Thoracic aortic dissection is the most frequent cause of aortic emergency, and unless it is rapidly diagnosed and treated, the result is death. Helical computed tomography (CT) permits the diagnosis of acute aortic dissection with a sensitivity and specificity of nearly 100%. This imaging modality also enables differentiation between proximal aortic dissection (type A in the Stanford classification) and distal aortic dissection (Stanford type B), which are treated differently and have different prognoses. In 70% of patients in whom nontraumatic acute thoracic aortic dissection is diagnosed after evaluation with helical CT, scans show the typical signs of aortic dissection, with rupture and displacement of the intima. CT also can depict other pathologic entities with similar clinical manifestations, such as intramural hematoma and penetrating atherosclerotic ulcer. Awareness of the different radiologic appearances of these disease entities is essential for differential diagnosis. More than one-third of patients with aortic dissection show signs and symptoms indicative of systemic involvement. Because branch-vessel involvement may increase morbidity and mortality, in this group of patients it is important to evaluate the entire aorta so as to determine the distal extent of the dissection and detect any systemic involvement.
Introduction
Acute thoracic aortic dissection is a life-threatening condition that requires immediate diagnosis and treatment. Acute aortic dissection is the most common cause of aortic emergency. Its prevalence exceeds that of thoracoabdominal aortic aneurysm rupture (1).

Computed tomography (CT), because of its speed and its wide availability, is currently the most common diagnostic imaging method for the study of acute aortic dissection. Helical CT enables the diagnosis of acute aortic dissection with a sensitivity and specificity of nearly 100% (2,3). CT scans in 70% of cases of acute aortic dissection depict an intimal flap. CT also is useful for identifying atypical forms of dissection, such as intramural hematoma (IMH) and penetrating atherosclerotic ulcer. These three entities are clinically indistinguishable; all may produce chest or back pain in a patient with hypertension. CT also enables the evaluation of other thoracoabdominal structures. A thorough assessment is especially important to detect possible complications of aortic dissection, such as ischemic and obstructive diseases, which can increase morbidity and mortality.

Thoracic aortic dissection may be described as acute or chronic, depending on its clinical manifestation. Dissection is considered acute if the symptoms last less than 2 weeks and chronic if they last longer (4). Seventy-five percent of deaths from the condition occur within 2 weeks after the initial manifestation of symptoms (5). In addition, dissection is classified according to the extent of involvement of the thoracic aorta. The original system for classification of aortic dissection, the DeBakey system, has been superseded by the Stanford system, which includes two types based on whether surgery is required (6). Dissection affecting the ascending aorta or the aortic arch (Fig 1a, 1b) is classified as Stanford type A (DeBakey types I and II) (6,7) and accounts for 75% of cases of aortic dissection. Acute type A dissection should be repaired immediately to avoid fatal complications, such as extension to the pericardium, the pleural space, the coronary arteries, or the aortic valvular ring. Chronic proximal dissection, also classified as Stanford type A, is usually associated with medial layer anomalies (eg, cystic medial necrosis), and it too must be surgically treated. Acute dissection that begins distal to the left subclavian artery is classified as Stanford type B (DeBakey type III). The patient affected by Stanford type B dissection is treated medically for hypertension unless complications occur (eg, abdominal organ ischemia or persistent pain) that indicate a progression of the dissection and, eventually, the need for surgery.

The classic signs of typical aortic dissection depicted at CT are rupture and displacement of the intima. However, atypical dissections caused by IMH and penetrating atherosclerotic ulcer may manifest similar signs. Recognition of the complications that often accompany aortic dissection is important for achieving accurate diagnosis and effective treatment. In this article, we show imaging findings in typical and atypical aortic dissections and describe their differentiation.
In describing the appearance of aortic dissec-
tion at CT, we distinguish between features ob-
served on CT scans obtained with contrast mate-
rial enhancement and those observed on scans
obtained without contrast material enhancement.
When acute aortic dissection is suspected, unen-
hanced CT scans should always be obtained to
detect IMH. It is important to evaluate the entire
aorta so as to determine the distal extent of the
dissection and to detect ischemic disease.

**Imaging Technique**

At our institution, helical CT examinations are
performed with a single-detector scanner with a
0.75-second rotation time. The examination be-
gins with the acquisition of an unenhanced CT
scan. Images are obtained with a table feed of 10
mm, collimation of 7 mm, and reconstruction
increment of 7 mm. Coverage begins 3 cm above
the aortic arch and continues to the upper side of
the femoral head. Unenhanced CT scans are use-
ful for diagnosing IMH and acute hemorrhage
(pleural, pericardial, or mediastinal).

After unenhanced CT, contrast-enhanced CT
is performed with a bolus injection of 120 mL of
nonionic contrast material at a rate of 3 mL/sec
through a 21-gauge catheter. The catheter should
be positioned in the right arm, if possible, to
avoid opacification of the left brachiocephalic
vein, which could result in a perivenous artifact
that substantially degrades visualization of the
origin of the brachiocephalic artery. We use a
scanning delay of 25 seconds. In general, optimal
imaging of the thoracic aorta and abdominal aorta
is obtained with scanning delays of 20–30 and
30–40 seconds, respectively. Enhanced CT is
performed with the following parameters: 190
mA, 120 kV, pitch of 1.5, 5-mm collimation, and
3- or 5-mm reconstruction increment. Coverage
begins 3 cm above the aortic arch and continues
to the bifurcation of the iliac artery.

Helical CT is the most common initial diag-
nostic test performed when acute aortic dissection
is suspected. Because of the wide availability of
helical CT and because it enables rapid diagnosis
in emergent situations, in many institutions heli-
cal CT is the method of choice for evaluating the
aorta in acute situations. Most magnetic reso-
nance (MR) imagers have limited capability for
depiction of acute aortic disease and are used
mostly for evaluation of patients with stable or
chronic aortic conditions. One advantage of MR
imaging over CT is the ability of the former to
depict complications such as left ventricular dys-
function and valvular regurgitation (7,8). The
recently developed 8- and 16-element multidetec-
tor CT scanners enable coverage of the entire
aorta, from the valve to the femoral heads, in
20–30 seconds of scanning time and with 1-mm
section thickness. The new imagers undoubtedly
will further increase the accuracy of CT for detec-
tion of aortic disease.

**Typical Aortic Dissection**

Typical aortic dissection is produced by an inti-
mal tear that allows blood to enter the medial
layer, giving rise to two lumina, one true and one
false (Fig 2).
Figure 3. Rupture of a Stanford type A typical aortic dissection in an 18-year-old patient with aortic coarctation. (a) Unenhanced CT scan shows an aneurysm and displaced intima in the ascending aorta. The intima (arrow) is hyperattenuated due to severe anemia. (b) Contrast-enhanced CT scan shows the intimal flap (arrowhead) in the ascending aorta, coarctation (*) in the descending aorta, and substantial collateral circulation through bronchial and intercostal arteries (arrows). Mediastinal hemorrhage and bilateral pleural effusions are also evident.

Figure 4. Stanford type B typical aortic dissection. (a) Unenhanced CT scan depicts displaced intimal calcifications (arrow) in the descending aorta. (b) Contrast-enhanced CT scan shows an intimal flap (arrow) in the descending aorta.
Although hypertension is the most frequent factor predisposing patients to aortic dissection, other conditions also are associated with dissection, including Marfan syndrome and Turner syndrome, other connective tissue diseases, congenital aortic valvular defects, aortic coarctation (Fig 3), aortic aneurysm, infection and other causes of aortitis, and pregnancy (9,10). Cocaine use also has been associated with aortic dissection in otherwise healthy, normotensive patients (11).

**Findings at Unenhanced CT**

Occasionally, on unenhanced CT scans in aortic dissection, one may see internal displacement of intimal calcifications (Fig 4a). This finding can be problematic because it may be confused with an aneurysm with calcified mural thrombus (Fig 5). High attenuation of the false lumen at unenhanced CT may help the imaging specialist differentiate between the two conditions (12).

**Findings at Contrast-enhanced CT**

The main finding on contrast-enhanced CT scans of aortic dissection is an intimal flap that separates the true lumen from the false lumen (Fig 3b, Fig 4b, Fig 6). Accurate differentiation at CT between the true and the false lumen was relatively unimportant previously because surgery was the main therapy used; however, this distinction recently has become particularly important for planning endovascular treatment of dissection (13). The slender linear areas of low attenuation that occasionally appear in the false lumen on CT images, known as the cobweb sign, are specific to the false lumen and may aid in its recognition.
Figure 7. Stanford type B typical aortic dissection. Sequential contrast-enhanced CT scans show a cobweb sign (arrow)—linear traces of low attenuation—in the false lumen. The beak sign (arrowhead) is caused by a wedge-shaped protrusion of the hematoma in the false lumen. A motion artifact can be seen in the ascending aorta. The true lumen (+ in b) is compressed and adopts a crescent shape.

Figure 8. Stanford type A typical aortic dissection with intimointimal intussusception. Contrast-enhanced CT scans show the circumference of the intimal intussusception (arrows) in the aortic arch (a) and the intimal flap (arrows) in the aortic root (b). Note the intimal flap in the descending aorta in b.
These findings correspond to residual ribbons of the media, incompletely sheared away during the dissection process (14). Two other useful indicators of the false lumen are a larger cross-sectional area and the beak sign. The latter is the cross-sectional imaging manifestation of the wedge of hematoma that cleaves a space for the propagation of the false lumen (Fig 7) (15). On most contrast-enhanced CT scans, however, the true lumen may be identified by its continuity with an undissected portion of the aorta.

Intimointimal intussusception is an unusual type of aortic dissection produced by circumferential dissection of the intimal layer, which subsequently invaginates like a windsock. The intimal tear usually begins near the coronary orifices (Fig 8) (16,17). In intimointimal intussusception, CT scans may show one lumen wrapped around the other lumen in the aortic arch, with the inner lumen invariably being the true lumen.

An aortic aneurysm with intraluminal thrombus may be difficult to distinguish from a dissection with a thrombosed false lumen. The fact that dissection generally has a spiroidal shape, whereas a thrombus tends to maintain a constant circumferential relationship with the aortic wall, can help in visual differentiation between the two. Furthermore, a mural thrombus usually has an irregular internal border, whereas a dissection usually has a smooth internal border. Intimal calcification occurring in an aortic aneurysm is typically located at the periphery of the aorta (Fig 9) (18).

Intramural Hematoma

In contrast to typical aortic dissection, in which there is an intimal tear, IMH is caused by a spontaneous hemorrhage of the vasa vasorum of the medial layer, which weakens the media without an intimal tear (Fig 10) (19).
The clinical manifestations and the risk factors in IMH are similar to those in typical aortic dissection. IMH accounts for approximately 13% of the prevalence of acute aortic dissection (20).

IMH is commonly classified according to the Stanford system as typical aortic dissection. Many recommend that IMH of Stanford type A be treated surgically (21,22). Some authors have suggested that, in light of the high mortality and morbidity associated with aortic surgery, supportive medical treatment with frequent follow-up imaging may be a rational management option (23,24). Song et al (23) hypothesized that the absence of intimal tear and of continuous flow communication in IMH probably indicate a better clinical outcome than that in typical aortic dissection.

Findings at Unenhanced CT
On unenhanced CT scans, IMH appears as a crescent-shaped area of attenuation in the aortic wall, corresponding to a hematoma in the medial layer. The hematoma may or may not compress the aortic lumen (25). Intimal calcifications also may be displaced by IMH (Fig 11a). It is important to perform unenhanced CT as the first imaging evaluation when aortic dissection is suspected, because contrast material within the vessel may obscure IMH.

Findings at Contrast-enhanced CT
Unlike the false lumen in typical aortic dissection, the crescent-shaped area of IMH remains unenhanced after contrast material administration, and no intimal tear is seen on contrast-enhanced CT scans (Fig 11b). An additional observation that may help differentiate IMH from the thrombosed false lumen in typical aortic dissection is that the latter tends to spiral longitudinally around the aorta, whereas the former tends to maintain a constant circumferential relationship with the aortic wall (18).

Although some have hypothesized that aortic IMH is a precursor to aortic dissection (20,26), the precise relationship between IMH and aortic dissection remains unclear. Several investigators have attempted to assess the usefulness of CT findings for predicting the progression of aortic IMH to aortic dissection. The maximum aortic diameter (≥50 mm), estimated on the basis of the initial CT scan, is predictive of progression in type A IMH (27,28). Findings such as type A IMH, thick hematoma with compression of the true lumen, pericardial effusion, or, less important, pleural effusion were useful for predicting progression to aortic dissection (29). A thicker hematoma may indicate more-active bleeding from the ruptured vasa vasorum, which may result in increased weakening of the intima of the affected aorta (29) (Figs 12, 13).
Figure 11. Type A IMH. (a) Unenhanced CT scan depicts crescent-shaped areas with high attenuation (arrows) extending along the walls of the ascending and descending aorta. The displaced intimal calcifications in the descending aorta indicate a subintimal location. A pericardial effusion (arrowheads) also is visible. (b) Contrast-enhanced CT scan shows no enhancement of attenuation in the crescent-shaped areas (arrows). IMH is less apparent here than on the unenhanced CT scan in a.

Figure 12. Unenhanced CT scan shows Stanford type B IMH (arrow) compressing the lumen of the descending aorta, as well as pleural effusion. These findings increase the likelihood of the hematoma progressing to dissection. The faint lines in the ascending aorta are artifacts.
Penetrating atherosclerotic ulcer is defined as an ulceration of atheromatous plaque that has eroded the inner, elastic layer of the aortic wall, reached the medial layer, and produced a hematoma in the media (30). Involvement of the media can sometimes be complicated by aneurysmal dilatation or, more rarely, rupture (Fig 14). Some authors have theorized that most saccular aneurysms are caused by a penetrating atherosclerotic ulcer (Fig 15) (31–33).

**Figure 13.** Evolution of IMH to typical dissection. (a) Unenhanced CT scan shows a crescent-shaped area of high attenuation in the descending aorta, indicating intimal displacement (arrow). (b) Contrast-enhanced CT scan acquired at the same time as a shows no enhancement of the crescent-shaped area (arrow). (c) Contrast-enhanced CT scan acquired 1 week later because the patient reported persistent pain shows aortic dilatation and dissection of the lumen (arrow).
Figure 14. (a–d) Diagrams illustrate the four stages in the formation of a penetrating atherosclerotic ulcer: aortic atheroma (a), benign intimal plaque ulceration contained in the intima (b), medial hematoma with potential adventitial false aneurysm (c), and transmural rupture (d). (e) Photograph of an autopsy specimen shows severe atherosclerotic changes in the descending aorta, with ulceration of the media (arrows) and IMH (*). Scale is in centimeters.
Unlike typical aortic dissection, penetrating atherosclerotic ulcers most often occur in elderly patients with severe underlying atherosclerosis. These ulcers typically involve the aortic arch and descending thoracic aorta and occur rarely in the ascending aorta, where rapid blood flow from the left ventricle provides protection against atherosclerosis (34).

As for type B typical aortic dissection, the most widespread treatment for penetrating atherosclerotic ulcers is medical therapy. Surgery is performed in patients who have hemodynamic insta-
bility, persistent pain, aortic rupture, distal embolization, or rapid enlargement of the aortic diameter. It is important to emphasize that surgical repair of a penetrating atherosclerotic ulcer is generally more complex and extensive than surgical repair of type B typical aortic dissection; much of the aortic wall may have been damaged by ulceration and may have to be replaced. Aortic dissection, in contrast, usually can be treated with a graft at the site of the proximal intimal tear (30,33–35). Consequently, aortic grafting for penetrating atherosclerotic ulcer may be associated with higher morbidity (eg, increased risk of paraplegia) because of a greater compromise of the spinal cord blood supply during surgery (33).

**Findings at Unenhanced CT**

In penetrating atherosclerotic ulcer, extensive atherosclerosis and IMH of variable extent are visible on unenhanced CT scans. Frequently the IMH is focal because of medial fibrosis caused by atherosclerosis (34). Displaced intimal calcifications also are often seen (Fig 16a).

**Findings at Contrast-enhanced CT**

On contrast-enhanced CT scans of penetrating atherosclerotic ulcer, a collection of contrast material is seen outside the aortic lumen (Fig 16b). The appearance of the lesion is similar to that of a peptic ulcer. Lesions can be single or multiple. Penetrating atherosclerotic ulcer is often associated with thickening of the aortic wall, which appears enhanced (35).

Atheromatous ulcers that are confined to the intimal layer sometimes have a radiologic appearance similar to that of penetrating atherosclerotic ulcer. Therefore, particular care should be taken in making a diagnosis of penetrating atherosclerotic ulcer if the lesions are discovered incidentally in an asymptomatic patient and if associated focal IMH is absent (Fig 17) (36). It has been recommended that patients in whom the finding is incidental should receive the same CT follow-up as those with thoracic aortic aneurysms, because one-third of ulcerlike lesions may progress, resulting in mild intervallic aortic enlargement (32).

When rupture and mediastinal hemorrhage occur, it is nearly impossible to differentiate between a ruptured aneurysm and a complicated atherosclerotic ulcer. In both cases, immediate surgical treatment is required.
Complications

Death from thoracic aortic dissection is usually caused by acute aortic regurgitation, major branch-vessel obstruction, pericardial tamponade, or aortic rupture. Complications arising from thoracic aortic dissection may occur in the thorax or in an extrathoracic location. More than one-third of patients with aortic dissection show signs and symptoms secondary to other organ system involvement. The most common mechanism is the development of ischemia secondary to obstruction of branch arteries originating from the aorta, such as renal arteries. A branch-vessel obstruction could be caused by an extension of the dissection process into the wall of the vessel or a direct compression of the branch artery by an expanding false lumen. Another mechanism of organ system involvement is rupture of the dissected aorta, causing blood to leak into the surrounding structures. Such events are usually fatal (7). It is therefore important in thoracic aortic dissection to evaluate the entire aorta so as to determine the distal extent of the dissection and to detect possible abdominal ischemic disease that might increase morbidity and mortality (3,8).

Hemopericardium, Mediastinal Hematoma, Hemothorax

The risk of fatal aortic rupture in patients with untreated proximal aortic dissection is approximately 90%. Seventy-five percent of ruptures take place in the pericardium, the left pleural cavity, or the mediastinum. The signs of aortic rupture include hyperattenuating mediastinal, pericardial, or pleural fluid collections on unenhanced CT scans (Figs 18, 19) and irregularity of the aortic wall and extravasation of vascular contrast material on contrast-enhanced CT scans.

Rupture of a type A dissection into the pericardial cavity may result in acute pericardial effusion, with a high risk of cardiac tamponade—the most frequent cause of death in patients with this complication. Although the presence of pericardial effusion is not always secondary to a rupture or leak from the dissected aorta, the presence of any pericardial effusion is an ominous sign (7).

Mediastinal Hematoma Dissecting the Sheath of the Pulmonary Arteries

A rare complication of type A aortic dissection is mediastinal hematoma that dissects the sheath of the pulmonary arteries. Blood flows from the ascending aorta to the interstitial space bordering the pulmonary arteries. Rupture usually occurs in the posterior wall of the ascending aorta, adjacent to the right pulmonary artery. Blood seeping from the ruptured aorta can reach the lung through the bronchovascular sheaths (37; Matsuoka Y, unpublished data, March 1999). We have observed two patients with this rare complication (Figs 20, 21).
Figure 20. Rupture of Stanford type A typical aortic dissection. (a) Contrast-enhanced CT scan shows stenosis of the pulmonary arteries, which are enveloped in a hemorrhagic sheath. (b) Magnified view shows the detail of the stenosis around the left lower lobe pulmonary artery (arrows). (c) Scan at lung window setting shows areas of alveolar opacity in the right upper lobe caused by diffusion of blood through the peribronchovascular hilar sheath. (d) Posterior view of the autopsy specimen shows hemorrhage in the ascending aorta (arrows) and surrounding the pulmonary arteries (arrowheads). (e) Drawing provides a posterior view of the anatomic pathway from the ascending aorta to the pulmonary interstitium.
Neurologic Complications
Cerebral ischemia associated with aortic dissection is caused by supraaortic trunk involvement and is unusual, occurring in only 5%–10% of patients with aortic dissection. If neurologic symptoms are absent, surgical repair is limited to the ascending aorta because aortic arch replacement is associated with a high mortality (Fig 22) (38).

Abdominal Complications
Obstruction in the main abdominal arterial branches (celiac, superior mesenteric, renal, and inferior mesenteric arteries) can be demonstrated with contrast-enhanced CT. The frequency of such obstruction has been reported as 27% (39). There are two principal mechanisms of branch-vessel compromise: static, if the dissection flap intersects or enters the branch-vessel origin (Fig 23); and dynamic, if the intimal flap spares the branch vessel but prolapses and covers the branch-vessel origin like a curtain. The shape of the true lumen viewed in cross section on CT scans may be an indicator of possible branch-vessel ischemia: If the true lumen has a crescent shape or is concave to the false lumen (see Fig 7b), there may be a pressure deficit in the true lumen of the aorta and its branches, with a consequent risk of ischemia. The distinction between the two mechanisms of branch-vessel compromise is important because they require different modes of percutaneous treatment. Static obstruc-
tion is treated locally with an intravascular stent. Dynamic obstruction is treated with a fenestration of the intimal flap to decrease pressure in the false lumen (40,41).

Many aortic dissections of type B extend to the iliac arteries without clinical repercussions (Fig 24) (3). If the results of clinical examination suggest ischemia of the legs, that diagnosis can be confirmed with helical CT.

**Conclusions**

CT should be the first diagnostic test performed when acute aortic dissection is suspected. We believe it is important to perform unenhanced CT first to prevent masking of IMH. Management varies according to classification (Stanford type A or B).

Advances in percutaneous treatment make it important to distinguish between the true lumen and the false lumen; it is essential to know the radiologic findings in typical aortic dissection, IMH, and penetrating atherosclerotic ulcer. Moreover, the entire aorta must be evaluated to determine the extent of the dissection and to identify possible branch-vessel involvement.

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**References**

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