

Type B Intramural Hematoma of the Aorta: Evolution and Prognostic Value of Intimal Erosion

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ABSTRACT

Purpose: The prognosis and treatment of type B intramural hematoma (IMH) remain unclear. Intimal erosions could be the target of preventive endovascular treatment, but we have no therapeutic criterion on which to decide preventive treatment.

Materials and Methods: A prospective multislice computed tomography (CT) study was carried out in 44 patients with type B IMH to assess morphologic evolution and intimal erosion to determine reliable predictive factors that would permit endovascular treatment. Follow-up range was 24–1,440 days.

Results: Intimal anomalies consisted of intimal erosion and aortic branch artery lesions. Fifty-eight intimal anomalies were seen on initial CT in 38 patients (86%). Twenty-five anomalies in 22 patients were considered as intimal erosions, of which nine (36%) were visible only on delayed-phase CT. Hematoma regressed in 23 patients (53%). Twenty-one patients (47%) showed morphologic progression. Eleven of these (52%) required endovascular treatment. Twenty of the 25 initial intimal erosions (80%) progressed and caused 19 of the 21 morphologic evolutions (90%). Progression was related to initial intimal erosion and to IMH thickness. Intimal erosion measuring greater than 10 mm had unfavorable progression at 1 month (positive predictive value, 100%).

Conclusions: Complications or morphologic progression were related to a preexisting intimal anomaly visualized on initial CT. Multislice CT with systematically delayed phase and millimetric thin slices could increase the detection rate of intimal anomalies.

ABBREVIATION

IMH = intramural hematoma

Intramural hematoma (IMH) accounts for approximately 5%–15% of acute aortic syndromes (1–3). There is no clear consensus regarding optimal management strategies, but involution of the ascending aorta (ie, type A IMH) or presence of a giant ulceration are important predictors of poor outcome (4–7).

Patients with IMH limited to the arch or the descending aorta (ie, type B IMH), and who are without complications on admission, present the greatest challenge (8). Based on analogies to classical aortic dissection, treatment with watchful follow-up and antihypertensive therapy appears as a safe strategy; however, despite optimal medical treatment, in some cases the disease progresses, most often without clinical symptoms. We have no therapeutic criteria on which to decide preventive treatment (9–11). Because of advances in imaging techniques, intimal anomalies are visualized increasingly often during the initial investigation of IMH. They are often considered as irregular atheromatous plaques (7) or may go unnoticed (10,11), although intimal integrity is impossible to affirm regardless of the imaging technique used (12).

The current study prospectively analyzed 44 patients with type B IMH without complications on admission. Its aim was to examine simultaneously the morphologic evolution of the aorta and all computed tomography (CT) images that were compatible with intimal erosion to

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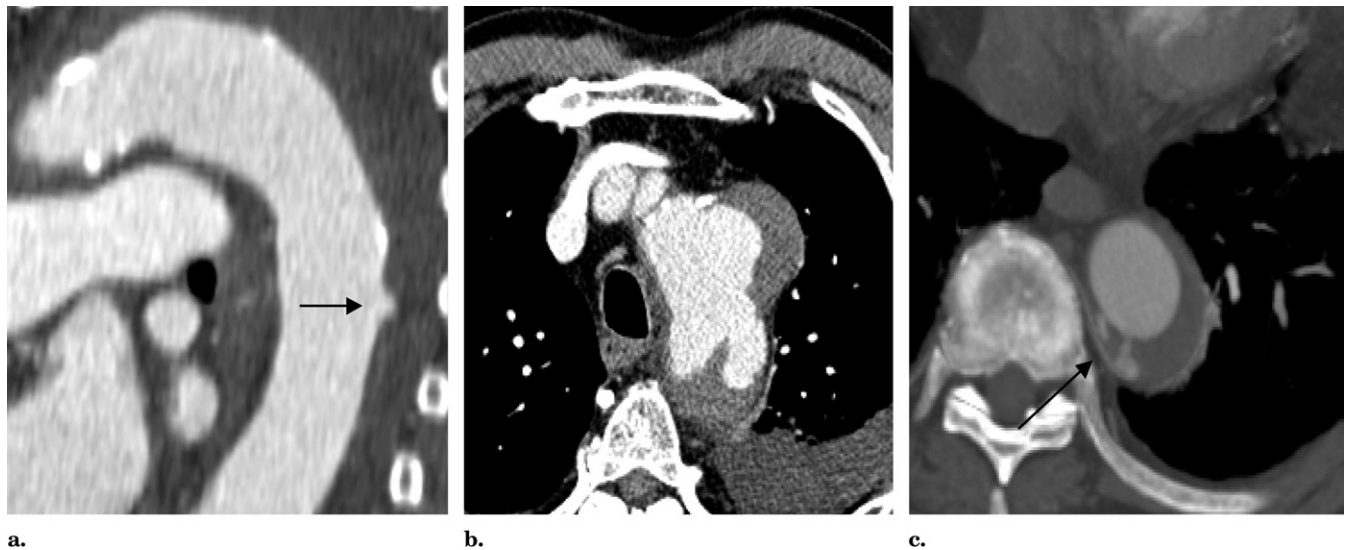


Figure 1. Intimal anomalies: intimal erosion (black arrow, **a**) and large intimal erosion (or penetrating aortic ulcer) that extends through the aortic wall and appears as a saccular aneurysm (**b**). These lesions were excluded. (**c**) Aortic branch artery lesion (black arrow). Note the distribution along the internal wall, the absence of intimal defect, and communication with an intercostal artery.

determine the prognostic implication of intimal anomalies.

MATERIALS AND METHODS

Patient Selection

From January 2003 to December 2008, 284 patients were admitted to our institution for nontraumatic acute aortic syndrome. The diagnosis of IMH was established by CT scan within 24 hours of the onset of pain and was reconfirmed in all cases with transesophageal echocardiography and/or magnetic resonance imaging. Patients whose initial symptoms had begun more than 48 hours before the initial onset of pain were excluded. The diagnosis of IMH was made in the presence of aortic wall thickening in circular or crescentic shape with central displacement of intimal calcifications and evidence of intramural accumulation of blood (ie, high attenuation value on unenhanced CT). Patients with an intimal flap were excluded.

Patients with type B IMH (limited to the aortic arch or descending aorta) and without complications or indication to an endovascular treatment after the first 24 hours (aortic rupture, penetrating aortic ulcer with depth to 30mm, aortic dissection with visceral ischemia or aortic diameter greater than 50 mm) were included in a prospective study to assess the clinical and morphological evolution of IMH and to examine all images compatible with intimal anomalies on the initial CT scan. The study was approved by our hospital ethics committee and informed consent was obtained for all patients.

Imaging parameters were as follows: 16-slice multidetector CT was performed with 16×1.5 mm collimation, table feed of 3.4 mm per rotation, tube rotation time of 420 ms, tube voltage of 120 kV, and reference tube current–

time product of 530 with tube modulation software (CareDose 4D; Siemens, Forchheim, Germany). For 64-slice dual-tube multidetector CT, 24×1.5 mm collimation, table feed of 3.4 mm per rotation, tube rotation time of 500 ms, tube voltage of 100 kV, and reference tube current time product of 530 with the same tube modulation were used.

The data were reconstructed with an effective slice thickness of 2 mm applied, with a reconstruction increment of 1 mm. Nonionic contrast material (120 mL iomeprol; Iomeron 400; Bracco–Byk Gulden, Konstanz, Germany) was injected into an antecubital vein at a flow rate of 4 mL/s.

Unenhanced and two-phase contrast material–enhanced helical scans of the whole aorta were obtained. For the first acquisition (arterial phase), automated detection of peak bolus enhancement in the aortic root was used for timing of the bolus, and a threshold of 100 HU was chosen for the start of the acquisition. Late acquisition (venous or delayed phase) was started 60 to 70 s after the initiation of contrast material injection.

The following intimal anomalies were considered separately (**Fig 1**). To detect intimal erosion, all breaks in the intimal contour with areas of contrast medium within the haematoma were analyzed, whatever their size or the CT phase (arterial or venous). The indispensable criterion for diagnosis was intimal discontinuity on CT with no communication with the collateral artery from the aorta (**Fig 1a**). All patients with intimal lesions extending beyond the external border of the aortic wall (**Fig 1b**) were excluded because we considered that healing of the aorta was not possible and that these lesions already represented a complication. (This was also considered in cases of giant penetrating aortic ulcer.) In these cases, assessment of predictive factors is useless.

Aortic branch artery lesions (**Fig 1c**) were seen as small collections of contrast medium in the hematoma and

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