Nontraumatic Acute Aortic Emergencies: Part 1, Acute Aortic Syndrome

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OBJECTIVE. The aim of this article is to illustrate the imaging findings and analyze the spectrum of findings seen in patients with acute aortic syndrome. We also will discuss the overlaps in pathophysiologic and imaging findings among aortic syndromes.

CONCLUSION. Acute aortic syndrome includes acute aortic dissection, intramural hematoma, and penetrating atherosclerotic ulcer. The most common clinical presentation is severely painful and potentially life-threatening abnormalities of the aorta. Differentiating among these aortic diseases is impossible by symptoms or physical evaluation. Therefore, any clinical suspicion should prompt immediate action including confirmatory noninvasive imaging. Prognosis of acute aortic syndromes is clearly related to prompt diagnosis and appropriate management. Accurate imaging interpretation can modify the natural history of acute aortic syndrome and improve prognosis.

Keywords: acute aortic syndrome, aorta, aortic dissection, CT, intramural hematoma, penetrating atherosclerotic ulcer

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In this article, the pathophysiology and imaging findings of acute aortic syndrome are briefly reviewed. Part 2 [1] focuses on the pre- and postsurgical complications related to aortic aneurysm in an emergency clinical setting.

Acute aortic syndrome comprises three interrelated entities—namely, aortic dissection, intramural hematoma (IMH), and penetrating atherosclerotic ulcer (PAU) [2] (Fig. 1). Population-based studies suggest that the incidence of acute aortic dissection ranges from 2.6 to 3.5 cases per 100,000 persons per year; two thirds of the affected patient population is male. Hypertension and a variety of genetic disorders with altered connective tissues are the most prevalent conditions that increase the risk of acute aortic syndrome [3–6] (Appendix 1).

Disruption of the media layer of the aorta is the feature common to the entities that make up acute aortic syndrome. One in eight patients diagnosed with acute aortic dissection have either an IMH or a PAU, and these life-threatening conditions are clinically indistinguishable. Excruciating pain is the most common presenting symptom of acute aortic syndrome. The localized pain and associated symptoms reflect the initial disruption of the intimal layer and may change as the dissection extends along the aorta or involves other arteries or organs. Compared with acute aortic dissection, IMH and PAU have an unpredictable evolution without treatment. Therefore, IMH and PAU require surveillance and a multidisciplinary therapeutic approach.

MDCT, transesophageal echocardiography (TEE), and MRI are valuable tests in the diagnosis of acute aortic syndrome [7]. The role of chest radiography for diagnosis or surveillance in this setting is very limited. Catheter angiography is used as a means for treating complications of the disease by implanting an endovascular prosthesis. The essential goals of imaging are to confirm the diagnosis of an aortic wall lesion; to ascertain the site, extension, and complications of disease; and to plan the most appropriate and timely management approach. Universal availability, rapid acquisition, and high accuracy make MDCT the imaging modality of choice in the emergency setting [4]. Obtaining unenhanced CT images helps to differentiate among the various components of acute aortic syndrome (Table 1).

Aortic Dissection

Aortic dissection results from separation of the aortic wall layers. Elevated blood pressure with the concomitant degenerative changes in the aortic media is the most common trigger for aortic dissection. Other conditions that can result in separation of the aortic intima and adventitia include Marfan syndrome, Turner syndrome, other connective tissue diseases,

TABLE 1: CT Appearance of Acute Aortic Syndrome

Pathologic Entity	CT Appearance	
	Without IV Contrast Material	With IV Contrast Material
Aortic dissection	Inward displacement of intimal calcification	Intimal flap, double lumen
Intramural hematoma	High-attenuation crescentic thickening of aortic wall	Aortic wall thickening
Penetrating atherosclerotic ulcer	Difficult to diagnose on unenhanced CT	Focal ulceration penetrating through aortic intima into aortic wall

congenital aortic valvular defects, aortic coarctation, aortic aneurysm, aortitis (infective or inflammatory), and pregnancy. The right lateral wall of the ascending aorta and the proximal segment of the descending thoracic aorta are the sites of maximum hydraulic stress and are the most common sites of an entrance tear in the intima [8]. The blood with the aortic lumen finds direct access to the layers of the aortic wall through the intimal tear and propagates within the aortic wall (media). Subsequent dissection progression and propagation can be both antegrade and retrograde from the site of the intimal tear and result in the formation of a true lumen and false lumen, with the false lumen having pressures greater than or equal to the true lumen [9].

Classification of aortic dissection is based on the location and extension of the dissection, and the DeBakey [10] and Stanford [11] classification systems are most commonly followed (Fig. 2). In the emergency radiology setting, the Stanford classification is preferred because it dictates immediate clinical management: surgical (type A) versus medical (type B).

The clinical presentation of aortic dissection can be very deceptive and findings on physical examination are nonspecific and may be variable. Patients may present with a classic history of acute-onset central chest pain that radiates to the back. Syncope can result from hypotension secondary to cardiac tamponade or from obstruction of cerebral vessels [12].

The sensitivity and specificity of TEE, MDCT, and MRI in the diagnosis of aortic dissection are comparable [13, 14]. However, MDCT has a sensitivity of 100% and a specificity of 98% and is superior to TEE and MRI in ruling out thoracic aortic dissection especially in the acute setting [14]. The entrance tear, in general, is perpendicular to the long axis of the aorta and is detected mainly by analyzing the images acquired in the axial or transverse plane.

The classic intimal flap (Fig. 3) separating the true lumen and false lumen is seen in approximately 70% of cases of aortic dissection [15]. A Stanford type A dissection (Fig. 3) involves the ascending aorta and can extend into the descending aorta. A Stanford type B dissection (Figs. 4 and 5) involves the descending aorta beyond the origin of the left subclavian artery. Dissection of the entire intima results in a circumferential flap [15] (Fig. 3). Other atypical configurations of the intimal flap are summarized in Appendix 2 [15]. The true lumen is continuous with the nondissected portion of the aorta and may be identified in most cases [16]. Sometimes differentiation of the true lumen from the false lumen is difficult, particularly in cases with involvement of the aortic root and especially in those with circumferential dissection involving the root.

The two most useful indicators of the false lumen are the beak sign, which is a wedge of hematoma that creates space for the propagation of the false lumen, and the larger cross-sectional area of the false lumen compared with the true lumen [12, 17] (Fig. 6). The "cobweb" sign (i.e., ribbons of media that are incompletely sheared off by the dissection) and an intimomedial flap rupture communicating with the false lumen (Fig. 5) are highly specific in the identification of the aortic false lumen [17, 18], but these signs are of limited usefulness because they are infrequently encountered. False lumen thrombus, partial or complete (Fig. 6), is significantly more frequently seen in chronic dissection. Distinction between the true lumen and false lumen is essential for both surgical repair and planning a percutaneous intervention; Table 2 lists the CT features of the true and false lumina.

A type A aortic dissection can be lethal and requires urgent surgical treatment. Complications related to type A aortic dissection include pericardial tamponade, coronary artery dissection or occlusion, and organ malperfusion. A type B aortic dissection is less problematic compared with a type A dissection and can be treated conservatively. However, immediate aortic repair using an endovascular stent or surgical procedure is indicated in patients who develop ischemic complications such as renal failure or visceral ischemia or who show signs of an impending rupture. An acute type B aortic dissection with partial thrombosis of the false lumen requires more intensive follow-up. These dissections have a significantly higher annual growth rate than dissections with patent or complete thrombosis of the false lumen and patients may benefit from prophylactic intervention [19].

Intramural Hematoma

IMH is defined as acute hemorrhage contained within the layers of the aortic wall, thus creating a false lumen. Originally IMH was described as a novel variant of aortic dissection with no visible intimal flap or apparent intimal tear [2, 20]. This concept is outdated, and modern imaging technology can usually detect small communications between the aortic lumen and the hemorrhage within the wall [21]. Hu et al. [22] successfully detected subtle intimal tears in acute IMH using intravascular ultrasound, but their findings are beyond the scope of this article.

IMH may originate spontaneously as a consequence of a penetrating ulcer or after thoracic trauma. Many investigators have reported an overlap between classic aortic dissection and IMH and suggest that the hematoma results from microscopic tears in the aortic intima contrary to the earlier belief that it results from rupture of the vasa vasorum. Aortic wall hemorrhage can lead to aortic wall infarction, which is believed to be the precursor of acute aortic dissection [20]. Current opinion about IMH is that it can be a variant or a precursor of aortic dissection, and many investigators have suggested that IMH is synonymous with a thrombosed type or noncommunicating aortic dissection [23]. Separation of aortic wall layers is filled with thrombus rather than free-flowing blood in what would otherwise be the false lumen of a classic dissection.

Acute IMH accounts for 5–15% of all cases of acute aortic syndrome [24]. The clinical manifestations of IMH are similar to those of other acute aortic syndromes, and patients predominantly present with acute chest pain. Differentiation of IMH from aortic dissection cannot be made on clinical grounds. In

TABLE 2: CT Findings Useful in Distinguishing Between the True Lumen and False Lumen

CT Finding	True Lumen	False Lumen
Communication with aorta	Directly communicates with aorta	Not connected to unaffected aorta
Intimomedial flap rupture	Smaller than false lumen	Communicates with false lumen
Caliber	Displaced inward	Larger than true lumen
Intimal flap	Calcification along intimal flap	Surface of intimal flap is convex
Characteristic features	Enhances more than false lumen	"Cobweb" sign and beak sign
Enhancement		Slow flow, so hypodense compared with true lumen

one study, patients with IMH described more severe initial pain than those with aortic dissection, but patients with IMH were less likely to have ischemic leg pain, pulse deficits, or aortic valve insufficiency [20]. Moreover, IMH required a longer time to diagnose and patients with IMH underwent a greater number of diagnostic tests [20].

A hyperdense crescent on unenhanced CT is the most common finding of IMH (Figs. 7 and 8). The absence of an obvious communication between the true and false lumina explains the absence of flow on color Doppler imaging and the lack of enhancement with contrast administration on CT, MRI, or angiography. Rarely, IMH may originate within an ulceration in an atherosclerotic aortic plaque. One way to differentiate IMH from a thrombosed false lumen of aortic dissection is that IMH maintains a constant circumferential relationship with the aortic wall (Fig. 7), whereas a dissection tends to spiral longitudinally. The term "primary intimal tear" is advocated by some authors to describe an intimal disruption in patients with the dissection-variant IMH rather than using "ulcerlike lesions" [23].

Prognosis of IMH is variable. Spontaneous regression can occur in 10% of patients. Progression to classic aortic dissection (Fig. 9) is seen in 28–47%. The risk of aortic rupture in IMH is 20–45% [24]. Similar to type A and type B aortic dissections, surgery is advocated in patients with type A IMH and initial medical therapy is advocated in patients with type B IMH [25].

Penetrating Atherosclerotic Ulcer

In PAU, an atherosclerotic plaque erodes the internal elastic lamina into the media of the aortic wall [2]. This plaque is a manifestation of a severely diseased intima and occurs in the background of advanced atherosclerosis. PAU typically occurs in an elderly individual with multiple risk factors for atherosclerosis and associated comorbidities of atherosclerotic disease, such as coronary artery disease, peripheral arterial disease, or abdominal aortic aneurysm [26]. In contrast to patients with PAU, patients with aortic dissection are relatively younger and, apart from hypertension, may not have any other risk factors for atherosclerosis [6, 27]. However, in the absence of atherosclerosis, PAU can occur in young patients with a connective tissue disorder or after rupture of the mycotic plaque. The incidence of PAU in patients presenting with acute aortic syndrome is estimated to be between 2.3% and 7.6% [28, 29]. Early identification of aortic penetrating ulcer is critical. The atheromatous plaque can rupture and precipitate intramural hemorrhage. This hemorrhage can cause a limited intramedial dissection or saccular pseudoaneurysm [6]. PAU has often been clustered with IMH as a cause of acute aortic syndrome [24].

The clinical manifestations of PAU may be the same as those of aortic dissection. In the acute setting, contrast-enhanced CT is the modality of choice for diagnosing PAU [4]. An important imaging feature of PAU is the outpouching of the aortic wall with jagged edges, usually in the presence of extensive aortic atheroma and intimal calcification [2, 30] (Fig. 10). Batt et al. [31] and Tsuji et al. [32] stated that an ulcerative lesion not extending beyond the aortic wall on angiograms or CT images should be added to the definition of PAU. Atheromatous ulcers have a similar appearance, but they are confined to the intimal layer. PAUs are more commonly (> 90%) located in the descending thoracic aorta, where atherosclerotic changes are more common [27, 33]. As discussed earlier, ulcerations of PAU are often accompanied by hemorrhage in the aortic medial layer. Unenhanced CT frequently shows high-density hematoma surrounding the ulceration (Fig. 11).

IMH with PAU is significantly associated with a progressive disease course, whereas IMH without PAU typically has a stable course [24]. Additionally, progression of PAU induces saccular aneurysms (Fig. 12), aortic dissection (Fig. 11), or aortic rupture [34]. The incidence of complications in patients with PAU in one study was up to 70% [34]. Tittle et al. [35] reported a 38% incidence of aortic rupture in patients with PAU presenting as an acute aortic syndrome and an even higher incidence when PAU involved the ascending aorta. Regardless of location, PAUs tend to have an unfavorable prognosis, with a higher incidence of aortic rupture than aortic dissection [29, 30]. Aortic dissection secondary to an aortic ulcer is shorter in extension, is farther away from classic entrance tears, and has a thicker calcified and static flap. Differentiation among these complications and PAU is often difficult, and these various pathologic conditions overlap. Invasive treatments such as surgery and stent-grafting are indicated in acute or symptomatic cases, but course observation including periodical evaluation using imaging techniques is recommended in asymptomatic or chronic cases [25, 36].

Conclusion

Acute aortic syndrome comprises interrelated emergent aortic conditions with a similar and overlapping clinical presentation. Immediate diagnostic imaging (TEE, MDCT, MR angiography) plays a pivotal role in the diagnosis and management. Management of aortic dissection depends on the location of the dissection and extent of the disease. IMH and PAU can be unstable or unpredictable and therefore need a specific therapeutic approach [25]. MDCT is a valuable tool for the diagnosis of acute aortic syndrome, especially in the acute setting.

Understanding the pathophysiology of these interrelated aortic diseases and having a familiarity with the spectrum of imaging findings of each of these entities can facilitate prompt diagnosis.

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(Figures and appendixes start on next page)

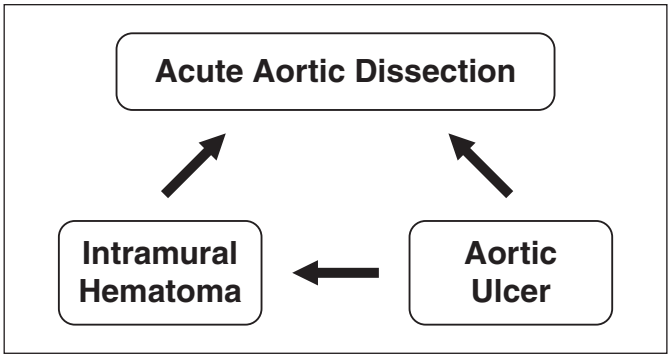

Fig. 1—Acute aortic syndrome. Arrows indicate possible progression of each of these aortic lesions. (Reproduced from *Heart* [2] [Vilacosta I, Román JA. volume 85, pp. 365–368, © 2001] with permission from BMJ Publishing Group Ltd.)

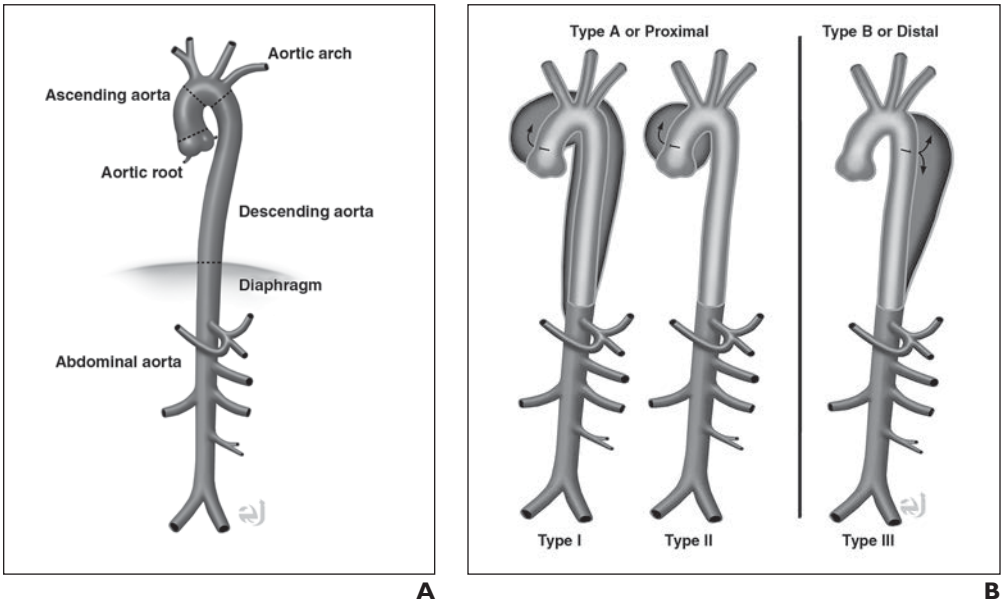

Fig. 2—Diagrammatic representations of anatomy of aorta and classifications used to characterize aortic dissection. (Drawings by Jablonowski E)
A, Diagram shows anatomy of aorta. Dotted lines mark borders of each structure.
B, Diagrams illustrate types of aortic dissections according to DeBakey [10] and Stanford [11] classifications. In emergency radiology setting, Stanford classification is preferred because it dictates immediate clinical management: surgical (type A) versus medical (type B). Arrows show flow of blood from true lumen to false lumen in dissection.

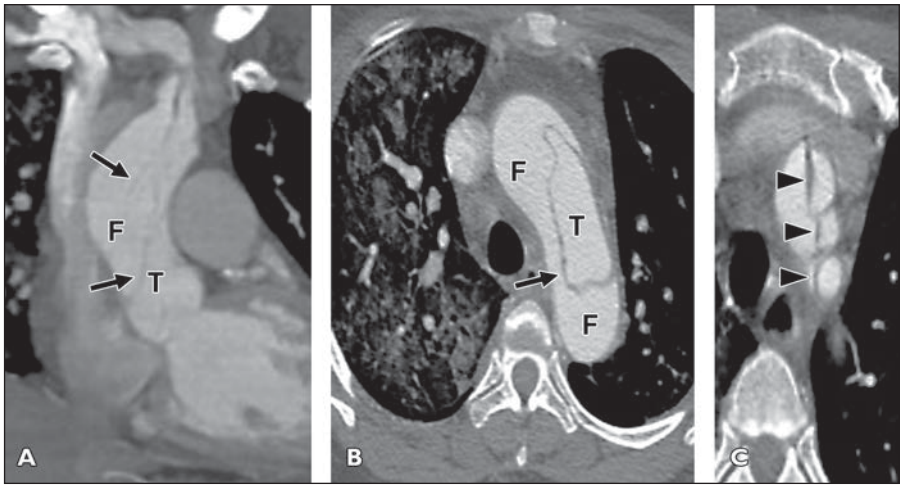

Fig. 3—Aortic dissection, type A according to Stanford classification, in 75-year-old man who presented with acute aortic pain.
A–C, Coronal (**A**) and axial (**B** and **C**) contrast-enhanced CT images show displaced intimal flap (*arrows*, **A** and **B**) extending from aortic root through great vessels (*arrowheads*, **C**). Double-barrel lumen with intimal flap dividing true (T, **A** and **B**) and false (F, **A** and **B**) lumina is classic feature of acute aortic dissection.

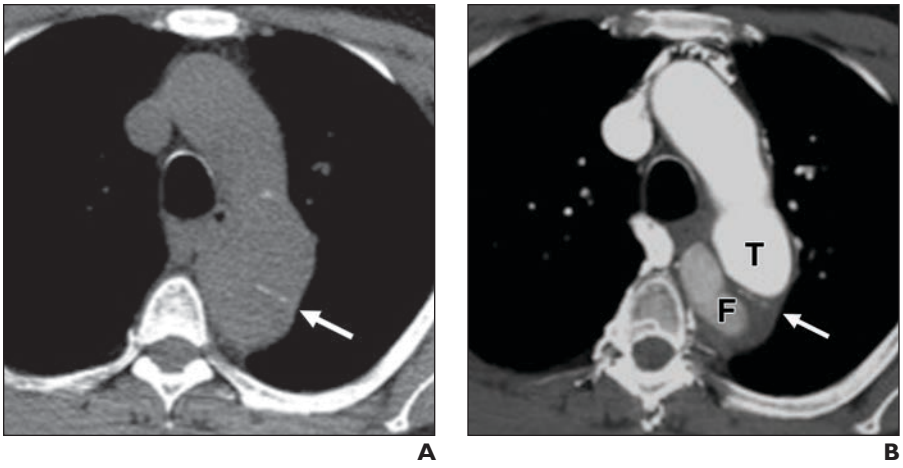

Fig. 4—Aortic dissection, type B according to Stanford classification, in 56-year-old man who presented with acute aortic syndrome.
A, Axial unenhanced CT image shows displaced intimal calcifications (*arrow*), which are suggestive, but not diagnostic, of acute aortic syndrome.
B, Contrast-enhanced CT image shows type B dissection (*arrow*). Note differential enhancement of true (T) and false (F) lumina; true lumen directly continues with aorta and enhances more than false lumen.

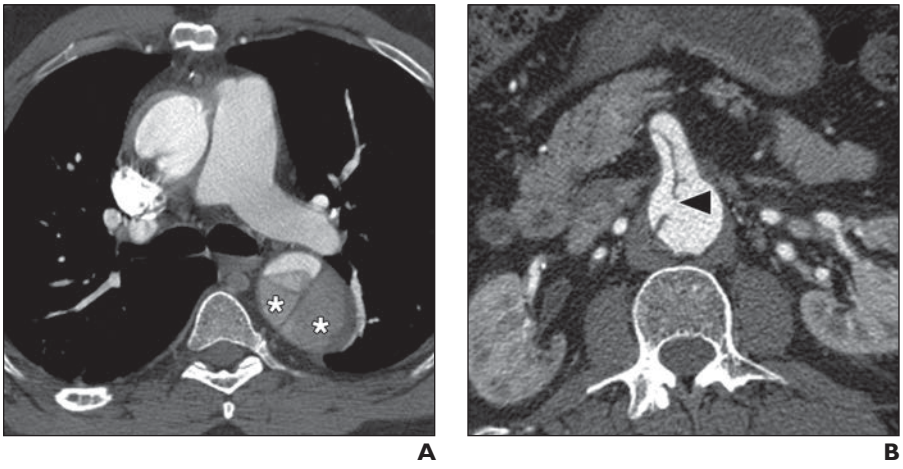

Fig. 5—Aortic dissection, type B according to Stanford classification, in 48-year-old man who presented with chest discomfort.
A, Axial contrast-enhanced CT image shows multiple false lumina (*asterisks*) (Mercedes-Benz sign) in proximal descending thoracic aorta.
B, Axial CT image obtained at level of superior mesenteric artery (SMA) shows "intimomedial rupture" sign (*arrowhead*), indicating direction of tear of intimomedial entrance is from true to false lumen. Note extension of intimal flap into proximal SMA.

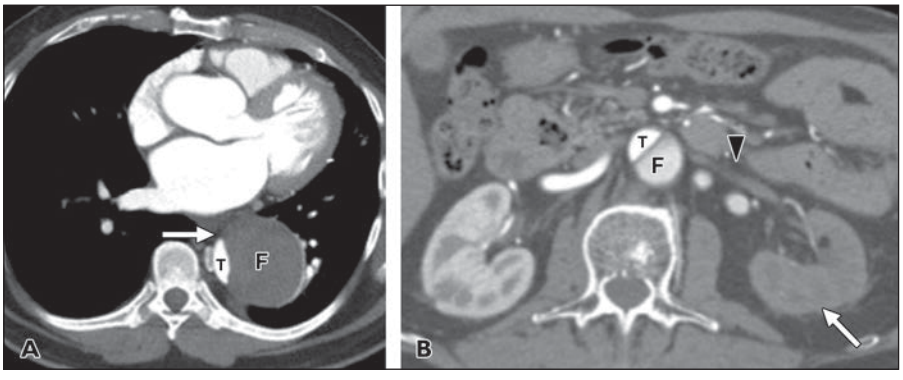

Fig. 6—Aortic dissection, type B according to Stanford classification, in 55-year-old man who presented with acute-onset chest pain.
A, Axial contrast-enhanced CT image through thorax shows large thrombosed false lumen (F) and beak sign (*arrow*); note larger cross-sectional area of false lumen compared with true lumen (T).
B, Axial CT image obtained at level of kidneys reveals unopacified false lumen flap extends into left renal artery (*arrowhead*) and there is no contrast enhancement of left kidney (*arrow*), which is consistent with profound ischemia and infarction. This type of obstruction is treated with intravascular stent placement. T = true lumen, F = false lumen.

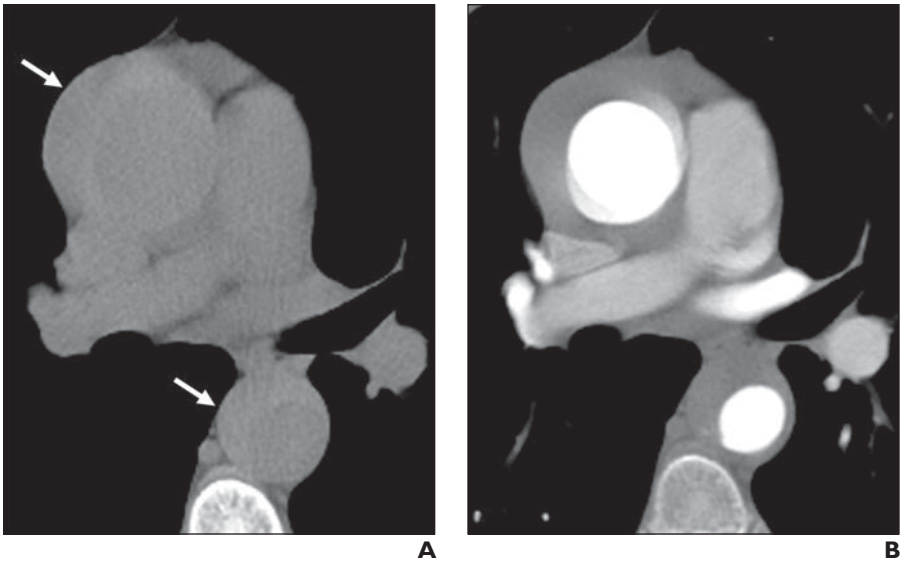

Fig. 7—Dissection variant, intramural hematoma (IMH), type A according to Stanford classification, in 60-year-old woman who presented with acute chest pain.
A, Unenhanced CT angiogram shows hyperdense crescents (*arrows*) along anterolateral surface of ascending aorta and descending aorta.
B, Axial contrast-enhanced CT angiogram shows neither flow channel nor intimal flap, which are characteristic of IMH. One way to differentiate IMH from thrombosed false lumen of aortic dissection is that IMH maintains constant circumferential relationship with aortic wall, whereas dissection tends to spiral longitudinally.

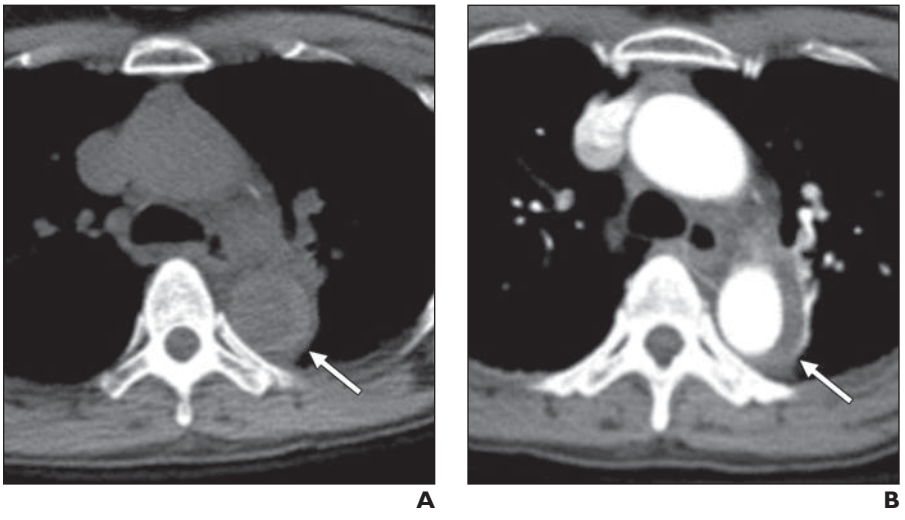

Fig. 8—Intramural hematoma (IMH), type B according to Stanford classification, in 64-year-old woman who presented with acute chest pain.
A, Axial unenhanced CT image shows crescentic high attenuation within aortic wall (*arrow*). Displaced intimal calcifications may also be seen (not shown).
B, Contrast-enhanced CT image shows smooth crescentic region of hypoattenuation (*arrow*).

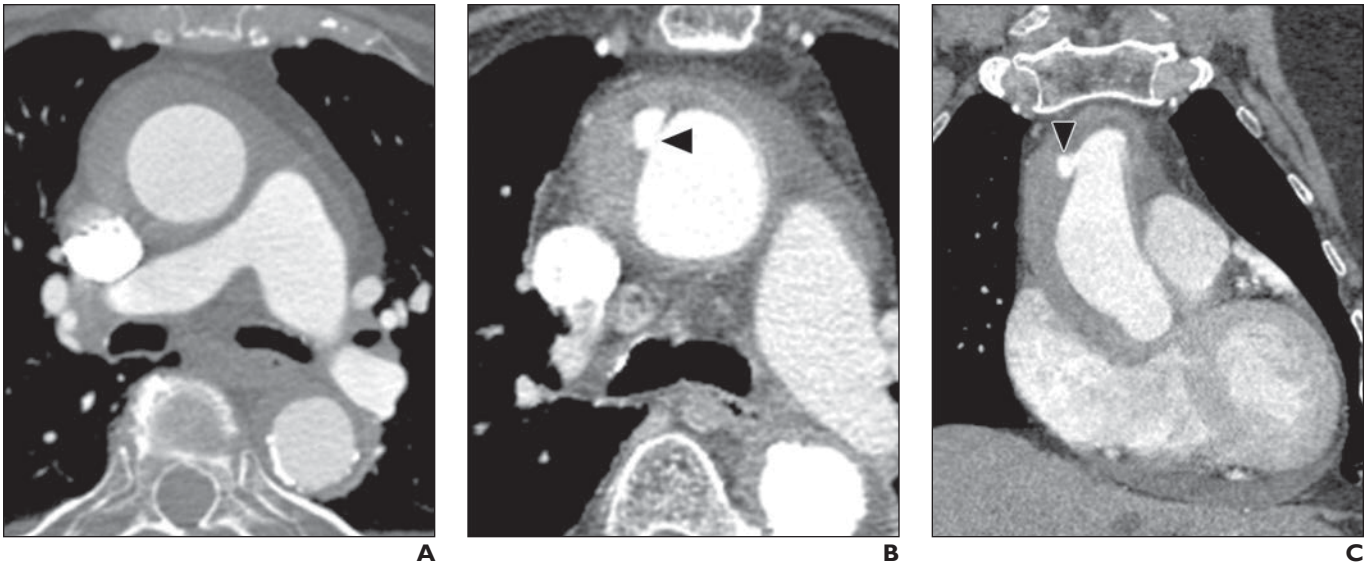

Fig. 9—Intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU), and type A aortic dissection, according to Stanford classification, in 82-year-old nonsmoking woman with history of hypertension, coronary artery disease, and dyslipidemia who presented with severe chest pain.
A, Initial CT angiogram obtained on day 1 shows dense circumferential mural thickening of ascending aorta extending through arch and proximal descending aorta; diagnosis of type A IMH was made.
B and **C,** CT angiography was repeated 72 hours after presentation because patient reported that progressive chest pain had worsened. Axial (**B**) and sagittal (**C**) CT angiograms show worsening, enlarged IMH, which appears more dense than on day 1 (**A**), and reveal interval increase in aortic root diameter. New 1-cm penetrating ulcer (*arrowheads*) is seen along lateral aspect of ascending aorta. At surgery, ascending aortic IMH with active bleeding into type A dissection was found. Ulcer was repaired with Hemashield vascular graft and aortic valve required resuspension with pledgeted suture.

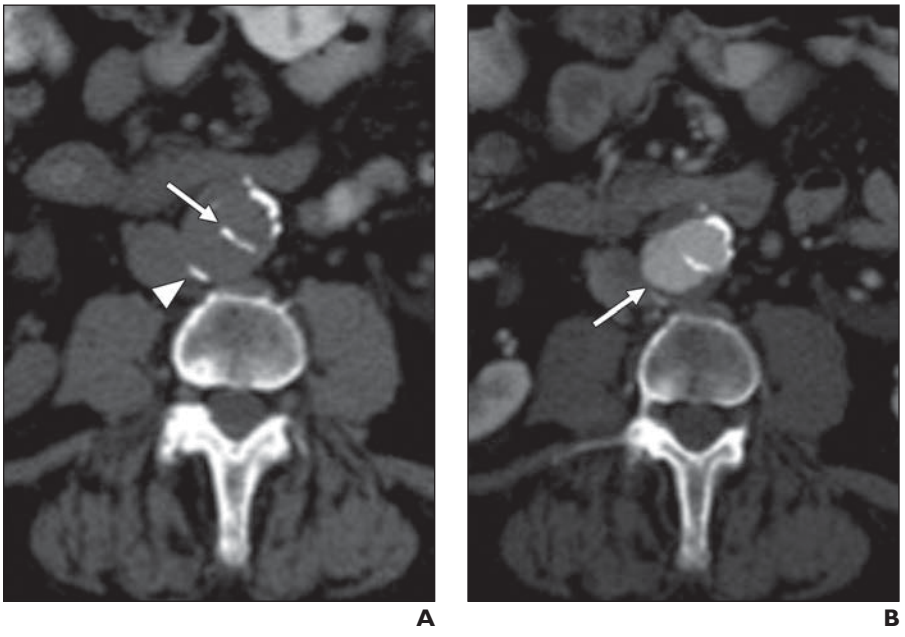

Fig. 10—Penetrating atherosclerotic ulcer (PAU) in 75-year-old man.
A, Axial unenhanced CT image shows atherosclerotic calcification of aortic wall (*arrowhead*) and medially displaced intimal calcifications (*arrow*).
B, Contrast-enhanced CT image shows focal collection of contrast material outside aortic lumen (*arrow*). PAU can be differentiated from other acute aortic diseases by presence of extensive atherosclerotic disease and vessel ectasia and by lack of compression of aortic lumen.

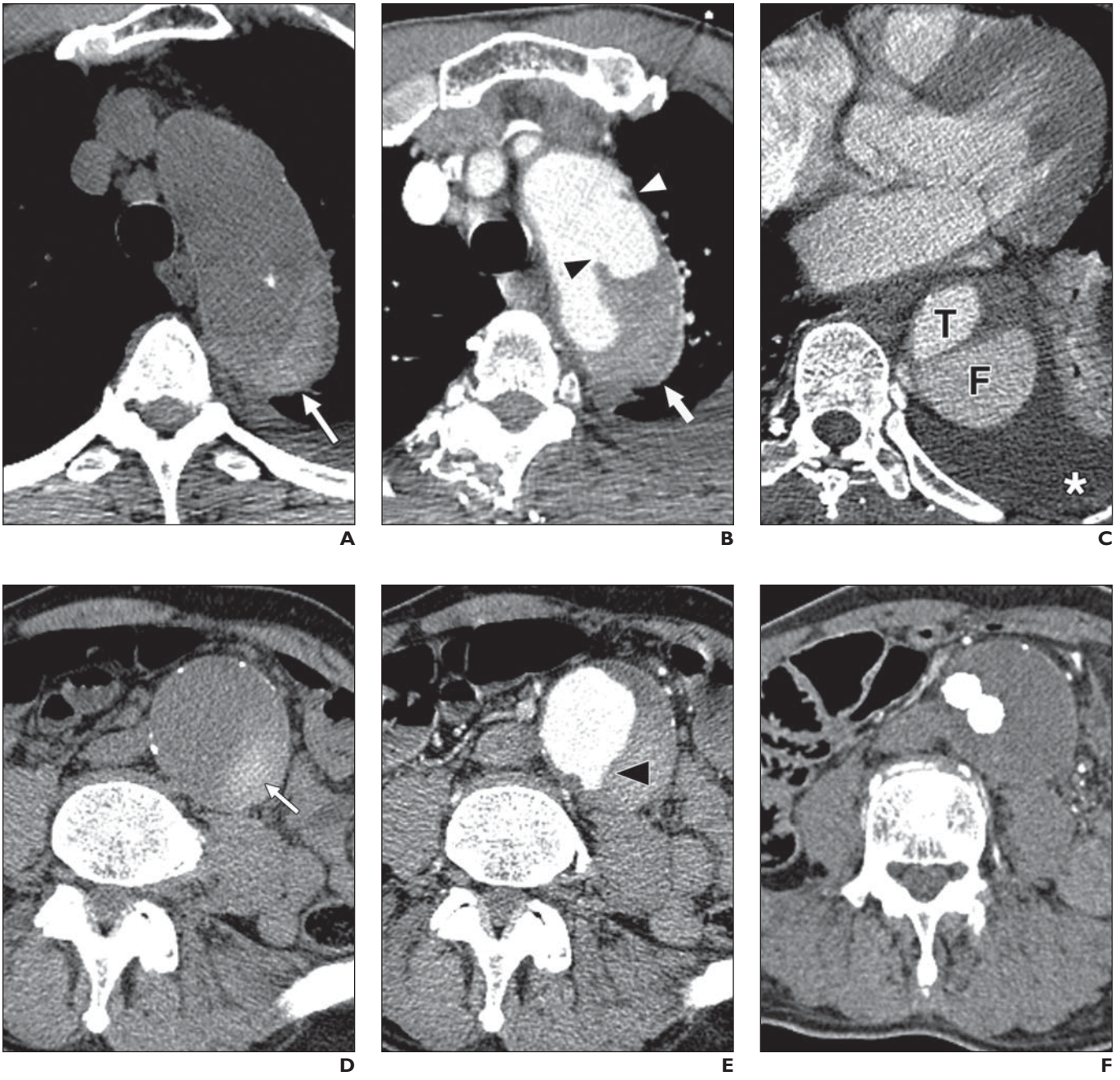

Fig. 11—Penetrating atherosclerotic ulcer (PAU) and intramural hematoma (IMH) in type B aortic dissection in 70-year-old man with known type B aortic dissection, according to Stanford classification, from PAU who presented with new-onset abdominal pain.
A, Unenhanced CT image of upper chest reveals hyperdense IMH along proximal descending aorta (*arrow*).
B, Contrast-enhanced CT image obtained at same level as **A** reveals craterlike lesion in lateral wall of aortic arch (*arrowheads*); this lesion, which shows associated hemorrhage (*arrow*), is consistent with PAU.
C, Contrast-enhanced CT angiogram of lower chest depicts true (T) and false (F) lumina of type B aortic dissection. Note bilateral lower lobe atelectasis and left pleural effusion (*asterisk*).
D, Unenhanced CT image obtained 2 cm above level of aortic bifurcation shows high-attenuation thickening of aortic wall (*arrow*), representing IMH.
E, Axial contrast-enhanced CT image shows PAU posteriorly (*arrowhead*).
F, Contrast-enhanced CT image obtained after aortobiiliac stent-graft placement shows interval resolution of IMH.

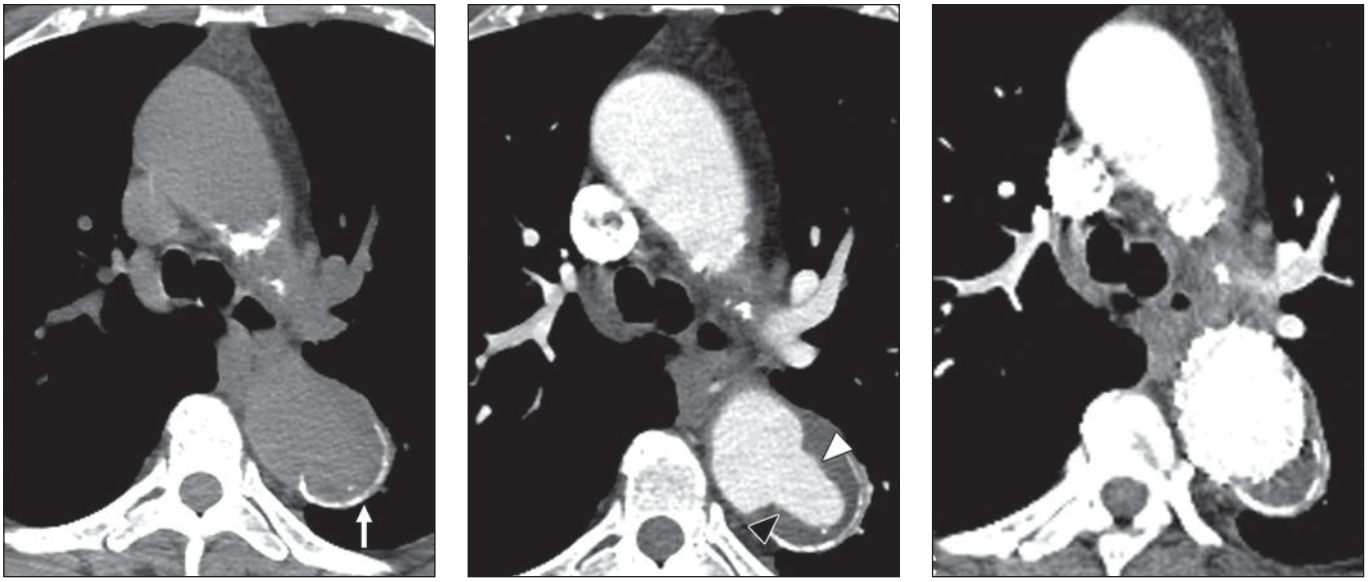

Fig. 12—Saccular aneurysm from penetrating atherosclerotic ulcer (PAU) in 66-year-old-woman with medical history of hypertension who presented with severe left-sided chest pain.
A, Unenhanced CT image shows aneurysmal dilatation of proximal descending thoracic aorta with extensive atherosclerotic mural calcification (*arrow*).
B, Axial contrast-enhanced CT image illustrates contrast material–filled saccular aneurysm caused by PAU (*arrowheads*).
C, Contrast-enhanced CT image obtained after endovascular aneurysm repair.

APPENDIX 1: Factors That Contribute to Acute Aortic Syndrome

Conditions
- Long-standing arterial hypertension
- Atherosclerosis
- Dyslipidemia
- Pregnancy
- Connective tissue disorders: hereditary fibrillinopathies, Marfan syndrome, Ehlers-Danlos syndrome, hereditary vascular disease
- Congenital anomalies: bicuspid aortic valve, coarctation
- Vascular inflammation: giant cell arteritis, Takayasu arteritis, Behçet disease, syphilis, Ormond disease

Trauma
- Deceleration trauma
- Iatrogenic trauma: cardiac or aortic surgery

Lifestyle
- Smoking

APPENDIX 2: Atypical Features of the Intimal Flap [14]

Atypical Configuration of the Intimal Flap
- Dissection of the entire intima with a circumferential intimal flap
- Filiform (extremely narrow) true lumen, which can have ischemic complications
- Calcified false lumen, which is seen in chronic dissection
- A three-channel aorta (Mercedes-Benz sign) (Fig. 3) or an aorta with several false channels
- Intimomedial intussusception

FOR YOUR INFORMATION

The reader's attention is directed to part 2 accompanying this article, titled "Nontraumatic Acute Aortic Emergencies: Part 2, Pre- and Postsurgical Complications Related to Aortic Aneurysm in the Emergency Clinical Setting," which begins on page 666.