DOI: https://doi.org/10.17816/rmmar654012

Diagnosis of Acute Aortic Syndromes Using Computed Tomography

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ABSTRACT

Acute aortic syndrome refers to a group of life-threatening conditions characterized by acute injury to the aortic wall, primarily involving disruption of the intima and media. Acute aortic syndrome encompasses a spectrum of interrelated and overlapping clinical and morphological entities, including classic aortic dissection, intramural hematoma, penetrating aortic ulcer, and limited intimal tear. Differentiation of these variants based on clinical presentation and physical examination findings is not feasible. Imaging plays a pivotal role, and definitive diagnosis and classification of acute aortic syndrome variants are possible only through imaging studies. Multidetector computed tomography, transesophageal echocardiography, and magnetic resonance imaging are the primary imaging modalities used for the diagnosis of acute aortic syndrome, with contrast-enhanced multidetector computed tomography considered the gold standard. When classical imaging signs of a specific type of acute aortic syndrome are present, diagnosis is generally straightforward. However, a broad spectrum of imaging findings exists. In some cases, a single computed tomography scan may not allow for reliable differentiation among acute aortic syndrome variants. This limitation arises from the fact that these pathological entities may occur independently, progress from one to another, or coexist. The pathophysiology and clinical course of intramural hematoma and penetrating atherosclerotic ulcer of the aorta remain subjects of ongoing debate. In particular, the classification of intramural hematoma as a distinct acute aortic syndrome variant continues to be controversial. This article provides a concise overview of the current understanding of the pathophysiology, natural history, prognosis, and multidetector computed tomography-based diagnosis of the less common acute aortic syndrome variants, specifically intramural hematoma and penetrating atherosclerotic ulcer.

Keywords: aortic dissection; aortic intramural hematoma; computed tomography; acute aortic syndrome; penetrating atherosclerotic ulcer; aortic ulcer.

To cite this article

Ryazanov VV, Sadykova GK, Gozheva AR, Kutsenko VP, Menshikova SV. Diagnosis of Acute Aortic Syndromes Using Computed Tomography. *Russian Military Medical Academy Reports*. 2025;44(1):113–121. DOI: https://doi.org/10.17816/rmmar654012

Received: 06.02.2025 **Accepted:** 02.03.2025 **Published:** 31.03.2025

ECO•VECTOR

УДК 616.132-002.1-073.756.8
DOI: https://doi.org/10.17816/rmmar654012

Диагностика острого аортального синдрома при компьютерной томографии

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АННОТАЦИЯ

Острый аортальный синдром — это внезапно возникшие состояния в основе которых лежит поражение стенки аорты в виде разрушения интимы и медии. Острый аортальный синдром включает в себя взаимосвязанные, пересекающиеся клинически и морфологически патологические состояния: классическое расслоение аорты, интрамуральную гематому, пенетрирующую аортальную язву, очаговый надрыв аорты. Различить варианты острого аортального синдрома по симптомам и при физикальном обследовании невозможно. Визуализация играет ведущую роль и варианты острого аортального синдрома могут дифференцироваться только с помощью визуализационных методов. Мультиспиральная компьютерная томография, чреспищеводная эхокардиография и магнитно-резонансная томография являются основными методами визуальной диагностики острого аортального синдрома, но безусловный приоритет принадлежит компьютерной томографии с внутривенным введением контрастного вещества. В случае классических визуализационных проявлений отдельно взятого варианта острого аортального синдрома сложностей в постановке диагноза, как правило, не возникает, но существует широкий спектр результатов визуализации. В ряде случаев дифференцировать варианты острого аортального синдрома при однократном компьютерно-томографическом исследовании невозможно. Это обусловлено тем, что патологические состояния могут существовать самостоятельно, переходить из одного в другое или сочетаться друг с другом. В настоящее время предметом многочисленных споров остаются патофизиология и течение интрамуральной гематомы, пенетрирующей атеросклеротической язвы аорты. Оспаривается целесообразность включения в классификацию острого аортального синдрома интрамуральной гематомы как отдельного варианта. В рамках статьи кратко представлено современное понимание патофизиологии, течения, прогноза, компьютерно-томографической диагностики редко встречающихся вариантов острого аортального синдрома: интрамуральной гематомы и пенетрирующей атеросклеротической язвы аорты.

Ключевые слова: диссекция аорты; интрамуральная гематома аорты; компьютерная томография; острый аортальный синдром; пенетрирующая атеросклеротическая язва; язва аорты.

Как цитировать

Рязанов В.В., Садыкова Г.К., Гожева А.Р., Куценко В.П., Меньшикова С.В. Диагностика острого аортального синдрома при компьютерной томографии // Известия Российской военно-медицинской академии. 2025. Т. 44, № 1. С. 113–121. DOI: https://doi.org/10.17816/rmmar654012

Рукопись получена: 06.02.2025 **Рукопись одобрена:** 02.03.2025 **Опубликована:** 31.03.2025

ЭКО•ВЕКТОР

INTRODUCTION

Acute aortic syndrome (AAS) is a sudden-onset of conditions with similar clinical manifestations, accompanied by aortic wall damage. Various manifestations of AAS are due to aortic intima and media disruption [1]. AAS includes interrelated and clinically and morphologically overlapping conditions: classic aortic dissection (AD), intramural hematoma (IMH), penetrating aortic ulcer (PAU), and localized aortic rupture.

Rubin et al. proposed an alternative classification of AAS based on electrocardiography (ECG)-gated computed tomography (CT) angiography, encompassing AD, PAU, and ruptured aortic aneurysm. The authors did not include IMH in the classification, indicating that it should be regarded as an imaging marker of an acute pathological process [2].

Most studies considered IMH and PAU as variants of aortic dissection within the unified AAS classification. However, the pathophysiology and natural course of IMH and PAU remain debatable, and these conditions are not always associated with aortic dissection [3].

Variants of AAS have similar clinical presentation, with chest pain being the most common symptom—often described as sudden in onset, sharp, intense, and tearing [4]. Distinguishing among AAS variants based on symptoms and physical examination alone is not feasible. Imaging plays a crucial role in differential diagnosis. This may seem to be a straightforward task; however, owing to the wide spectrum of imaging findings, particularly when only a single study is performed, it is often difficult to identify the specific pathology. This limitation is because of the fact that these pathological entities may occur independently, evolve from one into another, or coexist. For example, IMH may progress to aortic dissection, coexist with dissection in separate aortic segments as a mixed lesion, or result from a penetrating atherosclerotic ulcer [5]. The present study aim to evaluate the differences among the three AAS variants: classic AD, IMH, and PAU.

GENERAL COMPARATIVE CHARACTERISTICS OF ACUTE AORTIC SYNDROME VARIANTS

In the general population, AAS incidence is estimated at 3.5–6 cases per 100,000 individuals per year, with AD being the most common manifestation, accounting for 85%–95% of all cases [6]. The incidence of aortic ulcer among patients with AAS is 2%–7% of cases. In contrast to AD and PAU, the prevalence of IMH within AAS range from 5% to 27%. Notably, the incidence of IMH was higher in Asian cohorts compared with the International Registry of Acute Aortic Dissection: 28.9% vs 5.7%. This may be because IMH is diagnosed more frequently in Asian hospitals than in Western centers. Moreover, the low IMH rate in the international registry may be attributed to the fact that the registry's participating centers are specialized hospitals, and some IMH cases may have gone undiagnosed at primary care centers. Additionally, discrepancies in reported incidence may result from the use of different imaging modalities [2, 6–8].

Typically, patients with IMH and PAU are older than those with classic AD (69±10 vs 62±14 years). IMH and PAU usually occur in individuals in their 70s, 80s, or 90s with a history of hypertension. Patients with PAU frequently have advanced atherosclerosis and calcification of the aorta and of the visceral, brachiocephalic, and peripheral arteries [7, 9].

The localization and extent of AD and IMH are classified using the DeBakey and Stanford systems (Fig. 1). In emergency settings, the Stanford classification is preferred because it determines management strategy: surgical treatment for type A and conservative treatment for type B [5, 8].

Classic AD begins with an intimal tear, allowing circulating blood to enter the media and form an intimo-medial flap that separates the aortic lumen into true and false lumens (Fig. 2*a*). IMH and PAU are not characterized by the formation of an extensive intimo-medial flap or presence of two distinct lumens within the aorta [7] (Fig. 2*b*, *c*). This study focuses on IMH and PAU, whereas computed tomography features of AD at different stages can be found in the authors' article [10].

In classic Stanford type A and B dissections, the intimal tear occurs at sites of greatest hydraulic stress, namely, along the right lateral wall of the ascending aorta or in the descending thoracic aorta near the ligamentum

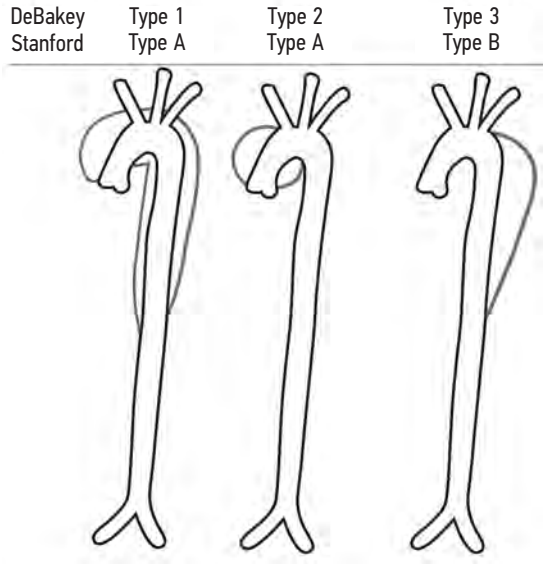

Fig. 1. DeBakey and Stanford classifications of aortic pathology.
Рис. 1. Классификации поражений аорты по Де Бейки и Стэнфорду.

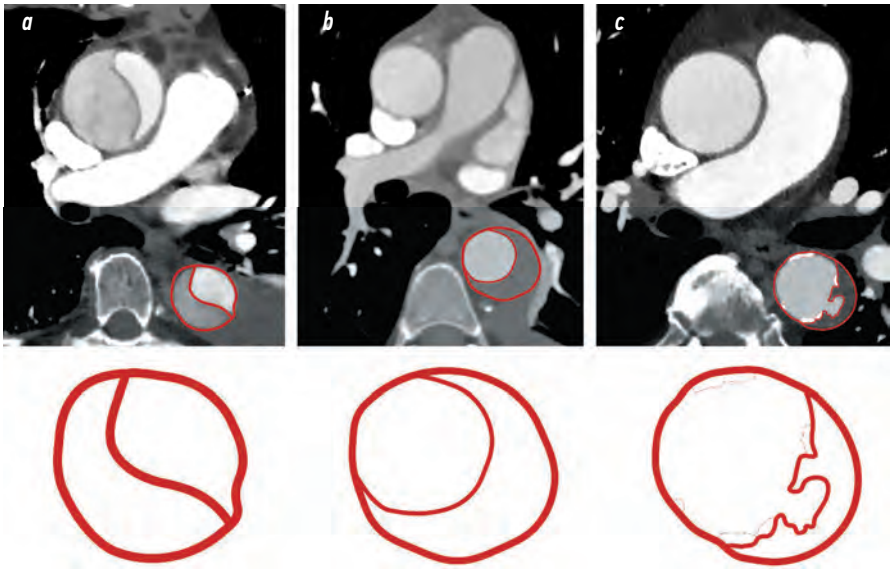

Fig. 2. Aortic dissection (*a*), intramural hematoma (*b*), and penetrating atherosclerotic ulcer (*c*) in the descending aorta, with corresponding schematic illustrations.
Рис. 2. Расслоение аорты (*a*), интрамуральная гематома (*b*), пенетрирующая атеросклеротическая язва (*c*) в нисходящей аорте с соответствующими схемами.

arteriosum. PAU and IMH predominantly affect the descending aorta (90% of PAU cases and 50%–85% of IMH cases). When IMH occurs in combination with PAU, the descending aorta is commonly involved, with the ascending aorta affected in only 9% of cases. Conversely, in isolated IMH, the ascending aorta is involved in 26% of cases [6, 8].

Notably, IMH and PAU frequently occur in aortas with larger diameters than those observed in classic dissection (6.2 and 5.5 cm vs 5.2 cm) and are mostly associated with an underlying aortic aneurysm [11].

Unlike classic dissection, IMH and PAU do not result in damage to or occlusion of aortic branches and are not commonly associated with limb or visceral ischemia.

Clinically, IMH and PAU follow a more malignant course, particularly when they occur in combination, and are associated with a higher risk of aortic rupture compared with classic AD [8, 11]. Furthermore, IMH and PAU located in the ascending aorta or proximal descending aorta present a higher overall complication rate [2].

IMAGING DIAGNOSIS OF ACUTE AORTIC SYNDROME

Currently, imaging findings play a central role in facilitating timely and appropriate management of AAS.

In AAS, imaging primarily aim to detect abnormalities of the aortic wall and assess the location, extent, and complications of the disease. Radiology reports should specify the type of aortic involvement according to the Stanford classification, maximum aortic diameter, maximal thickness of the intramural hematoma, presence of focal contrast enhancement within the hematoma, length and depth of any ulcer-like projection, presence of pleural or pericardial effusion, and changes in the periaortic fat [5].

Multidetector CT, transesophageal echocardiography (TEE), and magnetic resonance imaging (MRI) are the primary modalities for the imaging evaluation of AAS [7, 8]. CT is the preferred imaging modality in emergency departments owing to its availability, rapid data acquisition, high diagnostic accuracy, noninvasiveness, and wide field of view enabling comprehensive anatomic assessment of the entire aorta and its branches, whereas MRI and TEE are used as supplementary techniques [4, 5, 7, 8, 12]. Color Doppler echocardiography may be preferable to any other imaging modality for detecting subtle intimal injuries; however, this topic is beyond the scope of the present study.

CT should be performed with and without contrast enhancement to detect acute hematoma. The use of ECG-gated CT allows for motion artifact-free imaging, although it is not always feasible because of additional time requirements and technical constraints [4, 5, 12].

Thin-section CT images (<1.25 mm) are used for assessment; however, non-contrast images reconstructed with a 5 mm slice thickness can be useful for visualization of IMH owing to volume averaging and reduced image noise. Applying a narrow window setting (width: 200 HU; level: 40 HU) enhances hematoma visualization on non-contrast CT scans [5].

CT angiography should be performed using a high-concentration iodine contrast agent (300–350 mg/mL), injected at 2–5 mL/s, to achieve an aortic enhancement of 150–250 HU during the arterial phase [5, 12].

Contrast-enhanced imaging should include the entire aorta, from the proximal segments of the arch branches to the iliac arteries. This approach is confirmed by the potential for aortic dissection to extend into the supra-aortic vessels and propagate distally over a considerable length.

INTRAMURAL HEMATOMA

IMH has been attributed to spontaneous rupture of the *vasa vasorum*, resulting in hemorrhage within the aortic wall without overt intimal disruption. The preserved integrity of the intimal layer is a key distinction between IMH and classic AD. However, several reports have described small intimo-medial tears that are not visible on preoperative imaging but are found during surgery. With advances in modern imaging, particularly the improved spatial resolution of thin-slice multidetector CT, such subtle intimo-medial tears can now be identified in patients with IMH [2, 5, 6, 9].

There is ongoing debate regarding the role of microintimal tears and the primary triggering factor in IMH pathogenesis. Is it a microintimal tear or rupture of the *vasa vasorum*? Which event occurs first—the rupture of the *vasa vasorum* or the intimal-medial tear? Several specimens from patients with AD and IMH have demonstrated erythrocyte extravasation from the *vasa vasorum*, even in the absence of detectable intimal-medial tears. In both AD and IMH, aortic wall dissection has been observed to follow the path of the *vasa vasorum*. Some studies showed that dysfunction of the *vasa vasorum* due to prolonged ischemia may be the common initiating event in both AD and IMH, with rupture of the *vasa vasorum* and development of intimal-medial tears occurring secondarily [5].

According to current understanding, IMH may be a subtype or precursor of AD. Many studies considered IMH as a variant of AD with a sealed and thrombosed false lumen [2, 6, 8, 9]. Approximately 12%–47% of patients with IMH progress to AD, supporting the impression of a continuum between these conditions. Currently, it is assumed that some cases of AAS that were misclassified as IMH on CT imaging were cases of AD with a small entry tear that spontaneously sealed, with no distal reentry and a completely thrombosed false lumen [2, 5, 6].

Gutschow et al. proposed using the number of intimal-medial tears for distinguishing IMH from AD: AD is characterized by two intimal-medial tears—an entry tear (from the lumen into the wall) and exit tear (from the wall back into the lumen)—whereas IMH involves only an entry intimal-medial tear. Moreover, the mean size of the intimal-medial tear is significantly smaller in IMH (1.8±1.0 cm) than in AD (2.9±1.2 cm) [5].

Hemorrhage from the *vasa vasorum* may play a primary pathogenic role in true cases of IMH, wherein no intimal-medial tear is identified on imaging or during surgery [9]. Additionally, IMH may occur secondarily in the context of a penetrating aortic ulcer. In PAU, the hematoma is limited in extent, despite the presence of direct communication between the aortic lumen and media [2].

IMH may regress with complete resolution or progress to classic dissection, limited intimal tear, or pseudoaneurysm formation. The most serious complication is aortic rupture [2, 4, 11, 13].

There are two types of limited intimal tears associated with IMH. The first type is characterized by an intima disruption with an ulcer-like projection that develops during hematoma evolution. This is believed to represent the true entry tear in AD with a completely thrombosed false lumen [4]. The second type is manifested by an intimal disruption at the origin of the aortic branch vessel.

Notably, an ulcer-like projection observed during the acute phase of IMH, particularly in the ascending aorta or aortic arch, is associated with poor prognosis and frequently progresses to AD, pseudoaneurysm, or rupture. In contrast, an ulcer-like projection identified in the subacute or chronic phase may regress or result only in aortic dilatation. Greater length and depth of the projection correlate with a higher rate of complications. The proposed threshold values for identifying high-risk patients are a length of 10–20 mm and depth of 5–10 mm [4, 5, 7]. The second type of tear occurs more commonly in the descending aorta and is not associated with IMH progression, although it may lead to incomplete hematoma resorption [2, 5].

On non-contrast CT, IMH appears as circular (ring-shaped) or semicircular (crescent-shaped) aortic wall thickening with increased attenuation, demonstrating attenuation values up to +60–70 Hounsfield units. In the subacute phase, IMH may be isodense with the blood in the aortic lumen on non-contrast CT. The thickened aortic wall does not enhance after intravenous contrast administration [4, 5, 7, 12]. Normally, aortic wall thickness is <3–4 mm on any imaging modality; therefore, a thickness >5 mm is considered pathologic [7, 12]. On CT, high-attenuation wall thickening extends longitudinally and transversely, but does not follow a spiral course [14]. In most IMH cases, no aortic lumen narrowing or intima displacement into the aortic lumen is observed. If intima displacement occurs, it is directed toward the lumen [4, 12]. On post-contrast CT, IMH is characterized by a single aortic lumen, a smooth inner aortic surface, and the absence of an intimal-medial flap [6] (Fig. 3*a–c*; Fig. 4*a–c*).

A localized area of contrast enhancement within the IMH on post-contrast CT images shows a limited intimal tear with contrast extravasation into the hematoma. An ulcer-like projection extends from the aortic lumen

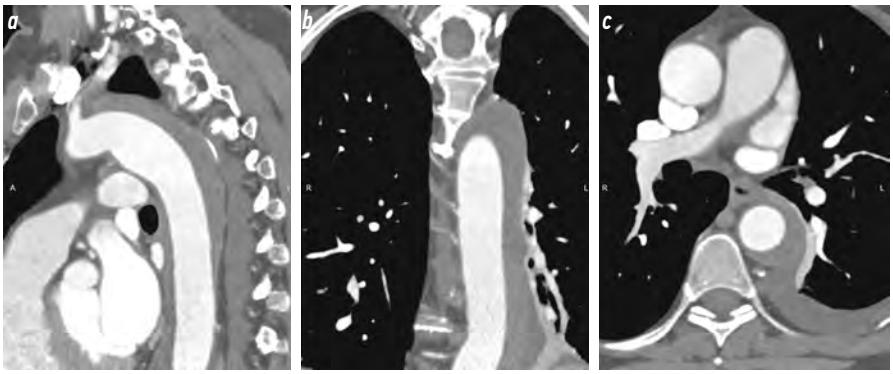

Fig. 3. Intramural hematoma in the descending aorta in sagittal (*a*), coronal (*b*), and axial (*c*) planes during the postcontrast phase, seen as a crescentic thickening of the aortic wall without luminal narrowing.
Рис. 3. Интрамуральная гематома в нисходящей аорте в сагиттальной (*a*), коронарной (*b*), аксиальной (*c*) плоскостях в постконтрастную фазу в виде полуциркулярного утолщения стенки без сужения просвета аорты.

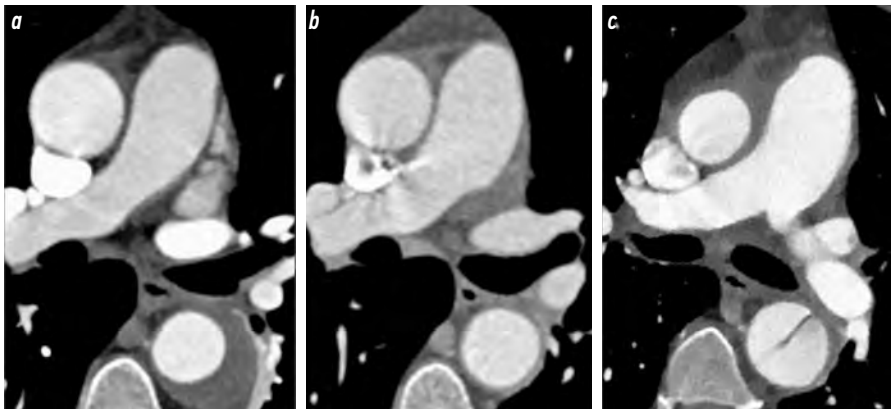

Fig. 4. Temporal changes in intramural hematoma in the descending aorta shown in Fig. 3 (*a*). Not visualized 11 months after the initial study (*b*), aortic dissection detected 1 month after the last scan (*c*).
Рис. 4. Интрамуральная гематома, представленная на рис. 3 в нисходящей аорте (*a*) в динамике. Через 11 мес от первоначального исследования гематома не визуализируется (*b*), через месяц от последнего исследования появилось расслоение аорты (*c*).

into the IMH, with a visible communication of >3 mm in type 1 [7]. In contrast, type 2 intramural flow is characterized by the absence of a visualized communication with the aortic lumen, typically measuring <2 mm. Type 2 is more frequently found in the descending aorta, and the vessel course can be followed [5].

CT enables identification of features associated with poor prognosis in patients with IMH [2, 4, 5, 7, 14], including the following:

- involvement of the ascending aorta;
- maximal aortic diameter >48–55 mm in the ascending aorta and >40–41 mm in the descending aorta;
- maximal hematoma thickness >10 mm;
- limited intimal-medial disruption with or without an ulcer-like projection during the acute phase of the hematoma;
- ulcer-like projection depth >10 mm; and
- presence of periaortic hematoma.

Recently, a novel CT finding, namely, the broken crescent sign, has been described and may indicate impending aortic rupture in patients with acute IMH. On non-contrast CT, it presents as a focal defect within the hyperdense crescentic hematoma. On post-contrast CT, it is characterized by a smooth, focal defect with blunt margins in the crescentic hematoma, corresponding to a localized bulge of the contrast-filled aortic lumen without contrast extravasation. According to the authors, this reflects a small adventitial tear with partial outward displacement of the intramural thrombus, resulting in the disrupted crescentic configuration on CT. Conversely, a PAU appears on CT as a contrast-filled outpouching with irregular, sharply angulated margins extending into the aortic media [14].

The prognostic significance of pleural and pericardial effusions in IMH remains controversial; however, most studies revealed an association with adverse outcomes [5].

PENETRATING ATHEROSCLEROTIC ULCER

PAU was first described by Stanson et al. in 1986. It is a pathological process characterized by ulceration of the surface of an atheromatous plaque extending through the internal elastic lamina into the aortic media [7, 8, 11–13].

However, not all aortic ulcers represent PAUs. The term aortic ulcer is a general morphological designation. It may reflect either a primary etiology—atherosclerotic, inflammatory, infectious, traumatic, or iatrogenic—or a secondary process occurring during IMH evolution [7, 8, 13, 15]. Penetrating aortic ulcers present as crater-like lesions extending from the aortic lumen into the periaortic space. These are focal abnormalities that do not propagate longitudinally [13, 16].

In AAS, the term most commonly refers to a PAU arising in the setting of progressive atherosclerosis [13]. In patients with PAU, the site of dissection corresponds to the ulcer location. Multiple PAUs of varying lengths and depths are identified simultaneously [11].

PAUs are frequently accompanied by hemorrhage into the aortic media, resulting in the formation of a localized IMH [7, 8, 11]. In the presence of an aortic ulcer, the associated IMH does not extend, possibly because of surrounding transmural inflammation and relative adhesion between the layers of the aortic wall. However, based on observations of IMH in the ascending aorta arising from a PAU in the descending aorta, some studies have reported the possibility of retrograde extension of IMH from the descending to the ascending segment [13]. IMH is considered a marker of acute and potentially unstable disease in patients with PAU [12].

The natural course of PAU remains controversial. Previously, it was believed that PAU followed a malignant course, with a high risk of rupture or progression to overt AD; however, subsequent studies have shown that its clinical trajectory may be more favorable in certain cases [7]. Although spontaneous aortic rupture rarely occurs in PAU in the absence of marked progressive dilatation, it appears to occur more frequently in PAU than in AD [7, 8]. Additionally. PAU may lead to the formation of fistulous communications with adjacent anatomical structures [12].

On CT, the primary sign of PAU is a contrast-filled ulcer crater, frequently seen as a saccular outpouching within a thickened aortic wall in the setting of advanced atherosclerosis [7, 11, 12]. A crucial feature of PAU is the presence of irregular crater margins accompanied by unevenly thickened and calcified intima [8]. PAU does not involve formation of an intimal flap dividing the aorta into true and false lumens. PAU size may vary from a few millimeters to 2.5 cm, with a depth of up to 3 cm [12]. On non-contrast CT, the hematoma adjacent to the ulcer appears as a hyperdense area (Fig. 5*a–d*).

DIFFERENTIAL DIAGNOSIS OF INTRAMURAL HEMATOMA AND PENETRATING ATHEROSCLEROTIC ULCER ON COMPUTED TOMOGRAPHY

From an imaging standpoint, the primary differential diagnosis of IMH includes classic AD with a completely thrombosed false lumen and mural thrombus formation.

When distinguishing IMH from other aortic conditions involving mural thrombi, the first diagnostic step is to identify the intimal layer and assess the inner surface of the thickened aortic wall. IMH is differentiated from mural thrombus based on its relationship to the intima: the mural thrombus lies external to the intima, whereas the hematoma in IMH is located beneath it, resulting in outward and inward displacement of the intimal layer.

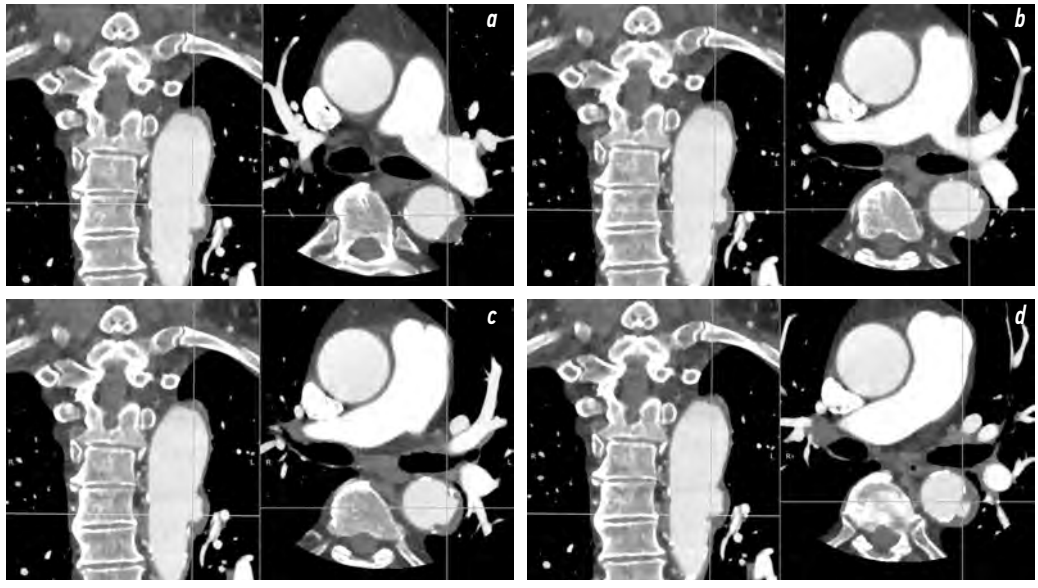

Fig. 5. Penetrating atherosclerotic ulcer in the frontal and axial planes at corresponding levels (*a–d*).
Рис. 5. Пенетрирующая атеросклеротическая язва во фронтальной плоскости и в аксиальной плоскости на соответствующих уровнях (*a–d*).

DOI: https://doi.org/10.17816/rmmar654012

The inner margin of an IMH appears smooth, whereas mural thrombi exhibit irregular borders. On non-contrast CT, acute IMH presents as a hyperdense area, whereas mural thrombi are usually hypodense [2, 4, 17].

Acute-phase IMH can be differentiated from AD when classic signs of dissection are present, namely, an intimal-medial flap dividing the aorta into two communicating lumens. However, early diagnosis of IMH may be challenging when the false lumen in AD is completely thrombosed. In such cases, contrast-enhanced CT may reveal a small intimal tear or contrast communication; however, these findings are rarely detected in the acute phase. In select cases, a definitive diagnosis may only be established intraoperatively by identifying the entry tear [2, 4].

The differential diagnosis of PAU includes ulcer-like projections associated with IMH and ulcerated atherosclerotic plaques [7].

An ulcerated atherosclerotic plaque is marked by irregular margins and confinement to the intimal layer; it does not extend beyond the calcified intima and is not associated with an IMH. In contrast, a PAU is an ulcerative lesion that penetrates through the intima into the aortic wall and is identified by a saccular outpouching in the aortic wall surrounded by a hematoma [7, 8, 11, 17].

One of the most diagnostically challenging distinctions is that between PAU and an ulcer-like projection associated with IMH. A PAU may give rise to an IMH, whereas an ulcer-like projection may develop during the natural course of IMH, and both may appear identical on CT. A distinguishing feature is that an ulcer-like projection is not visualized on initial CT but becomes detectable on follow-up imaging. Some studies considered an ulcer-like projection to represent a newly formed intimal defect, whereas other studies indicated that a preexisting intimal-medial tear may not have been visible on initial imaging. In the acute phase of IMH (within 14 days of symptom onset), an ulcer-like projection is usually not associated with an intimal-medial flap, whereas in the subacute or chronic phase (after 14 days), a short flap is often detectable [5, 7].

Ultimately, PAU cannot be differentiated from an ulcer-like projection in IMH without follow-up imaging [5]. Given the time-dependent morphological changes in AAS variants and diagnostic limitations of a single CT scan, follow-up CT examinations are critical.

CONCLUSION

Patients with different morphological variants of AAS often present with similar clinical manifestations. However, the underlying pathophysiology and CT features differ. In contrast to IMH and PAU, an extensive intimal-medial flap is identified in classic AD, dividing the aortic lumen into true and false lumens. In certain cases, a single CT scan is insufficient for distinguishing between AAS variants. Follow-up CT imaging and comparison with prior studies are required to differentiate aortic dissection with complete thrombosis of the false lumen from IMH and distinguish an ulcer-like projection in IMH from a PAU. At present, no formal guidelines exist regarding the timing and frequency of follow-up CT imaging in patients with AAS. Therefore, decisions should be individualized and guided by a multidisciplinary team—including a radiologist—based on clinical and radiologic findings.

ADDITIONAL INFO

Author contribution. All authors have made a significant contribution to the development of the concept, research and preparation of the article, read and approved the final version before publication.
Competing interests. The authors declare that they have no competing interests.
Funding source. This study was not supported by any external sources of funding.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.
Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.
Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

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