Chest Pain, Diaphoresis, and Dyspnea in a Hypertensive 53-Year-Old Man

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Aortic dissection is a life-threatening condition requiring urgent diagnosis and treatment. The initial challenge for the physician lies in distinguishing aortic dissection from more common conditions such as myocardial infarction that also are characterized by chest pain. Subsequent management depends on imaging techniques that define whether just the descending aorta is affected or its more proximal portions as well. Mortality and morbidity are high, especially when the ascending aorta is involved.

A 53-year-old man with a history of hypertension presented with a 6-hour history of substernal chest pain. The pain began abruptly while he was eating breakfast that morning. The pain was rated 9/10 in intensity and was associated with shortness of breath and diaphoresis. The pain did not radiate and he denied any alleviating or aggravating factors.

His past medical history was significant for a 10-year history of hypertension. His mother has hypertension and is 75 years of age. His father had a history of coronary artery disease and died from complications of prostate cancer at 72 years of age. The patient had a 25-pack-year history of tobacco smoking. He had consumed 6 beers daily for the past 30 years, and his last drink was 4 days prior to admission. Medications at the time

CME INFORMATION

TARGET AUDIENCE

The March/April Clinical Case of the Month is intended for family physicians, general internists, general practitioners, emergency medicine physicians, cardiologists, and cardiovascular surgeons.

EDUCATIONAL OBJECTIVES

After reading this article, physicians should be able to discuss the physiological mechanisms of acute aortic dissection, to identify the physical examination findings associated with aortic dissection, to identify the radiographic features of aortic dissection, and to describe the pharmacologic and surgical management of aortic dissection.

CREDIT

The LSMS Educational and Research Foundation designates this educational activity for a maximum of two (2) hours of category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

DISCLOSURE

Dr. Awtrey has nothing to disclose.
Dr. Gupta has nothing to disclose.
Dr. Kelly has nothing to disclose.
Dr. Glancy discloses that he is Editor of this journal.
Dr. Harrison has nothing to disclose.
Dr. Lopez discloses that he is a member of the LSMS *Journal* Board and the LSMS *Journal* Editorial Board.

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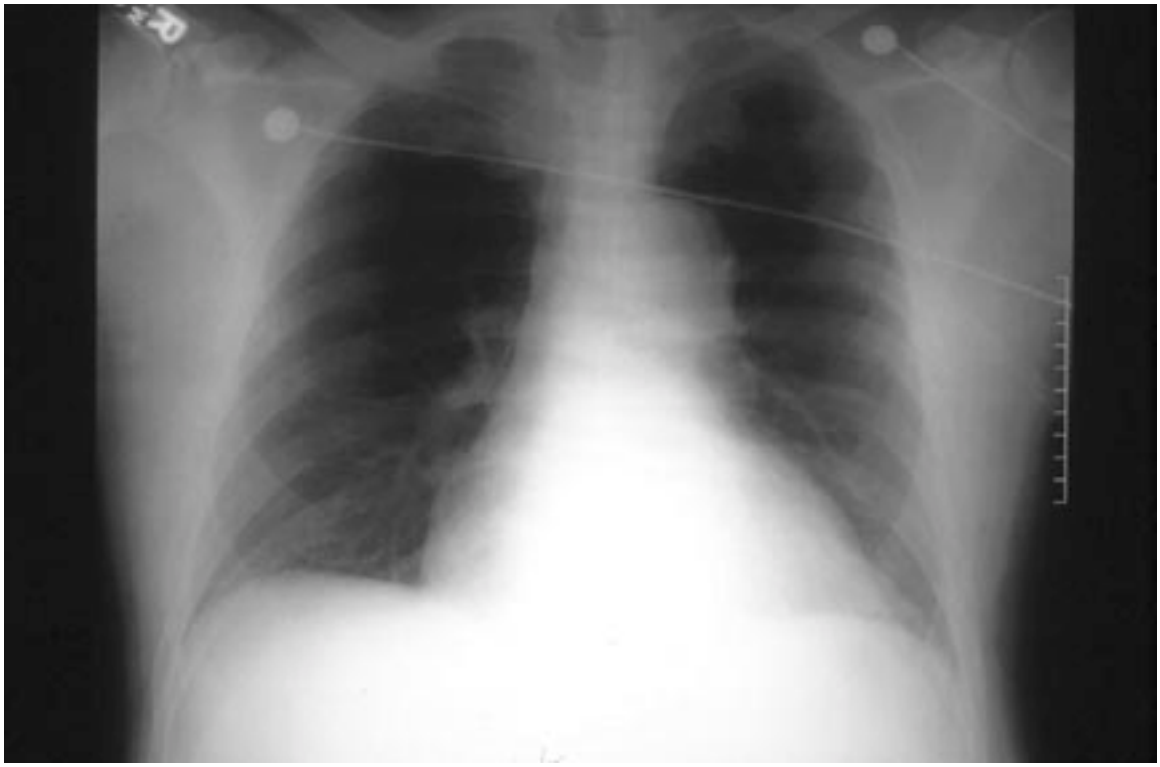

Figure 1. Portable anterioposterior radiograph of the chest showing a prominent cardiac silhouette and aortic knob.

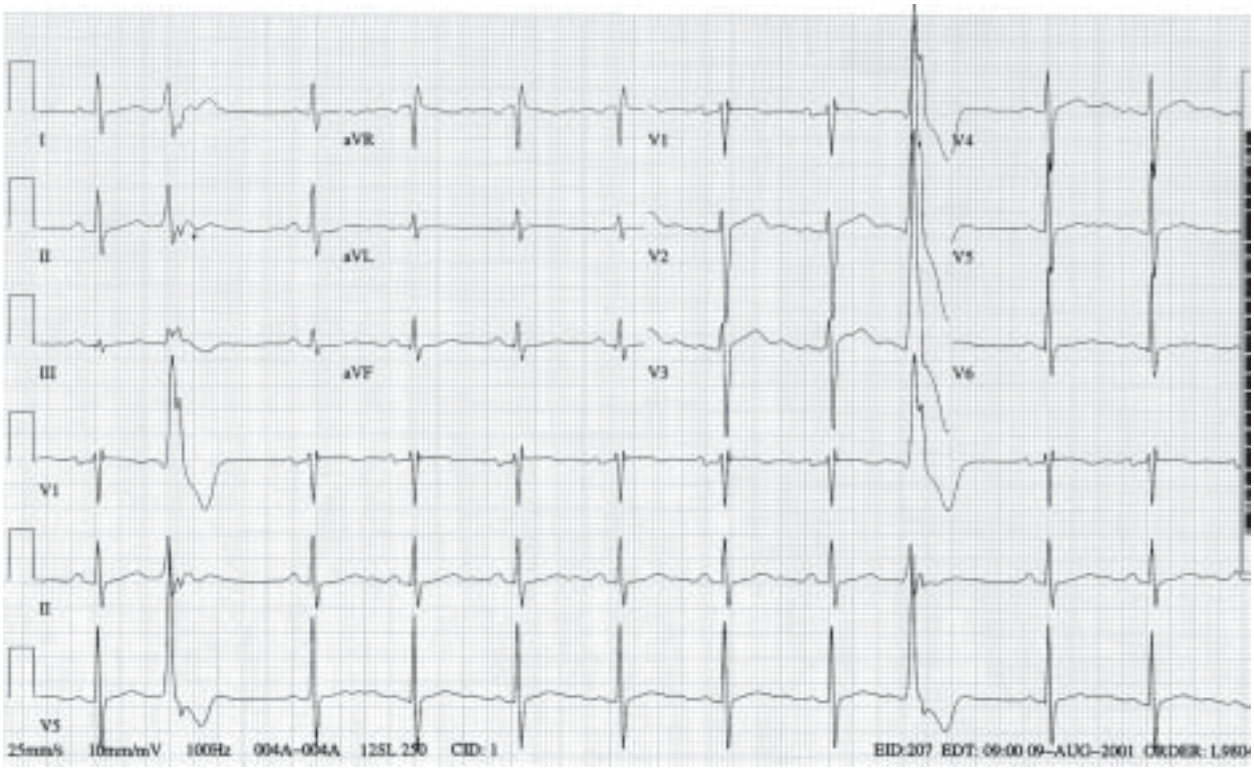

Figure 2. Twelve lead electrocardiogram showing normal sinus rhythm with occasional premature ventricular complexes, incomplete right bundle branch block, and nonspecific ST-T and U wave abnormalities.

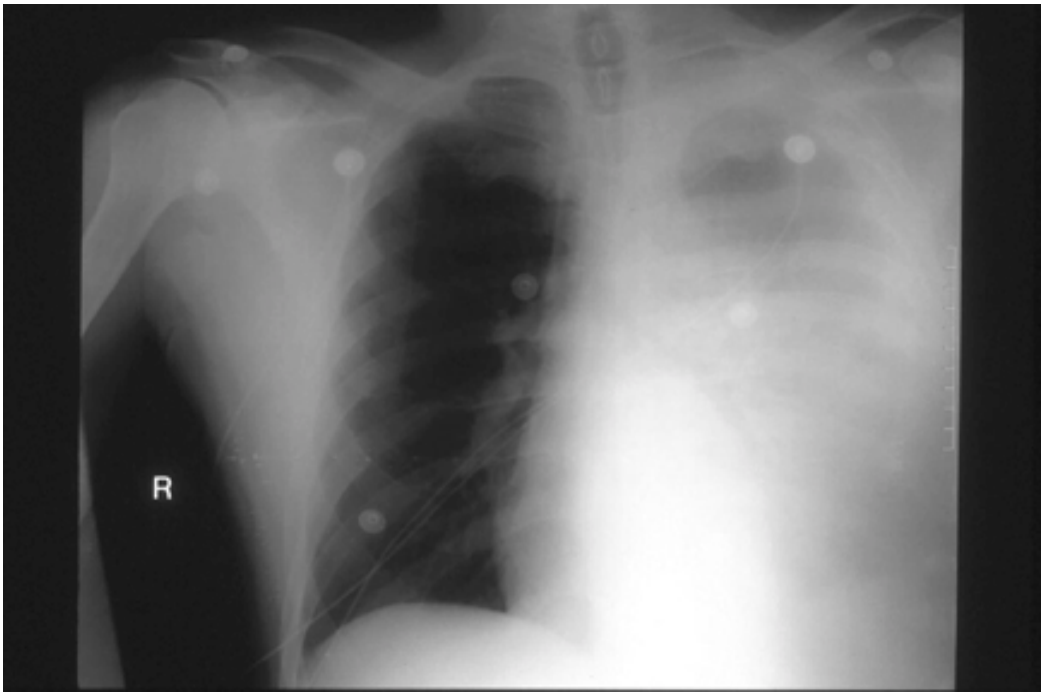

Figure 3. Portable anterioposterior radiograph of the chest showing interval development of large left pleural effusion.

of admission included clonidine. Review of systems revealed impotence for 1 year and dyspnea on exertion (1/2 block) for 1 month.

On initial physical examination, he was in no distress. His vital signs were temperature 98^0 F, a regular pulse of 68 beats per minute, blood pressure of 200/110 mmHg, 20 respirations per minute, and an oxygen saturation of 98% on 2 liters of oxygen per minute by nasal cannulae. Jugular venous pressure was estimated to be 8 cm H_2O. Cardiovascular examination was significant for a 1/6 systolic flow murmur heard only at the upper left sternal border. Auscultation revealed clear lung fields. Abdominal examination was without abnormality, and extremity exam revealed equal femoral and dorsalis pedis pulses bilaterally. A blood chemistry and complete blood cell count manifested only an abnormal potassium of 3.7 meq/L (3.8-5.2)and a MCV of 78 u^3 (80-100). Levels of cardiac enzymes obtained 3 times at 6-hour intervals were normal. Chest radiograph demonstrated a prominent cardiac silhouette and aortic knob (Figure 1), and a twelve-lead electrocardiogram was abnormal but showed no evidence of myocardial infarction (Figure 2). All other initial laboratory data were normal.

He was treated for an acute coronary syndrome with nitroglycerin paste, heparin, aspirin, and a beta blocker. He had relief of his chest pain, and the nitroglycerin was discontinued. Later his chest pain recurred along with hypotension and tachycardia requiring intravenous fluids and vasopressors. He subsequently complained of left-sided lower abdominal pain and numbness in his lower left leg, where pulses were no longer palpable. Chest radiograph performed at this time revealed a large left pleural effusion (Figure 3). A transesophageal echocardiogram revealed an aortic dissection (AD) at the level of the aortic arch (Figure 4). He was emergently taken to the operating room, but expired during surgery due to intra-abdominal aortic rupture with exsanguination.

DISCUSSION

AD typically affects patients in the sixth and seventh decades with a male-to-female ratio of 2:1. Acute AD often presents with the sudden onset of substernal chest pain that classically is described as severe and tearing with radiation to the back and infrascapular region. This pain is usually associated with diaphoresis and can migrate with propagation of the dissection. AD may also present with focal paresis, syncope, or dyspnea. There may be abdominal or extremity pain with or without chest or back pain. Pain both above and below the diaphragm suggests dissection.

The anatomical level of the aorta that is involved determines the classification of AD. Debakey classifies AD as type I when the ascending and descending aorta are involved, type II when the dissection involves only

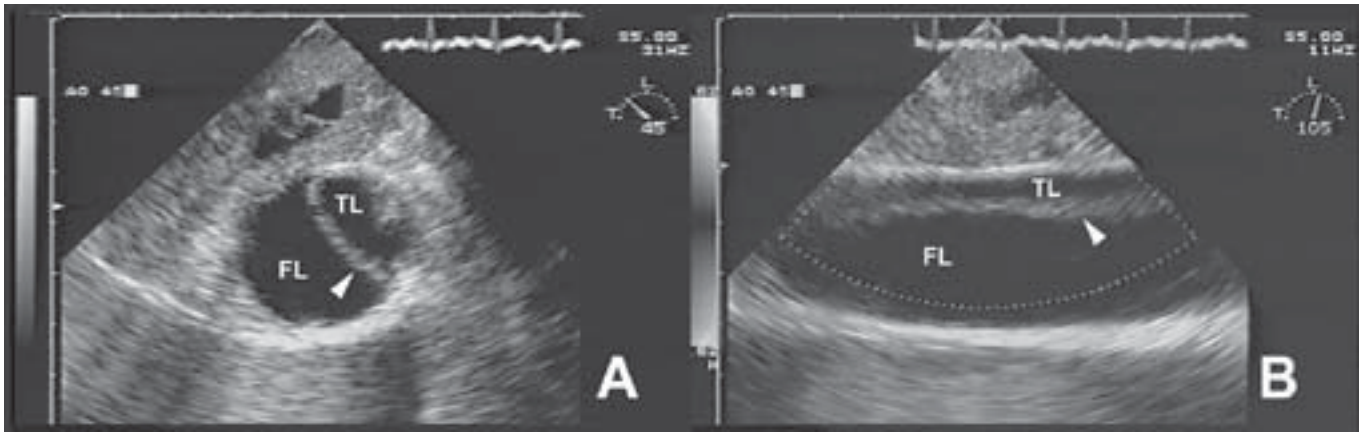

Figure 4: Still figures from the transesophageal echocardiogram showing a transverse view in systole (A) and a longitudinal view (B), both at the level of the proximal descending thoracic aorta. The true lumen (TL) is separated from the false lumen (FL) by the dissection flap (arrow head). In the transverse view systolic bulging of the flap from the TL into the FL correctly identifies each.

the ascending aorta, and type III when only the descending aorta is involved.[1,5,6] In the Stanford classification, type A describes any dissection involving the proximal aorta, and type B describes dissection limited to the distal aorta. Classification of the dissection dictates management options, ie, surgical versus medical.

Risk factors that predispose to AD include systemic hypertension and diseases leading to cystic medial necrosis (CMN)[6] of the aorta. CMN typically affects the proximal aorta resulting in circumferential weakness and dilatation, leads to aneurysms of the ascending aorta, and is prevalent in patients with the Marfan syndrome and Ehlers-Danlos syndrome type IV.[1] CMN may also be seen in pregnancy and with tricuspid aortic valves.

Physical findings suggestive of AD include hypertension or hypotension, loss of pulses, aortic regurgitation, pulmonary edema, and neurological findings due to carotid artery obstruction. In addition, hemiplegia, hemianesthesia, spinal cord ischemia, bowel ischemia, and myocardial ischemia have also been observed. The challenge for the physician lies in distinguishing AD from other conditions characterized by chest pain.

Acute aortic regurgitation is an important finding in patients with proximal or Stanford type A dissection. It results from a medial tear that widens the aortic root or a disruption of the leaflets by a dissecting hematoma.[1] Signs of aortic regurgitation include a widened pulse pressure, a diastolic murmur, and congestive heart failure. When AD involves the ascending aorta, chest radiographs may reveal a widened superior mediastinum. A new pleural effusion usually signifies aortic rupture. Electrocardiograms that show no evidence of ischemia may be helpful in distinguishing AD from myocardial infarction.

DIAGNOSIS

Aortography

Long considered the gold standard, aortography is 88% sensitive and 94% specific in identifying AD.[2] The diagnosis is based on direct angiographic signs that are considered diagnostic and indirect angiographic signs that are considered suggestive of the diagnosis. Direct signs include visualization of the double lumen or the intimal flap with indirect signs including compression of the true lumen by the false lumen, aortic wall thickening, and aortic regurgitation.[2] Advantages of aortography include its ability to detect many of the complications of aortic dissection, but disadvantages include a limited sensitivity, the risk of complications associated with intravascular contrast, and the delay of results.

Computed Tomography (CT)

Diagnosis of AD includes the use of both direct and indirect criteria. Sharing the same direct criteria with aortography, CT scans add widening of the aortic lumen and central displacement of intimal calcium deposits to the indirect criteria.[2] Sensitivity and specificity have been shown to be 83% to 94%, and specificity 97%.[2,4] Aside from the ready availability of CT scan in almost every hospital, other advantages include its noninvasive nature and its ability to detect a mural thrombus and a pericardial effusion. Disadvantages include the use of intravascular contrast and its inability to detect aortic insufficiency and branch-vessel involvement.

Magnetic Resonance Imaging (MRI)

MRI is a newer tool for diagnosing AD. Direct diagnostic criteria include the presence of a double lumen with a visible intimal flap best seen when there is blood flow

in both the true and false lumens.[2] The appeal of MRI lies in its ability to provide better views of the dissection anatomy, including the extent of aorta affected and arch-vessel involvement, thereby identifying those patients who need immediate surgery. MRI can also be used to delineate dissection from other pre-existing aortic diseases without the need for contrast material. MRI has been demonstrated to have both a sensitivity and a specificity of 98%.[2,4] Disadvantages of MRI include its unavailability in emergent situations at many institutions and its inability to detect coronary arterial involvement.

Echocardiography

Both transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) have been evaluated for their efficacy in diagnosing aortic dissection. Many diagnostic criteria have been proposed with the most commonly utilized including visualization of an undulating intimal flap that separates the true and false lumens. TTE has demonstrated a sensitivity of 59% and a specificity of 83% with higher sensitivities for dissections that involve the ascending aorta.[4] The quality of images seen on TTE is affected by many factors, including body habitus (ie, quality is impaired with obesity and emphysema). The use of TEE overcomes many of these disadvantages because there is no interference from the chest wall or lung. Visualization of the aortic anatomy may also be enhanced because multiple sections of the aorta may be seen as the probe is advanced and subsequently withdrawn from the esophagus. Other advantages include portability of the study; the short time to completion; and its ability to diagnose aortic insufficiency, coronary artery involvement, and the presence of a pericardial effusion.[2] Sensitivities have been shown to be as high as 98% with specificities of 77 to 97%.[2,4] Disadvantages of TEE include its contraindication for use in patients with known esophageal disease and its dependence on the expertise of the operator and evaluator.

TREATMENT

The goal of acute management of patients with AD involves lowering systolic blood pressure regardless of the type of dissection suspected. Initial treatment includes the use of intravenous beta-blockers such as labetolol or propranolol to reduce blood pressure, heart rate, and inotropic state thereby reducing stress on the aortic wall and propagation of the dissection. When maximum beta blockage has occurred and the systolic blood pressure is still over 100 mmHg, intravenous sodium nitroprusside should be added in low doses and titrated upwards until systolic blood pressure is below 100 mmHg.[7]

Definitive therapy in patients with aortic dissection depends on the type of dissection. Type B dissections are usually treated medically with operation reserved for those who have complications including occlusion of a major aortic branch, uncontrolled hypertension, further propagation of the dissection, and / or evidence of aortic rupture. Acute type A dissections require immediate surgical intervention because these patients are at greatest risk for life-threatening complications. Surgical therapy involves excision of the intimal tear, obliteration of entry into the false lumen proximally, and reconstitution of the aorta with the use of a synthetic vascular graft if necessary.[7] In addition to the repairs of the dissection, aortic valve insufficiency can also be addressed with repair or replacement of the valve.

Long term therapy of patients who have either type A or type B dissections includes use of a beta-blocker, afterload reduction with an ACE inhibitor or hydralazine, and avoidance of strenuous physical activity with the goal of minimizing stress on the aorta.[7] Baseline MRI should be done prior to discharge from the hospital with follow-up scans at 6 months and 1 year. Surveillance scans should then be done at 1-to-2- year intervals with modification of treatment depending on any further complications.

REFERENCES

1. Dzau VJ, Creager MA. Aortic dissection. Sauci AS, Braunwald E, Isselbacker KJ (editors) In: Harrison T (editor). *Principles of Internal Medicine*, 14th edition. New York:McGraw-Hill;1998:1395-1397.
2. Cigarroa JE, Isselbacher EM, DeSanctis RW, et al. Diagnostic imaging in the evaluation of suspected aortic dissection - old and new directions. *NEJM* 1993;328:35-43.
3. Sullivan PR, Wolfson AB, Leckey RD, et al. Diagnosis of acute thoracic aortic dissection in the emergency department. *Am J Emerg Med* 2000;18:46-50.
4. Willens HJ, Kessler KM. Transesophageal echocardiography in the diagnosis of diseases of the thoracic aorta. *Chest* 1999;116:1772-1779.
5. Miles WM, Williams ES, Zipes DP. Aortic and periferal vascular disease. In: Andreoli, TE, Bennett JC, Carpenter CCJ, et al (editors).*Cecil's Essentials of Internal Medicine*. Philadelphia: WB Saunders and Co; 1997:102-107.
6. Schoen FJ. Aortic dissection. In: Cotran RS, Kumar V, Robbins L, et al (editors). *Robbins Pathologic Basis of Disease*, 5th edition. Philadelphia: WB Saunders and Co; 1994: 501-504.
7. Manning, WJ. Management of aortic dissection. In: Rose BD (editor). *UpToDate*. Wellesley, MA. UpToDate, 2001.

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CME QUESTIONS

To earn CME credit, read the preceeding CME article and complete the registration, evaluation, and answer form on page 103. Mail or fax the registration, evaluation, and answer form to the Educational and Research Foundation. Answers must be postmarked or faxed prior to April 30, 2003. Participants must attain a minimum score of 70% to receive credit.

For each question, choose the one answer that is most correct.

1. Physical findings often seen in patients with aortic dissection include:
   a) hypertension
   b) aortic regurgitation
   c) pulmonary edema
   d) all of the above

2. True or False: Pain both above and below the diaphragm accompanied by aortic regurgitation is more suggestive of aortic dissection than myocardial infarction.

3. Cystic medial necrosis of the aorta is associated with which of following diseases?
   a) Rheumatoid arthritis
   b) HIV
   c) The Marfan syndrome
   d) Kleinfelter's disease

4. Which diagnostic test is least helpful in diagnosing aortic dissection?
   a) Magnetic resonance imaging
   b) Aortogram
   c) Computed tomography
   d) Chest radiograph
   e) Electrocardiogram
   f) Transesophageal echocardiogram

5. True or False: Esophageal disease is a contraindication for the use of TEE.

6. True or False: Stanford type B dissections are usually initially treated with operation.

7. Which of the following drugs have a role in the management of aortic dissection?
   a) Beta blockers
   b) Sodium nitroprusside
   c) Both A and B
   d) None of the above

8. True or False: Acute Stanford type A dissections require immediate surgical intervention.