a combination of findings significantly increases the accuracy of clinical assessment for AD.

Electrocardiography

- **ECG findings** slideshow consistent with acute myocardial infarction, including ST-elevation myocardial infarction (STEMI), do not rule out AD. AD patients may have a normal ECG or their ECG could show the following:

- Non-specific ST-segment and T wave abnormalities
- ST-segment deviations, including elevation and depression
- New Q waves
- Left ventricular hypertrophy
- Atrial fibrillation or other tachydysrhythmia

- Acute myocardial infarction, including **STEMI** ecg , can occur after occlusion or dissection into coronary arteries (most commonly, the right coronary artery) associated with extension of the dissection flap.
  - Inferior STEMI is the most common presentation.

Radiographic Evaluation

- The classically described CXR abnormalities associated with AD are not reliably present, in addition to being non-specific and inadequate for ruling in or ruling out AD. ※
  - CXR may be useful to exclude other life-threatening causes of acute chest pain such as pneumothorax.
- A majority of patients with AD have abnormal CXR findings, so that a completely normal CXR result helps to decrease the likelihood of the disease (negative likelihood ratio, 0.3; 95% confidence limits, 0.2-0.4).※
  - However, 12.4% of cases of thoracic AD are reported to have a normal CXR.※
- The classically taught **CXR** image abnormalities are:
  - Widening of the mediastinum (61.6% of cases)
  - Widening of the aortic knob or abnormal contour (49.6% of cases)
  - Unilateral or bilateral pleural effusions (19.2% of cases)
  - The calcium sign - Separation of an intimal calcification from the outer border of the aortic knob by 1 cm or more (14.1% of cases).
- Absence of these classically taught CXR abnormalities may decrease the probability of disease.
- **CT angiography** image is the imaging modality of choice for the diagnosis of AD, given its speed, availability, high sensitivity and specificity, and the wealth of information provided by its study.※
  - CT angiography of the aorta is reported to have a sensitivity of 100% and a specificity of 100% for acute AD.※

- The American College of Emergency Physicians (ACEP) Clinical Guidelines support the use of CT angiography of the aorta as equivalent in diagnostic accuracy to TEE or MRA with a Level B recommendation.※

- Cardiology-based **transthoracic echocardiogram** is typically regarded as having insufficient sensitivity (73.7% to 100%, median 86.9%) or specificity (71.2% to 91.0%, median 81.1%) for making a definitive diagnosis of thoracic AD.※
  - Transthoracic echocardiogram is insensitive due to limitations in visualizing the aortic arch and descending aorta, as well as the potential for the patient's body habitus to limit imaging.
  - The ACEP Clinical Guidelines have given a Level B recommendation to not rely on an abnormal bedside transthoracic echocardiogram for a definitive diagnosis of thoracic AD.
  - The ACEP Clinical Guidelines provide a Level C consensus recommendation that if an AD is identified upon limited bedside ultrasound, surgical consultation and/or transfer to a higher level of care capable of definitively managing AD should occur.※
- **TEE** can be considered to establish the diagnosis of AD. However, TEE requires specialized equipment, clinicians, and sedation, which may prevent a timely diagnostic study.
  - The sensitivity of TEE is between 86% and 100% with a specificity of 90% to 100%.※
- **MRA** of the aorta has a high sensitivity (95%-100%) and high specificity (94%-98%) for thoracic AD. However, MRA of the aorta is a specialized exam with limited availability as well as a longer image acquisition time, which could delay diagnosis.※
  - MRA may be of higher utility for monitoring patients who are not undergoing operative management or patients in the postoperative surveillance period.※

Laboratory Evaluation

- Routine hematologic investigations are likely of limited utility in establishing the diagnosis of AD. However, such investigations may be helpful in establishing the severity of the patient's dissection in specific cases, such as acute renal failure, acute hepatitis secondary to ischemia, or lactic acidosis associated with hypoperfusion.
- A **type and screen with crossmatch** is essential for operative management and should be collected in the ED.
- Efforts continue to identify laboratory investigations that can help to rapidly identify individuals with AD.
  - Particular interest has been placed on the serum **D-dimer**, which, when <500 ng/mL [2.74 nmol/L] within 24 h of symptom onset, has demonstrated a negative likelihood ratio of 0.07 for AD.※

- Unfortunately, detectable serum D-dimer levels are significantly less common if a dissection flap does not exist and intramural hematoma is the only pathologic finding upon imaging.
- Because outcomes are similar between classic AD and intramural hematoma, the reduced specificity for intramural hematoma variants is of concern; as such, D-dimer testing to rule out AD does not meet the current standard of care.
- The American College of Emergency Physicians (ACEP) Clinical Guidelines have issued a Level C recommendation cautioning physicians not to rely on the D-dimer assay alone to exclude a diagnosis of acute AD.※

- Emerging research suggests that coupling the Aortic Dissection Risk Score, a clinical decision instrument, with a negative D-dimer assay may help to reduce unnecessary evaluation for AD in low risk populations. A prospective validation study is currently required prior to clinicians routinely applying this strategy for ruling out AD in low risk individuals without the use of an imaging study.※

Therapeutic Considerations

- Timely operative intervention in AD is key, as mortality is estimated to increase by 1% to 2% per h following the onset of symptoms.※
- After the diagnosis of AD in the ED, efforts should be made to control the patient's heart rate, followed by SBP, to reduce shearing stresses on the aortic intima.
  - Following adequate analgesia, a Class I recommendation based on expert consensus is to initiate intravenous β-blockade and titrate to a heart rate of 60 beats/min.
  - If the SBP remains >120 mm Hg following the initiation of β-blockade, an intravenous vasodilatory agent should be initiated to reduce the SBP to <120 mm Hg while maintaining adequate end-organ perfusion.※
    - 2-drug strategy: First, β-blockade; second, antihypertensive agent.
      - β-blockade
        - **Esmolol IV** is preferred if rapidly available for its titratability given as 500 μg/kg bolus over 1 min followed by 50 μg/kg/min continuous infusion; may titrate up by 25-50 μg/kg/min every 5-15 min to a max of 300 μg/kg/min, **then**
      - Antihypertensive agent
        - **Nicardipine IV** 5 mg/h; may titrate up by 2.5 mg/h every 5-15 min to a max of 15 mg/h, **or**
        - **Clevidipine IV** initial dose 1-2 mg/h IV infusion, titrate up by doubling the dose at 90-s intervals initially; once approaching target blood pressure, increase by

less than double dose and lengthen dose interval to 5-10 min, to a max of 32 mg/h **or**

- **Sodium nitroprusside IV** 0.3 µg/kg/min; may titrate up by 0.5 µg/kg/min every 5 min to a max of 10 µg/kg/min for a max duration of 10 min, **or**
- **Fenoldopam IV** 0.01-0.03 µg/kg/min; may titrate up by 0.05-0.1 µg/kg/min every 15 min to a max of 1.6 µg/kg/min.

- 1-drug strategy:
  - **Labetalol IV** 10-20 mg bolus over 2 min, then 20-80 mg bolus every 10 to 15 min to a max of 300 mg, **or** initiate labetalol continuous infusion at 0.5-2 mg/min, titrate up by 0.5 mg/min every 10 min to a max of 10 mg/min.
  - Labetalol is often inadequate by itself for blood pressure control.
- If there is evidence of acute aortic insufficiency, significant caution must be used when initiating β-blockade, because this intervention may blunt the patient's compensatory tachycardia, leading to cardiovascular collapse.※
- If β-blockade is contraindicated secondary to bronchospasm, consider a calcium channel blocker.※
  - Diltiazem IV 0.25 mg/kg bolus over 2 min, then initiate a continuous infusion at 5 mg/h, titrating up by 5 mg/h every 30 min to a max of 15 mg/h.

- The American College of Emergency Physicians (ACEP) Clinical Guidelines support recommendations to reduce the heart rate to 60 beats per min and SBP to <120 mm Hg. However, the guidelines note that no specific heart rate or blood pressure goal has demonstrated a reduction in morbidity or mortality.※

- While initiating heart rate and blood pressure control, urgent surgical consultation is advised for all ADs. Patients with Stanford Type A ADs should be taken for emergency surgical repair, whereas Stanford Type B ADs can be managed medically unless there is evidence of malperfusion syndrome, progression of dissection, enlarging aneurysm, or inability to control the blood pressure or pain.※
  - For Stanford Type A ADs, there is a significant difference in survival for those treated surgically versus those managed medically with heart rate and blood pressure control.
    - In-hospital mortality for a Stanford Type A AD managed surgically is currently 22% versus 57% for cases managed medically.※
    - Currently, Stanford Type A AD are surgically managed in an open fashion with graft repair of the aortic root and arch and may require aortic valve replacement.
    - Endovascular approaches utilizing stent grafts are not currently Food & Drug Administration-approved for this purpose.※

- For Stanford Type B AD, initial management is focused on excellent heart rate and blood pressure control to avoid propagation of the AD.
  - Currently, in-hospital mortality for a Stanford Type B AD is approximately 13%.※
  - Approximately one-third of patients with a Stanford Type B AD develop complications that require operative intervention such as malperfusion syndromes, signs of imminent rupture, expansion of aorta size, or hemodynamic instability.※
  - There is emerging evidence that stent grafting via thoracic endovascular aortic repair may provide for improved outcomes compared with medical management of Stanford Type B AD.※

- Poor prognostic indicators have been identified for both Stanford Type A and Type B AD.※
  - For Stanford Type A ADs, a previous history of aortic valve replacement, migrating chest pain, presenting in shock or with tamponade physiology, limb ischemia, or hypotension are statistically significant predictors of in-hospital mortality.
  - For Stanford Type B ADs, the presence of mesenteric ischemia, presenting with hypotension or shock, acute renal failure, an aorta diameter larger than 5.5 cm, periaortic hematoma, limb ischemia, and older age are statistically significant predictors of in-hospital mortality.

- Given similar mortality rates for individuals with intramural hematomas compared with true AD, a Class IIa Consensus Guideline exists that individuals presenting with an intramural hematoma should be treated in a fashion similar to an individual with a true AD at the level of the hematoma.※

Care of Patients Following Repair of Aortic Dissection

- Prognosis following open surgical repair of a Stanford Type A AD is excellent, with a 96.1% survival rate at 1 y and 90.5% survival rate at 3 y after discharge from aortic repair.※
- Individuals medically managed for a Stanford Type B AD have a survival rate of only 78% at 3 y following initial hospital discharge.※
- Following the repair of a thoracic AD, meticulous management of hypertension, dyslipidemia, and tobacco use are instrumental in mitigating future risk.※
- A Class IIa consensus recommendation exists for follow-up after the repair of an AD or for individuals with a finding of intramural hematoma. Repeat CT angiography or MRA of the aorta is recommended at 1 mo, 3 mo, 6, mo, and 12 mo and then annually.※

Prevention

- For general populations without specific risk factors, the control of hypertension and hyperlipidemia, smoking cessation, and the avoidance of sympathomimetic agents (cocaine, methamphetamine) may reduce the risk of AD. Currently, there are no recommendations for routine surveillance to reduce the risk of AD in general populations.※
- For individuals with anatomic or genetic risk factors for AD, there are disease-specific screening, prevention, and treatment recommendations:
- Bicuspid Aortic Valve:
  - First-degree relatives of a patient with a known bicuspid aortic valve should have their aortic valve morphology and aorta evaluated for asymptomatic aortic disease.※
  - If the aortic valve is being repaired or replaced, aortic root or ascending aorta replacement is indicated when the aorta measures >4.5 cm or when the ratio of the maximal cross-sectional area of the ascending aorta (cm^2) to the patient's height (m) exceeds 10.※
- Family History of Thoracic Aortic Aneurysm or Aortic Dissection:
  - First-degree relatives of a patient with a thoracic aortic aneurysm or previous AD should undergo aortic imaging to identify those with asymptomatic disease.※
- Ehlers-Danlos Syndrome:
  - For individuals with the vascular form of Ehlers-Danlos syndrome (Type IV), surgical intervention is recommended when the aorta has expanded to a diameter of 4.0 to 5.0 cm. However, surgical repair is often complicated by fragile vascular tissue, impaired wound healing, and hemorrhage.※
- Loeys-Dietz Syndrome:
  - At the time of diagnosis, individuals should have complete aortic imaging with repeat imaging at 6 mo to establish the rate of change. Afterward, annual surveillance magnetic resonance imaging (MRI)/MRA of the vasculature from the brain to pelvis is recommended.※
  - Surgical replacement of the aorta is indicated if the aortic diameter becomes larger than 4.2 cm on TEE or 4.4 to 4.6 cm on CT or MRI/MRA.※
- Marfan Syndrome:
  - Echocardiography with attention to the aortic root is recommended at the time of diagnosis and 6 mo later to determine whether the ascending aorta has increased in size. For individuals with an aortic root larger than 4.5 cm, annual echocardiographic surveillance is recommended.※
  - All patients with Marfan syndrome and an aneurysmal aorta should be placed on a β-blocking agent and consideration should be given to adding an angiotensin receptor

blocker to reduce hemodynamic stress on the aorta.※

- In general, in Marfan syndrome, if the ascending thoracic aorta exceeds 5 cm in diameter or there is a greater than 0.5-cm increase in size per y, aortic root and ascending aorta replacement is indicated. Alternatively, if the ratio of the maximal cross-sectional area of the ascending aorta (cm^2) to the patient's height (m) exceeds 10, aortic root and ascending aorta replacement is recommended. Women with Marfan syndrome who are considering becoming pregnant are an exception to this recommendation. Instead, prophylactic replacement of the aortic root and ascending aorta is recommended if the diameter exceeds 4.0 cm.

- Thoracic aortic aneurysm:
  - For patients without a clear genetic or valvular etiology for their thoracic aortic aneurysm, aortic surgery is indicated when the aorta is 5.5 cm or larger or if an annual rate of change exceeds 0.5 cm per y.※
- Turner Syndrome:
  - At the time of diagnosis, individuals should undergo imaging of the heart and aorta with specific attention to evaluation of the aortic valve morphology, coarctation of the aorta, or dilation of the ascending aorta. If initial imaging is normal, follow-up imaging is recommended every 5 to 10 y. If initial imaging is abnormal, annual imaging and follow-up are recommended.※

Additional Information

Suggested EM:RAP/EMA Link

- **Aortic Dissection** audio

Selected Society Guidelines

- **Diagnosis and Treatment of Aortic Diseases**
- **Issues in the Evaluation and Management of Suspected Acute Nontraumatic Thoracic Aortic Dissection**
- **Diagnosis and Management of Thoracic Aortic Disease**

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