

Long-Term Follow-Up of Aortic Intramural Hematoma Predictors of Outcome

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Background—Aortic intramural hematoma (IMH) evolves very dynamically in the short-term to regression, dissection, or aortic rupture. The aim of the present study was to assess the long-term clinical and morphological evolution of medically treated IMH.

Methods and Results—Fifty of 68 consecutive patients with aortic IMH monitored clinically and by imaging techniques at 3, 6, and 12 months and annually thereafter were prospectively studied. Mean follow-up was 45 ± 31 months. In the first 6 months, total IMH regression was observed in 14 and progression to aortic dissection in 18 patients; in 14 of these, the dissection was localized, and 12 later developed pseudoaneurysm. At the end of follow-up, the IMH had regressed completely without dilatation in 17 patients (34%), progressed to classical dissection in 6 (12%), evolved to fusiform aneurysm in 11 (22%), evolved to saccular aneurysm in 4 (8%), and evolved to pseudoaneurysm in 12 (24%). Evolution to dissection was related to echolucency ($P < 0.02$) and to longitudinal extension of IMH ($P < 0.01$). Multivariate analysis showed an independent association between regression and smaller maximum aortic diameter and between aneurysm formation and atherosclerotic ulcerated plaque and absence of echolucent areas in IMH.

Conclusions—The most frequent long-term evolution of IMH is to aortic aneurysm or pseudoaneurysm. Complete regression without changes in aorta size is observed in one third of cases, and progression to classical dissection is less common. A normal aortic diameter in the acute phase is the best predictor of IMH regression without complications, and absence of echolucent areas and atherosclerotic ulcerated plaque are associated with evolution to aortic aneurysm. (*Circulation*. 2003;108:583-589.)

Key Words: aorta ■ follow-up studies ■ imaging ■ magnetic resonance imaging ■ echocardiography

Intramural hematoma of the aorta (IMH) results either from spontaneous rupture of the vasavosorum of the aortic wall or from a penetrating atherosclerotic ulcer.^{1,2} Recent advances in imaging techniques have significantly improved the diagnosis and heightened clinical understanding of IMH, accounting for a frequency of 10% to 30% of all acute aortic syndromes.³⁻⁹ In its early phase, several studies have shown IMH to be a very dynamic process that leads to reabsorption, classical dissection, or aortic rupture.³⁻¹⁶ Recent studies using clinical and radiological findings identified prognostic markers associated with medium-term IMH progression or regression, with conflicting results.^{17,18} Therefore, additional studies using a wider range of imaging techniques are required to determine the predictive factors of long-term evolution of IMH.¹⁹

The aim of the present study was to assess prospectively the long-term clinical and morphological evolution of medically treated IMH using multiple imaging techniques in a series of consecutive patients from a single hospital surviving the acute phase of the disease.

Methods

Study Patients

From January 1990 to December 2000, 302 patients were prospectively identified at our center, a general referral hospital, as having acute nontraumatic aortic syndrome by at least 2 imaging techniques, transesophageal echocardiography (TEE) and MRI or contrast-enhanced computed tomography (CT), or anatomical findings at surgery or autopsy. The diagnosis of IMH was made according to previously established criteria.⁴⁻⁶ Cases with intimal flap were specifically excluded. In patients in whom noncommunicating aortic dissection had to be ruled out, the 3 imaging techniques were performed and the morphological sequential changes were assessed.

Clinical baseline findings included demographic data and cardiovascular risk factors. A patient was considered to have atherosclerosis whenever a history of ischemic heart disease, peripheral artery disease, or nonembolic cerebral vascular accident was documented.

Imaging Techniques

TEE and MRI or CT were performed in all patients during the acute phase. Echocardiographic examination was performed with a GE System Five or HP Sonos 1000 apparatus using a biplane probe up to 1995 and multiplane probe thereafter. Location, maximum thick-

Received December 16, 2002; de novo received March 4, 2003; revision received May 13, 2003; accepted May 14, 2003.

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DOI: 10.1161/01.CIR.0000081776.49923.5A

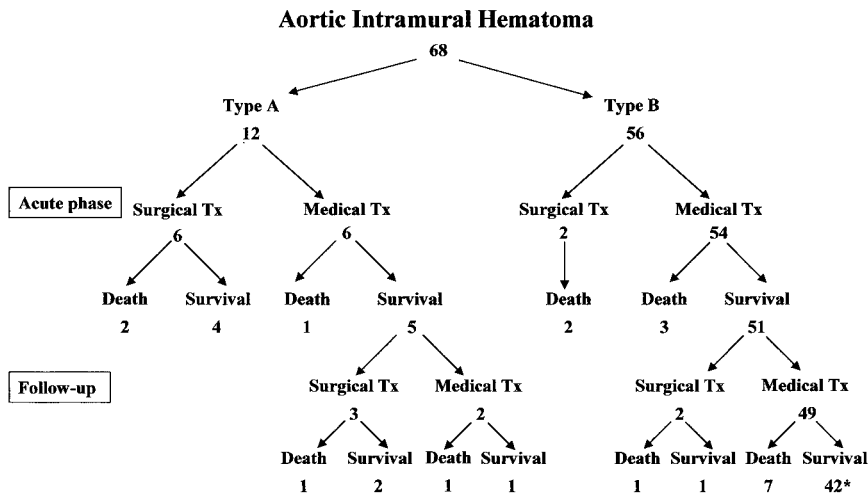


Figure 1. Evolution of 68 patients treated medically (medical Tx) or surgically (surgical Tx). *Six patients are survivors but were not included in the follow-up imaging technique protocol.

ening, circular or crescentic form, and echolucency of IMH were assessed by TEE as reported previously.^{5-7,20} Aortic atherosclerosis was defined by the presence of protruding fibrotic or calcified plaque and atherosclerotic ulcerated plaque when this plaque had a crater-like outpouching in the aorta wall with jagged edges.²¹ CT was performed with Twin Flash (Elscent) and Mx 8000 Quad (Philips) scanners and intravenous boluses with 80 to 150 mL of nonionic contrast medium. Helical CT was performed after administration of contrast material. MRI was performed with a superconducting MR 1.5T and 1.0T image (Magnetom Vision and Impac; Siemens). Transverse, oblique sagittal, and coronal imaging was obtained with gated ECG. T1-weighted spin-echo, HASTE sequence, and breath-hold gadolinium-enhanced rapid MR angiographic technique were performed in all studies. Maximum diameter of the aorta segment with IMH was measured by CT or MRI using transverse planes. When the aorta was tortuous, maximum diameter was measured by MRI choosing the diameter most perpendicular to the aorta wall on coronal or oblique sagittal views. If MRI was counterindicated, the measurement obtained by helical CT scan was used. IMH was defined as extensive when the longitudinal extension was greater than 15 cm by MRI or CT scan.

Follow-Up Protocol

Patients were followed-up both clinically (A.E. and A.S.) and by imaging techniques at 3, 6, and 12 months and annually thereafter. Clinical follow-up included strict systolic blood pressure control at ≤ 120 mm Hg and evaluation of the presence of chest pain and other complications. TEE and MRI or CT were performed at each control to determine maximum aortic diameter and morphological changes in the aorta wall.

The following 7 different patterns of evolution were considered: (1) regression, when IMH was totally reabsorbed on 2 imaging techniques, with neither aortic morphological changes nor increment higher than 10% of the basal aortic diameter; (2) classical dissection, when an intimal flap extending longitudinally and generating an aortic double lumen with flow in both lumina was observed; (3) localized dissection, when an intimal flap less than 50 mm with a single wide intimal tear with bidirectional flow was observed; (4) fusiform aneurysm, when the aorta was circularly dilated and its diameter increased $>10\%$ of the basal diameter, reaching a diameter >50 mm in ascending aorta, arch >45 mm in arch, and >40 mm in descending aorta; (5) saccular aneurysm, when a saccular dilatation >5 mm compared with the surrounding aortic lumen diameter developed; (6) pseudoaneurysm, when saccular dilatation developed from a localized dissection with disappearance of the intimal flap; and (7) persistence, when the IMH had not completely regressed or criteria of the previous groups were not fulfilled. Annual growth rate of the maximum aortic diameter was obtained from the difference in the diameter between initial and final measurements divided by the time elapsed between the 2 measurements.

Statistical Analysis

All values are expressed as mean \pm SD. Univariate analysis was performed on all clinical and morphological variables, with the χ^2 test and Fisher's exact test used for categorical variables and the Student's *t* test for continuous variables. Logistic regression models to identify independent predictors of final morphological outcomes and the development of severe complications were constructed. Variables showing a relationship with the outcome at the significance level of <0.1 in univariate methods were included in the model. A value of $P < 0.05$ was considered significant in all tests.

Results

Study Patients

Sixty-eight consecutive patients were diagnosed with IMH, 12 with type A and 56 with type B (Figure 1). Six type A and 2 type B patients were operated on during hospitalization because of complicated clinical course or indirect signs of aortic rupture. Eight patients died during hospitalization, 4 treated surgically (2 type A and 2 type B) and 4 treated medically (1 type A and 3 type B). Six of the 56 discharged medically treated patients were not included in the prospective follow-up protocol, 3 for residing far from our center, 2 for concomitant disease with high morbidity, and 1 for refusing to participate in the follow-up protocol. The patient cohort includes 50 of 68 consecutive patients; clinical characteristics and morphological baseline findings of IMH are shown in Table 1. No patient had a giant (discrete) ulcer image in acute phase. Patients were discharged with antihypertensive treatment.

The follow-up ranged from 2 to 114 months (mean, 45 ± 31 months; median, 43 months). No patient was lost to follow-up. Eleven patients presented with chest pain; in 6 the pain was consistent with an aortic origin and in the remaining 5 this was unclear. One patient had intermittent claudication, and 2 had renal failure. There were no other definite symptoms.

Type A

Two of the 5 surviving patients with an unoperated type A IMH underwent surgery within the first 3 months, 1 because of progression to type A dissection and 1 for progression to fusiform aneurysm after rebleeding in the aorta wall observed by MRI. IMH regressed within the first 3 months in 2 other

TABLE 1. Clinical Characteristics, Aortic Segments With IMH, and Baseline Morphological Findings (n=50)

Characteristic	No. (%)
Age, y	67±9
Men/women	45/5
Systemic hypertension	38 (76)
Smoking	26 (52)
Hyperlipidemia	12 (24)
Diabetes	7 (14)
Atherosclerotic disease	18 (36)
Abdominal aortic aneurysm	11 (22)
Aortic segments with IMH	
Descending thoracic aorta	46 (92)
Abdominal aorta	9 (18)
Aortic arch	7 (14)
Ascending aorta	5 (10)
Morphological findings	
Maximum diameter, mm	44.0±7.1
Maximum thickness, mm	13.4±4.1
Circular shape	16 (32)
Extensive	37 (74)
Echolucent areas	25 (50)
Atherosclerosis plaque	16 (32)
Atherosclerotic ulcerated plaque	10 (20)

patients, 1 of whom died from noncardiovascular cause at 8 months. The fifth patient showed persistence of IMH; dissection developed after 13 months and surgery was performed.

Type B

Of the 45 type B patients, 1 was treated surgically and 1 received an endovascular prosthesis for large saccular and fusiform aneurysm development, respectively. Five patients

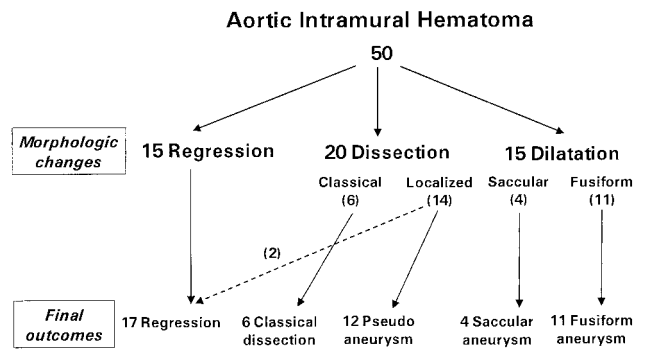


Figure 2. Different evolution patterns of IMH from morphological changes to final outcomes.

died suddenly, with aortic rupture being suspected, and 2 died from noncardiovascular causes (Table 2). Surgery or endovascular treatment was considered in 11 patients. Three were not accepted because of age (>75 years) and comorbidity, and 3 refused therapy. Five patients are now awaiting endovascular treatment.

Morphological Evolution

The IMH regressed (Figure 2) in 17 patients (34%). Regression occurred within 6 months in 14 of the 17 patients, in 2 of whom a previous localized dissection had been observed. With regression of the IMH, aortic diameter decreased in 9 cases (1 to 7 mm) and did not change or increased less than 1 mm in the remaining 8. Progression to dissection was observed in 20 patients (40%), with classical dissection in 6 (2 in ascending aorta and 4 in descending aorta) and localized dissection in descending aorta in 14. Eighteen of 20 dissections developed within 6 months. Twelve of these 14 localized dissections then evolved to pseudoaneurysm after an identical evolution pattern, as follows: localized dissection, progressive saccular dilatation, and disappearance of the intimal flap (Figure 3). Eleven patients (22%) showed pro-

TABLE 2. Characteristics of Patients With Severe Events During Follow-Up

Patients	Events	Type	Age, y	Interval, mo	Morphological Evolution	Diameter, mm	
						Basal	End Follow-Up
1	Sudden death	B	66	4	Fusiform aneurysm	54	61
2	Sudden death	B	70	12	Fusiform aneurysm	55	63
3	Sudden death	B	72	72	Fusiform aneurysm	50	67
4	Sudden death	B	40	60	Fusiform aneurysm	50	62
5	Sudden death	B	74	36	Type B dissection	46	70
6	Non-C death	B	79	72	Pseudoaneurysm	40	50
7	Non-CV death	B	75	36	Regression	35	34
8	Non-CV death	A	82	8	Regression	48	50
9	Death in surgery	B	65	84	Saccular aneurysm	50	94
10	Death in surgery	A	65	3	Fusiform aneurysm	52	64
11	Surgery	A	62	13	Type A dissection	61	70
12	Surgery	A	64	3	Type A dissection	54	60
13	Endovasc treat	B	62	65	Pseudoaneurysm	51	76

C indicates cardiac; CV, cardiovascular; and Endovasc treat, Endovascular treatment. Patient 6 died after stroke; patients 7 and 13 died of respiratory failure.

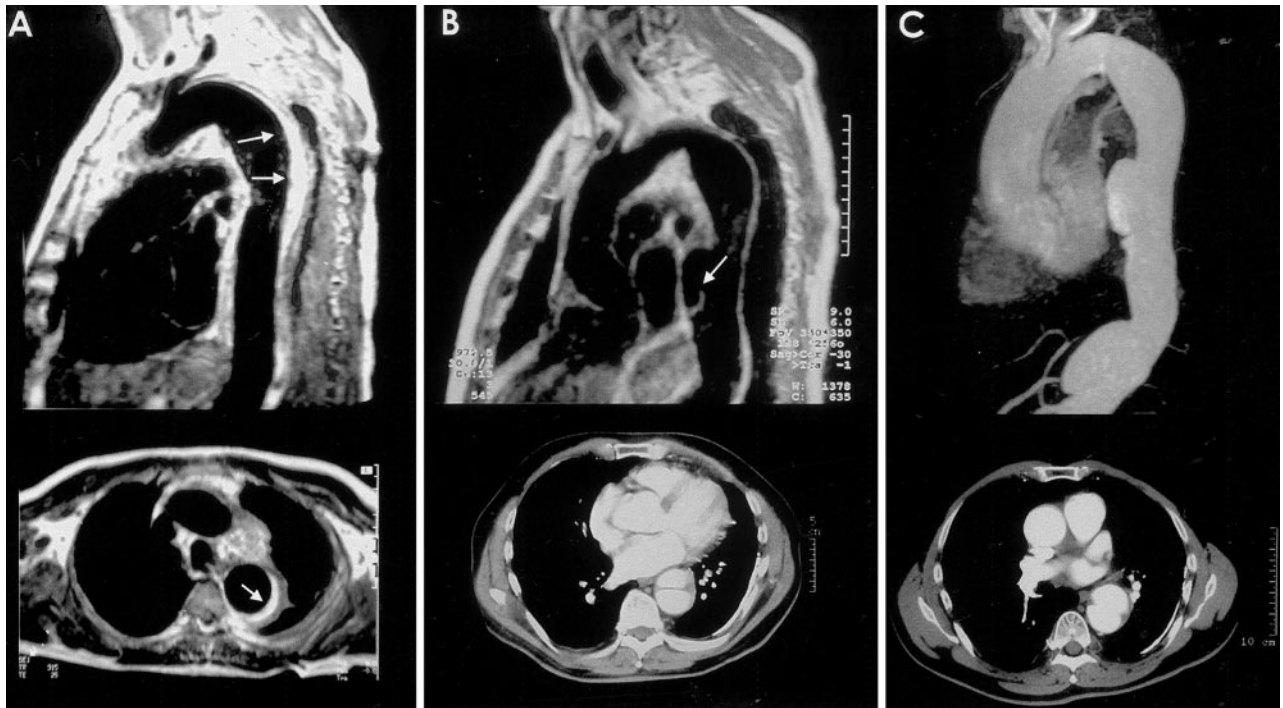


Figure 3. IMH evolving to pseudoaneurysm over a 4-year period. A, In the acute phase, MRI shows a high signal intensity suggestive of IMH. Top, Sagittal view demonstrates IMH in the upper and middle third of the descending thoracic aorta; bottom, IMH adopts a crescentic form in the axial view. B, At 6 months of follow-up, MRI in sagittal view (top) reveals a localized dissection; bottom, CT scan at this level displays typical dissection. C, At 4 years of follow-up, in sagittal view by gradient echo sequence (top), 2 pseudoaneurysm formations are observed, 1 in the site of localized dissection and another in abdominal aorta; bottom, CT scan shows that the intima has almost completely disappeared.

gressive aortic dilatation, eventually developing fusiform aneurysm (Figure 4). Another 4 cases of IMH (8%) with atherosclerotic ulcerated plaque evolved to saccular aneurysm; 1 of these required surgical treatment at 7 years of

follow-up (Figure 5). MRI showed signs of rebleeding in the aortic wall during follow-up in 3 asymptomatic patients, with 2 evolving to fusiform aneurysm and 1 to saccular aneurysm. The mean growth rate of the maximal aortic diameter was

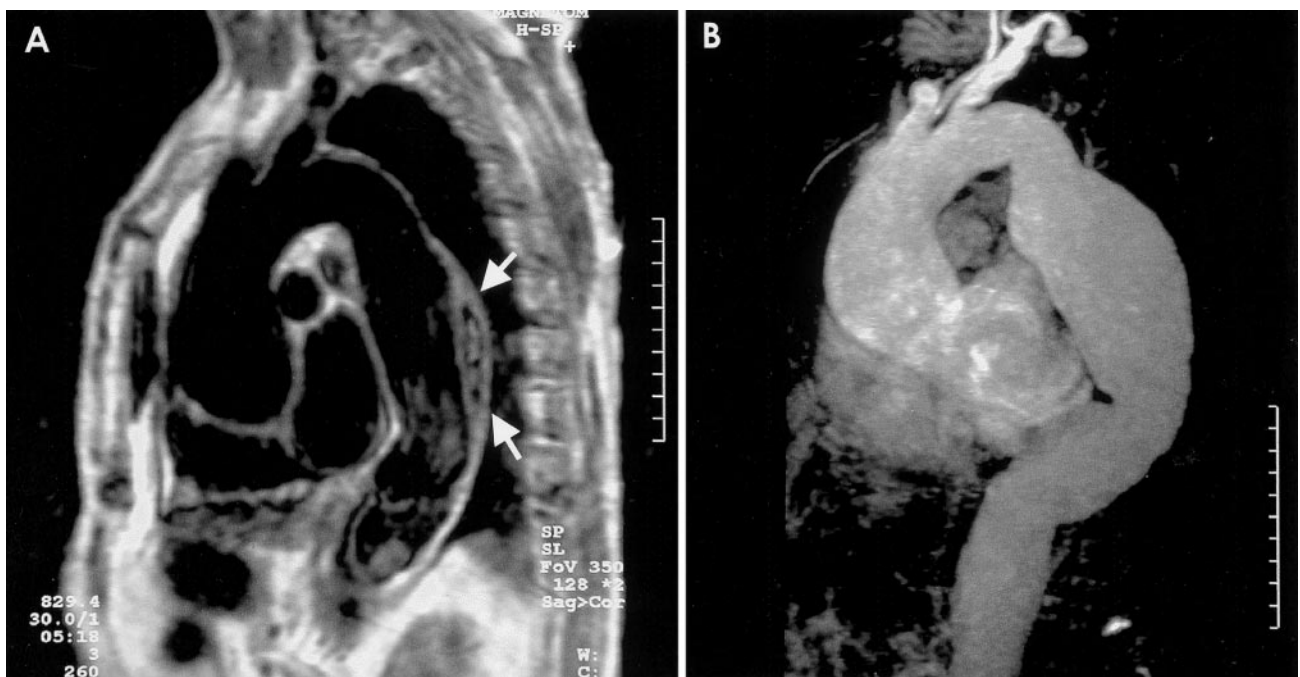


Figure 4. IMH evolution to fusiform aneurysm. A, MRI in sagittal view showing an IMH in the posterior aorta wall (arrows). B, Gradient-echo MRI confirming the evolution to fusiform aneurysm 5 years later.

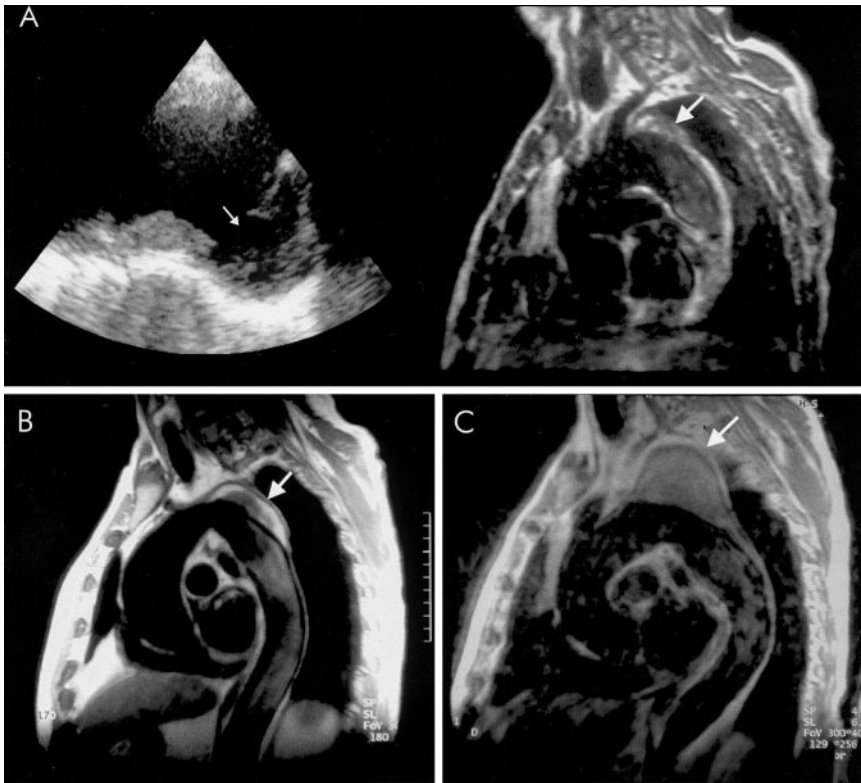


Figure 5. IMH evolution to saccular aneurysm. A, In acute phase, TEE (left) shows in transversal view an atherosclerotic ulcerated plaque (arrow) with IMH in the upper part of the descending aorta; MRI, T1-weighted, in sagittal view (right) reveals this ulceration (arrow) in the distal part of the aortic arch. B, MRI at 3 years reveals a high-density signal attributable to symptomless recurrent bleeding of the aortic wall. C, Seven years later, MRI shows a large saccular aneurysm (arrow) in the original site of the atherosclerotic ulcerated plaque.

significantly higher in the group that evolved to aneurysm formation (4.0 ± 5.4 mm) and classical dissection (4.9 ± 2.6 mm) than in the group with IMH regression (0.1 ± 1.7 mm, $P < 0.005$).

Prognostic Variables of IMH Evolution

The relationship between baseline morphological findings and evolution is listed in Table 3. Patients who showed regression of IMH had smaller aortic diameter (38.9 ± 4.5 versus 46.5 ± 6.9 mm, $P < 0.001$) and IMH thickening (11.8 ± 4.1 versus 14.1 ± 3.9 mm, $P < 0.05$) than the remaining group. The 11 patients who presented chest pain during follow-up did not evolve with regression ($P < 0.05$), 6 with aortic dissection and 5 with aneurysm formation. Variables

related to evolution to aortic dissection (classical or localized) were echolucency (14 of 18, 78%, versus 11 of 32, 34%; $P < 0.02$) and longitudinal extension of IMH (17 of 18, 94%, versus 20 of 32, 63%; $P < 0.01$). Twelve of the 14 cases with localized dissection evolved to aortic pseudoaneurysm formation. Patients who evolved to fusiform or saccular aneurysm more frequently had associated atherosclerotic disease in other sites (10 of 15, 67%, versus 8 of 35, 23%; $P < 0.05$), greater prevalence of atherosclerotic ulcerated plaque (7 of 15, 47%, versus 3 of 35, 9%; $P < 0.005$), and greater aortic diameter in acute phase (47.1 ± 5.8 versus 42.5 ± 7.3 mm, $P < 0.05$) than the rest.

Multivariate analysis only showed an independent association between regression and maximum aortic diameter in

TABLE 3. Relationship Between IMH Baseline Morphological Findings and Final Morphological Outcomes

Baseline Findings	Final Morphological Outcomes				
	Regression, n=17	Classical Dissection, n=6	Pseudoaneurysm, n=12	Aneurysm, Saccular, n=4	Aneurysm, Fusiform, n=11
Type A (%)	2 (40)	2 (40)	0	0	1 (20)
Type B (%)	15 (33)	4 (9)	12 (27)	4 (9)	10 (22)
Maximum diameter, mm	$38.9 \pm 4.5^*$	45.0 ± 5.7	46.2 ± 8.7	46.1 ± 5.6	$47.6 \pm 6.1^\dagger$
Maximum thickness, mm	$11.8 \pm 4.1^\dagger$	12.2 ± 3.6	14.4 ± 3.7	14.8 ± 3.0	14.3 ± 4.7
Echolucent areas (%)	7 (28)	5 (20)	10 (40)†	1 (4)	3 (12)
Extensive (%)	9 (24)	6 (16)	11 (30)	3 (8)	8 (22)
Circular shape (%)	5 (31)	1 (6)	6 (38)	1 (6)	3 (19)
Ulcerated plaque (%)	2 (20)	1 (10)	0	4 (40)*	3 (30)

* $P < 0.001$, † $P < 0.05$ for comparisons with the remaining values in each row.

acute phase (OR=0.7 [0.5 to 0.9]), with smaller diameter being an independent predictor of IMH regression. Furthermore, an independent association was also found between aneurysm formation and presence of atherosclerotic ulcerated plaque (OR=10.5 [1.3 to 85.3]), and absence of echolucent areas in the IMH (OR=0.13 [0.02 to 0.94]).

Predictive variables of severe complications, the group of patients previously described in Table 2, were maximum aortic diameter ($P<0.001$) and IMH thickness ($P<0.01$). Only basal aortic diameter was an independent predictor on multivariate study (OR=1.6 [1.1 to 2.4]).

Discussion

IMH has been increasingly recognized in acute aortic syndromes. In the present series, 22% of patients with acute aortic syndromes had IMH, a frequency consistent with that reported in other studies.^{6-9,12} Few studies exist on the predictors of medium- to long-term evolution patterns of IMH, and most of them are retrospective.^{17,18,22,23} Furthermore, the results of the 2 major studies,^{17,18} which used only clinical and radiological findings, show some disagreement. Our study represents a definite improvement on the previous information for the following reasons. First, it is prospective and was designed to investigate the morphological changes of IMH evolution. Second, we used a wider range of imaging techniques, TEE, MRI, or CT, in the acute phase and during follow-up. Third, to date, this is the longest prospective study on IMH with a low use rate of interventional procedures, which may reflect the natural evolution of medically treated aortic IMH patients surviving the acute phase.

IMH Evolution

Although controversy exists,^{4,13,24,25} the location of IMH may have strong prognostic significance, with type A having a higher rate of complications.^{4,13} Our results agree with this notion, but our number of patients with type A IMH is too small to draw definite conclusions. The present study shows that the most frequent morphological long-term evolution of IMH is to aortic aneurysm or pseudoaneurysm formation (54% of cases). Remarkably, regression without changes in aorta size or morphology is fairly common (34% in this series); on the other hand, progression to classic aortic dissection is less frequent (12%). Data on aneurysm formation after IMH stemmed only from retrospective series, and the frequency and progression of IMH were not defined.^{22,23} In agreement with findings reported in other series dealing with short-term IMH evolution,^{4-7,11,12,15} we observed IMH regression in 28% and aortic dissection in 36% of patients in the first 6 months of evolution. Nevertheless, it is noteworthy that most dissections were localized, which implied in most cases a different evolution pattern toward aortic pseudoaneurysm.

Prognostic Factors

Maximum aortic diameter in the acute phase is the variable of greatest prognostic yield for IMH regression, showing smaller maximum aortic diameter than the group that evolved to aorta aneurysm or dissection. These findings concur with those of previous studies, which reported similar results of

IMH regression or persistence in a short-term follow-up.^{12,16} Recently, Sueyoshi et al¹⁷ reported that an aortic diameter less than 40 mm and a thickness less than 10 mm identified the type B IMH regression group. By contrast, Ganaha et al¹⁸ suggested that the presence of penetrating atherosclerotic ulcer in acute phase was the factor that predicted progression or regression of IMH, with maximum aortic diameter lacking prognostic value. The frequency of penetrating atherosclerotic ulcer was strikingly high (52%). In this retrospective study, the baseline imaging technique was carried out within 2 weeks after the onset of symptoms, and 5 patients had a chronic IMH. Thus, localized dissections developing from an IMH could have been included as penetrating atherosclerotic ulcers.

In our study, with the use of TEE and MRI, we observed that most ulcer-like projections corresponded to localized dissections with a well-apparent intimal flap and a large single-entry tear. One of the most striking findings of the present study was that most localized dissections appeared within the first 3 months of evolution and developed progressive saccular dilatation with disappearance of the intima and subsequent pseudoaneurysm formation. In contrast to penetrating atherosclerotic ulcer, these ulcer-like images are not a cause of IMH but an early complication. On the other hand, the prognosis of these ulcer-like lesions is better than that of penetrating atherosclerotic ulcer,^{26,27} because some may show complete regression.^{17,23} Variables predicting progression to aortic dissection were echolucency and IMH extension. In a recent study by Song et al,²⁰ no prognostic significance was found between echo-free space detected in IMH and progression to dissection; however, in that study, only TEE was performed during a short follow-up, and therefore some localized dissections may have gone unnoticed.

Long-term evolution to fusiform aneurysm is related to greater aortic diameter in the acute phase, because structural weakness of the media and mechanical stress may favor fusiform dilatation.²² On the other hand, patients evolving to fusiform or saccular aneurysm had higher frequency of atherosclerotic disease and ulcerated plaque in the aorta, which points to the atherosclerotic process as an important cause of aneurysm formation. These findings suggest that IMH might be involved in the pathogenesis of chronic aneurysm. In patients with aortic atherosclerosis, some aorta wall hemorrhages may be symptomatic, as shown in the present series, and be diagnosed as IMH within acute aortic syndrome, whereas others might be asymptomatic with silent progression to aortic aneurysm. Remarkably, in the present series, 3 asymptomatic patients with aortic rebleeding during follow-up evolved to aneurysm formation.

Limitations

The number of patients with type A IMH in the present series is too small to adequately represent the long-term evolution of this subgroup, because 50% required surgical treatment during the acute phase owing to clinical complications. However, we did not exclude our type A cases, because our series included all medically treated IMH patients discharged after acute phase. In any event, our results with type A cases illustrate the complicated course of this IMH subgroup.

In summary, the most frequent long-term evolution of IMH is to aortic aneurysm or pseudoaneurysm. Complete regression without changes in aorta size or morphology is observed in only one third of cases in the first year of follow-up, and progression to classical dissection is less common. Evolution to aneurysm was more frequent in patients with associated atherosclerotic disease, atherosclerotic ulcerated plaques, and absence of echolucent areas in IMH, and progression to dissection was related to echolucency and greater IMH extension. A normal aortic diameter in acute phase was the best predictor of IMH regression without complications. The frequent evolution to aneurysm formation warrants a close long-term follow-up by imaging techniques when no IMH regression is observed in the first 6 months of follow-up.

Acknowledgments

The authors thank Aida Ribera for statistical analysis and Christine O'Hara for help with the English version of the manuscript.

References

- Gore I. Pathogenesis of dissecting aneurysm of the aorta. *Arch Pathol Lab Med.* 1952;53:142-153.
- Stanson AW, Welch TJ, Ehman RL, et al. A variant of aortic dissection: computer tomography and magnetic resonance findings. *Cardiovasc Imaging.* 1989;1:55-59.
- Yamada, Tada S, Harada J. Aortic dissection without intimal rupture: diagnosis with MR imaging and CT. *Radiology.* 1988;168:347-352.
- Nienaber CA, von Kodolitsch Y, Petersen B, et al. Intramural hemorrhage of the thoracic aorta: diagnostic and therapeutic implications. *Circulation.* 1995;92:1465-1472.
- Mohr-Kahaly S, Erbel R, Kearney P, et al. Aortic intramural hemorrhage visualized by transesophageal echocardiography: findings and prognostic implications. *J Am Coll Cardiol.* 1994;23:658-664.
- Harris KM, Braverman AC, Gutierrez FR, et al. Transesophageal echocardiography and clinical features of aortic intramural hematoma. *J Thorac Cardiovasc Surg.* 1997;114:619-626.
- Vilacosta I, San Roman JA, Ferreiros J, et al. Natural history and serial morphology of aortic intramural hematoma: a novel variant of aortic dissection. *Am Heart J.* 1997;134:495-507.
- Keren A, Kim CB, Hu BS, et al. Accuracy of biplane and multiplane transesophageal echocardiography in diagnosis of typical acute aortic dissection and intramural hematoma. *J Am Coll Cardiol.* 1996;28:627-636.
- Kang D-H, Song J-K, Song M-G, et al. Clinical and echocardiographic outcomes of aortic intramural hemorrhage compared with acute aortic dissection. *Am J Cardiol.* 1998;81:202-206.
- Robbins RC, McManus RP, Mitchell RS, et al. Management of patients with intramural hematoma of the thoracic aorta. *Circulation.* 1993;88(part 2):1-10.
- Moriyama Y, Yotsumoto G, Kuriwaki K, et al. Intramural hematoma of the thoracic aorta. *Eur J Cardiothorac Surg.* 1998;13:230-239.
- Nishigami K, Tsuchiya T, Shono H, et al. Disappearance of aortic intramural hematoma and its significance to the prognosis. *Circulation.* 2000;102(suppl III):III-243-III-247.
- Maraj R, Rerkpattanapipat P, Jacobs LE, et al. Meta-analysis of 143 reported cases of aortic intramural hematoma. *Am J Cardiol.* 2000;86:664-668.
- Choi SH, Choi S-J, Kim JH, et al. Useful CT findings for predicting the progression of aortic intramural hematoma to overt aortic dissection. *J Comput Assist Tomogr.* 2001;25:295-299.
- Song J-K, Kang D-H, Lim T-H, et al. Different remodeling of descending thoracic aorta after acute event in aortic intramural hemorrhage versus aortic dissection. *Am J Cardiol.* 1999;83:937-941.
- Kaji S, Nishigami K, Akasaka T, et al. Prediction of progression or regression of type A aortic intramural hematoma by computed tomography. *Circulation.* 1999;100(suppl II):II-281-II-286.
- Sueyoshi E, Imada T, Sakamoto I, et al. Analysis of predictive factors for progression of type B aortic intramural hematoma with computed tomography. *J Vasc Surg.* 2002;35:1179-1183.
- Ganaha G, Miller D, Sugimoto K, et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. *Circulation.* 2002;106:342-348.
- Nienaber C, Sievers H-H. Intramural hematoma in acute aortic syndromes: more than one variant of dissection? *Circulation.* 2002;106:284-285.
- Song J-M, Kang D-H, Song J-K, et al. Clinical significance of echo-free space detected by transesophageal echocardiography in patients with type B aortic intramural hematoma. *Am J Cardiol.* 2002;89:548-551.
- Vilacosta I, San Roman JA, Aragoncillo P, et al. Penetrating atherosclerotic aortic ulcer: documentation by transesophageal echocardiography. *J Am Coll Cardiol.* 1998;32:83-89.
- Ide K, Uchida H, Otsuji H, et al. Acute aortic dissection with intramural hematoma: possibility of transition to classic dissection or aneurysm. *J Thorac Imaging.* 1996;11:46-52.
- Sueyoshi E, Matsuoka Y, Sakamoto I, et al. Fate of intramural hematoma of the aorta: CT evaluation. *J Comput Assist Tomogr.* 1997;21:931-938.
- von Kodolitsch, Csosz SK, Koschyk DH, et al. Intramural hematoma of the aorta: predictors of progression to dissection and rupture. *Circulation.* 2003;107:1158-1163.
- Song J-K, Kim H-S, Kang D-H, et al. Different clinical features of aortic intramural hematoma versus dissection involving the ascending aorta. *J Am Coll Cardiol.* 2001;37:1604-1610.
- Harris JA, Bis KG, Glover JL, et al. Penetrating atherosclerotic ulcers of the aorta. *J Vasc Surg.* 1994;19:90-99.
- Quint LE, Williams DM, Francis IR. Ulcerlike lesions of the aorta: imaging features and natural history. *Radiology.* 2001;218:719-723.