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Assessment of Aortic Dissection and Hematoma

Frank A. Flachskampf, MD

Aortic dissection and aortic intramural hematoma are highly lethal diseases occurring mostly in patients with arterial hypertension or Marfan syndrome. Transesophageal echocardiography (TEE) is the diagnostic imaging procedure of choice in the unstable patient. Hallmarks of dissection are the presence of an intimal membrane, or flap, dividing a true and a false lumen, entry and reentry tears, periaortic hematoma, pericardial effusion or tamponade, and aortic regurgitation in dissection of the ascending aorta. TEE allows detection of involvement of the ascending aorta, which places the patient in an extremely high-risk group and necessitates urgent operation. The accuracy of TEE is similar to computed tomography and magnetic resonance imaging, with the added benefit of being feasible at the bedside or in the operating room. Precautions have to be taken during TEE to avoid an abrupt rise in blood pressure. Aortic intramural hematoma is often a precursor of classic dissection or accompanies it and can also be diagnosed by TEE, although the diagnosis is frequently difficult.

Aortic dissection is one of the deadliest cardiovascular catastrophes. Mortality of untreated dissection involving the ascending aorta has been estimated at 1% to 2% *per hour* during the first 2 days.^{1,2} Rapid diagnosis is, therefore, imperative. Transesophageal echocardiography (TEE) offers expeditious diagnosis of acute aortic pathology.

Aortic dissection and aortic intramural hematoma, its precursor, are probably caused by a rupture of the vasa vasorum in most cases. It is unclear whether spontaneous bleeding into the aortic wall can also cause dissection. The most frequent predisposing conditions for aortic dissection are hypertension (in approximately 80%) and Marfan syndrome, especially in young patients. The site of dissection can be any part of the aorta, although the ascending aorta (60% of

cases) and the thoracic descending aorta immediately distal to the take-off of the left subclavian artery (30% of cases) are the most common sites. A classification of aortic dissection by location and type is presented in Tables 1 and 2.

Ascending aortic dissection carries an extraordinarily high risk of early fatal complications, such as frank aortic rupture with fatal hemorrhage into the mediastinum, bleeding into the pericardial sac with tamponade, torrential aortic regurgitation, carotid ischemia with neurologic insult, and myocardial infarction. Dissection of the ascending aorta is thus an indication for emergent surgery, and its earliest possible recognition is of utmost importance. In contrast, descending aortic dissection, although far from benign, usually has a less deadly course. Dissection usually progresses in the direction of flow; however, retrograde dissection extending from dissections of the descending aorta into the aortic arch has been described and needs to be treated surgically if ascending aortic involvement is demonstrated.

Clinical signs that raise suspicion of aortic dissection are chest pain, especially radiating to the back, a diastolic murmur caused by aortic regurgitation, neurologic impairment, limb or organ ischemia, a pulse differential, and often shock or syncope. Myocardial ischemia is frequently the initial clinical suspicion and sometimes coexists owing to obstruction of a coronary lumen, mostly of the right coronary artery.

Echocardiographic Strategy and Technique

The clinical suspicion of a dissection should prompt a TEE examination, because usually only part of the ascending aorta is seen from the classic transthoracic windows, and suprasternal imaging often is too limited for exclusion of the disease.^{2,4,5} The findings of aortic regurgitation or pericardial effusion, or both, in the clinical context of severe chest pain should always raise a strong suspicion of aortic dissection. TEE, on the other hand, can image the entire thoracic aorta except for the distal ascending aorta, which is obscured by tracheal or bronchial interposition.

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Table 1. Classification of Aortic Dissection^a

	<i>Ascending Aorta</i>	<i>Aortic Arch</i>	<i>Descending Aorta</i>
DeBakey I	Yes	Yes	Yes
DeBakey II	Yes	No	No
DeBakey III	No	No	Yes
Stanford A	Yes	Possible	Possible
Stanford B	No	No	Yes

^aAdapted from reference 3.**Table 2.** European Society of Cardiology Classification^a

- Class I: classic dissection of all Stanford or DeBakey types
- Class II: intramural hematoma
- Class III: subtle circumscribed dissection representing a localized tear without clear-cut hematoma
- Class IV: plaque ulceration (mostly in the descending aorta, and often in the abdominal aorta)
- Class V: traumatic or iatrogenic (mostly catheter induced, retrograde) dissection. Importantly, dissections of classes II-IV may progress to classic (class I) dissection and are difficult or even sometimes impossible to visualize by transesophageal echocardiography (TEE).

^aAdapted from reference 1.

Dissection of the descending aorta distal to the diaphragm is rare, so that a negative TEE study largely rules out aortic dissection. If, however, the proximal ascending aorta is involved, the dissection membrane very often can be seen clearly on transthoracic echo, and the diagnosis can be made right away. TEE is the fastest, safest, and most widely available definitive means of securing all information necessary to decide whether the patient needs immediate surgery or not. Not all TEE exams yield conclusive and unambiguous results, however.^{6,7} Computed tomography or magnetic resonance imaging should be added, if needed. The carotid arteries, the abdominal aorta, and the renal arteries frequently can be well visualized by sonographic imaging.

The examiner who performs TEE in a patient in whom aortic dissection is suspected should ensure adequate blood pressure control, especially during the uncomfortable introduction of the instrument; acute deaths during TEE have been described. Recent oral intake (<4 hours) is only a

relative contraindication for an emergent study, but the problem of vomiting or aspiration should be anticipated and intubation for airway protection can be considered.

Echocardiographic Signs of Acute Aortic Dissection

The Intimal Flap Separating the True and False Lumen

The hallmark of aortic dissection on TEE is an intraluminal mobile, thin (few millimeters) membrane—the intimal flap—separating a true from a false lumen (Figures 1, 2 and 3). The membrane is almost always well delineated and immediately recognizable. The dissection membrane usually shows a pulsatile mobility with systolic convexity towards the false lumen. The following features characterize the false lumen as opposed to the true lumen:

- The false lumen mostly is larger than the true lumen.
- Flow in the false lumen is slower than in the true lumen, readily detectable by color Doppler. The false lumen often displays spontaneous echo contrast or thrombosis due to stagnant flow. Rupture or entry sites show pulsatile systolic flow into the false lumen and slower reverse flow during diastole. In rare cases, flow direction in the false lumen may be opposite of that within the true lumen.
- Because of the higher pressure in the true lumen, it pulsates in systole, displacing the intimal flap towards the false lumen.

Figure 1. Dissection of the ascending aorta is shown in transesophageal long-axis views of the ascending aorta and aortic valve. A flap (arrows) in the ascending aorta moves during the heart cycle. **(A)** Diastole. The flap impinges on the aortic valve, causing aortic regurgitation. **(B)** Systole. The flap is pushed away from the aortic valve by ejected blood. LA = left atrium; Ao = aorta.

Figure 2. Dissection of the ascending aorta is shown in a transesophageal short-axis view of the ascending aorta. The dissection flap is clearly visible (arrow).

lumen in systole and in the opposite direction during diastole. Multiple entry sites may exist. Often one or more re-entry sites further distally can be seen.

Detection of Complications of Aortic Dissection

Rupture

Fluid surrounding the aorta, as well as in the pleural and pericardial spaces, in the context of aortic dissection most likely represents blood and, thus, a rupture of the aorta. Prompt surgical intervention is indicated.

Entry and Re-entry Sites

The rupture or entry site can be visualized on color Doppler as fast flow from the true to false

Pericardial Tamponade

Fluid (blood) accumulation in the pericardial space is common with dissection of the ascend-

Figure 3. Dissection of the descending aorta. **(A)** A transesophageal short-axis view shows the true lumen (TL) is on top. The false lumen (FL) with dense spontaneous echo contrast (“smoke”) is at the bottom. **(B)** A long-axis view of the same descending aorta.

ing aorta, even if the membrane begins to be visible only at some distance from the aortic valve and pericardial space. This is an ominous sign of impending fatal hemorrhage into the pericardium. Signs of tamponade, such as diastolic collapse of the right atrium and ventricle, and exaggerated respiratory variation of transmitral and transtricuspid flow may be present.

Aortic Regurgitation

Dilatation of the aortic root secondary to dissection leads to central loss of coaptation of the aortic cusps, resulting in a central regurgitation jet. Additionally, in the presence of Marfan syndrome, aortic root dilatation, typically with effacement of the ridge of the sinotubular junction and with central regurgitation, often precedes aortic dissection. Another mechanism of aortic regurgitation is diastolic prolapse of the intimal flap through the orifice of the aortic valve interfering with leaflet coaptation. In these cases, the aortic valve usually does not need to be replaced. The least common mechanism for aortic regurgitation is leaflet prolapse caused by pressure of the false lumen impinging on the annulus.⁸ An assessment of the severity and the mechanism of aortic regurgitation and measurement of the aortic annulus diameter may help the surgeon to decide

whether to reconstruct or replace the aortic valve. Because onset of severe regurgitation is usually acute, the ventricle typically is not dilated.

Coronary Involvement

Frequently, in dissection of the ascending aorta the membrane arises at or involves the right coronary ostium. Rarely, the dissection may extend into the right coronary artery and even less frequently, the left. Hence, ischemia in the right coronary distribution is relatively frequent and may be visualized as inferior or right ventricular wall motion abnormality. The coronary ostia may also occasionally be obstructed by prolapse of the intimal flap during diastole or by distention of the false lumen, compromising flow.

TEE Examination of the Aorta

Assessment of the ascending aorta and arch by TEE is the most difficult part of the exam. It may not be possible to visualize some segments of the arch and the distal ascending aorta. This occurs because of trachea and bronchus interposition and because frequent artifacts, in particular reverberations from the left atrial or pulmonary

artery walls or from venous catheters in the pulmonary artery, may mimic dissection flaps.^{6,7}

The most important views of the ascending aorta and the aortic root are the long-axis view (at 120–150 degrees) and the high transesophageal short-axis view (at 30–60 degrees) (Figures 1 and 2). If a presumptive flap is detected, transitioning between these views increases confidence in the diagnosis and may visualize subtle morphologic details. These views also allow delineation of aortic valve morphology, coronary ostia, and mechanism and severity of aortic regurgitation.⁹ In the presence of a proximal (Stanford A) dissection, an entry site close to the aortic root usually can be seen. Often, the aortic root is massively enlarged, predominantly because of the larger false lumen. The flap frequently starts immediately superior to the right coronary ostium. The presence of a pericardial effusion, representing rupture and incipient hemorrhage into the pericardium, is an ominous sign.

The arch can be assessed by withdrawal of the probe from the position used to examine the descending aorta. The inner curvature and the anterior aortic arch wall are usually well seen all the way to the ascending aorta, and the flaps mostly are localized in proximity to this wall. The three supra-aortic great arteries—the innominate, the left carotid, and the left subclavian artery—as a rule are not clearly visualized, except for the take-off of the left subclavian artery.

Finally, the descending aorta is easily visualized in short-axis (0 degrees) and long-axis views (90 degrees) from the diaphragm to its origin at the take-off of the left subclavian artery (Figures 3 and 4).

Intramural Hematoma

Intramural hematoma is now recognized as a variant of aortic dissection, representing an incipient or contained dissection of the aortic wall, and occurring in 10% to 15% of patients with suspected aortic dissection.^{1,10-12} The prognosis seems not to differ substantially from a communicating aortic dissection, and it often coexists with or progresses to overt dissection. Involvement of the ascending aorta should thus prompt urgent surgical intervention. Risk factors are hypertension and atherosclerotic disease.

In some cases, at different levels of the aorta, intramural hematoma may coexist with dissec-

Figure 4. Intramural hematoma of the descending aorta is shown in a transesophageal short-axis view. Note massive thickening of the aortic wall (arrow).

tion. The clinical presentation, except for pulse differences or ischemic complications, is not distinguishable from dissection. Pericardial effusion may be present. Intramural hematoma is more frequent (or more frequently detected) in the descending than in the ascending aorta. Criteria for the diagnosis are a wall thickness of more than 7 mm or an echolucent zone in the aortic wall (Figure 4), or both. An echolucent zone has been reported in 70% to 80% of cases. There is, however, considerable overlap between atherosclerotic wall thickening and the 7 mm criterion for diffuse intramural hematoma.

Not all cases of intramural hematoma show a clear echo-free intramural space. The appearance and continuity of the intima may help distinguish between an atherosclerotic plaque and an intramural hematoma. In the former, the intima can be seen underlying the plaque and remains in apposition to the other layers of the aortic wall. The surface of the plaque is more often irregular and may have adherent thrombus. In contrast, the intimal layer covers the surface of an intramural hematoma, which separates the intima from the media. Even with careful attention to these features, the diagnosis may be difficult or

impossible by TEE alone.¹³ Magnetic resonance imaging (preferably) or computed tomography should be considered in cases of suspicious aortic wall thickening.

Conclusion

Although transesophageal echocardiography facilitates prompt diagnosis of aortic pathology such as dissection and its associated complications, it may not fully distinguish between atherosclerotic plaque and intramural hematoma. If a suspicious aortic wall thickening is present that cannot be clearly explained by TEE, magnetic resonance imaging should be used.

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