Eur J Vasc Endovasc Surg (2019) 57, 350–359

Proposed Magnetic Resonance Imaging Criteria to Diagnose Intramural Haematoma and to Predict Aortic Healing after Acute Type B Aortic Syndrome

Adeline Schwein [a,b,*], Mohammad Khan [a,c], Matthew Bennett [a], Nabil Chakfé [b], Alan B. Lumsden [a], Jean Bismuth [a], Dipan J. Shah [a]

[a] Houston Methodist DeBakey Heart and Vascular Centre, Houston, TX, USA
[b] University Hospital of Strasbourg, Department of Vascular Surgery and Kidney Transplant, Strasbourg, France
[c] Flushing Hospital Medical Centre, Flushing, NY, USA

WHAT THIS PAPER ADDS

In this pilot study, three promising diagnostic magnetic resonance imaging (MRI) criteria were identified that may aid in the identification of intramural haematoma and seem to be associated with favourable aortic remodeling after acute type B aortic syndrome. These findings suggest an added benefit of diagnostic MRI for patients presenting with acute type B aortic syndrome as it might facilitate identification of a subgroup of patients likely to achieve aortic healing. Prospective validation of the MRI criteria is suggested.

Objective: Type B acute aortic syndrome (AAS) encompasses aortic dissection (AD) and intramural haematoma (IMH), the diagnosis, evolution, and treatment of which are subject to controversies. The aim of this pilot investigation was to assess the ability of specific magnetic resonance imaging (MRI) criteria to differentiate AD from IMH and predict optimal aortic remodeling following AAS.

Methods: In this retrospective study, all patients presenting between 2008 and 2015 with type B AAS, who had diagnostic MRI following admission, were included. Three MRI criteria were proposed to identify IMH: (i) no visualised entry tear; (ii) no contrast uptake in the aortic lesion on the first pass angiographic run; (iii) no contrast uptake in the aortic lesion on the equilibrium phase T1 sequence. On each patient's diagnostic and follow up imaging studies, the volume of (i) false lumen/IMH, (ii) total aorta, and (iii) true lumen were calculated. Using the Wilcoxon signed rank test, the evolution of these volumes according to the presence or absence of the aforementioned criteria were compared.

Results: Of 39 patients, in seven all MRI criteria were positive (group IMH) and 32 had one or more negative criteria (group AD). Patients with IMH and AD were similar with respect to sex, age, and delay between onset of symptoms and diagnostic and follow up imaging studies. Eighteen patients had a follow up imaging study after a mean period of 11.2 months: six in the IMH group and 12 in the AD group. Lesion volume decrease and relative true lumen volume increase were statistically significant in group IMH ($p = .046$ and $p = .046$, respectively), whereas there was a statistically significant increase of lesion volume ($p = .008$) in the AD group.

Conclusion: This pilot study proposed three simple MRI criteria to differentiate between AD and IMH. Once prospectively and clinically validated, this could have substantial therapeutic benefits as IMH are likely to heal spontaneously.

Keywords: Acute aortic syndrome, Aortic dissection, Aortic remodeling, Intramural haematoma, Magnetic resonance imaging
Article history: Received 23 February 2018, Accepted 12 September 2018, Available online 28 October 2018

INTRODUCTION

Acute intramural haematomas (IMH) and aortic dissections (AD) are the two most frequent pathologies belonging to the spectrum of acute aortic syndromes (AAS): life threatening diseases involving the thoracic aorta with similar clinical presentation (acute migrating chest or back pain and severe hypertension). Type B AAS do not involve the ascending aorta, have better short-term outcomes, and do not always require emergency surgical treatment, as opposed to type A AAS.[1,2] The goal of treatment for Type B IMH and AD, either medical or surgical, is to obtain favourable aortic remodelling, consisting of thrombosis or resorption of the haematoma or false lumen, and re-expansion of the true lumen.

* Corresponding author. University Hospital of Strasbourg, Department of Vascular Surgery and Kidney Transplant, 1 Place de l'Hôpital, 67091 Strasbourg, France.
E-mail address: adeline.schwein@gmail.com (Adeline Schwein).
1078-5884/
https://doi.org/10.1016/j.ejvs.2018.09.017

IMH was first described in 1920 as an "aortic dissection without intimal tear" on a postmortem examination.[3] Thanks to advances in *in vivo* imaging and pathophysiological understanding, many retrospective studies have been published in the literature in the past 15 years regarding the similarities and differences between IMH and AD, which have led to a large variety of opinions regarding their definition, natural history, and outcomes. This has ultimately resulted in many controversies regarding their diagnosis and management.[4–6] One general consensus is that patients should undergo regular imaging follow up to detect mid- and long-term complications.[7] The establishment of robust initial imaging criteria that would predict favourable long-term evolution is a key to the avoidance of unnecessarily aggressive management, unnecessary imaging follow up, and unnecessary expense.

Currently the diagnosis of IMH relies mostly on computed tomography (CT), which is usually preferred to magnetic resonance imaging (MRI) owing to its speed of acquisition and widespread availability. Nevertheless, it remains an irradiating and only static morphological imaging modality, which additionally requires the injection of nephrotoxic contrast agents in patients who may have acute renal insufficiency due to renal artery involvement. Furthermore, most contrast enhanced CT acquisitions for AAS are done during the first pass within a few seconds after injection (either arterial or venous phases) and may thus miss relevant late contrast uptake in the haematoma or false lumen.

MRI is a non-irradiating and dynamic diagnostic imaging tool considered as a leading technique for the diagnosis of AD, with a reported sensitivity and specificity of 98%.[8] While there are currently no robust described diagnostic imaging criteria that would identify and predict the long-term aortic evolution of acute type B AAS, either IMH or AD, it is believed that the additional features of MRI may allow new diagnostic definition and predictors of evolution.

The aim of this pilot study was to assess the ability of specific pre-specified diagnostic MRI criteria to differentiate acute type B IMH from AD and to preliminarily validate the differential diagnosis by the expected lesion resolution in patients with IMH during follow up.

MATERIALS AND METHODS

Patient selection

All patients admitted to the authors' institution and who underwent MRI for AD or IMH between December 2008 and May 2015 were reviewed. Inclusion criteria were type B aortic lesions according to the Stanford classification, and MRI performed within 14 days of symptom onset. Patient demographics, including sex, age, history of hypertension, dyslipidaemia, diabetes, smoking, coronary artery disease, renal function, and previous use of beta blockers or angiotensin converting enzyme inhibitors, were retrospectively recorded from medical records.

The study complied with the Declaration of Helsinki and was approved by the institutional review board.

Treatment

In the acute phase, patients received optimal medical treatment with regard to pain and blood pressure management.

Diagnostic MRI

Since 2008, a specific and systematic MRI protocol for clinically stable patients with type B AAS has been used, which includes the following: (i) first pass angiography done either with (a) time resolved magnetic resonance angiography (MRA) with a repetition time (TR) of 2.61 ms, TE of 0.94 ms, slice thickness of 1.40 mm, base resolution of 320, phase resolution of 100%, and voxel size of $1.4 \times 1.4 \times 1.4$ mm on the 1.5 T scanner; or, on the 3.0 T scanner, with a TR of 2.84 ms, TE of 1.02 ms, slice thickness of 2.00 mm, base resolution of 384, phase resolution of 84%, and voxel size of $1.4 \times 1.2 \times 2.0$ mm; or with (b) contrast enhanced three dimensional MRA using a gadolinium based contrast agent infused at the rate of 2.0 mL/s with the images acquired at a flip angle of 19°, TR of 2.34 ms, TE of 0.84 ms, slice thickness of 1.20 mm, base resolution of 416, phase resolution of 60%, and voxel size of $1.2 \times 1.2 \times 1.2$ mm on the 1.5 T scanner. On the 3.0 T scanner, the sequence used flip angle of 25°, TR of 2.91 ms, TE of 1.05 ms, slice thickness of 1.40 mm, base resolution of 384, phase resolution of 89%, and voxel size of $1.4 \times 1.2 \times 1.4$ mm. Of note, in patients with a contraindication to gadolinium based contrast agent (glomerular filtration rate < 30 mL/min/1.73 m^2, dialysis, or acute kidney disease), infusion of 90 mg ferumoxytol (Feraheme®)—a superparamagnetic iron oxide—was used to enhance vascular delineation after exclusion of iron overload by laboratory testing and MRI assessment of liver and cardiac T2-star, per the imaging laboratory's protocol. (ii) A long inversion time (TI) delayed enhancement (Ti 600) sequence was obtained approximately 10 min after intravenous gadolinium contrast administration using a TI set at 600 ms. The typical TE was 1.1 ms, with a voxel size of $2.0 \times 2.0 \times 6.0$ mm and a flip angle of 40°. Each image was acquired during mid-diastole within a single heartbeat. (iii) Three dimensional, T1 weighted gradient echo sequence (T1-VIBE) was acquired with TR of 4.44 ms, TE of 2.11 ms, slice thickness of 1.50 mm, base resolution of 288, phase resolution of 75%, flip angle of 10°, and voxel size of $1.3 \times 1.3 \times 1.5$ mm on the 1.5 T scanner. On the 3.0 T scanner images were acquired with TR of 3.89 ms, TE of 1.38 ms, slice thickness of 2.00 mm, base resolution of 320, phase resolution of 75%, and voxel size of $1.7 \times 1.3 \times 2.0$ mm.

The maximum aortic diameter, measured perpendicular to the centreline using the software Osirix (Pixmeo, Geneva, Switzerland) at the level of the ascending aorta, aortic arch, isthmus, and mid-descending and distal descending aorta were retrospectively recorded from the MRI report.

Each patient's diagnostic MRI was reviewed independently by a blinded investigator (AS) to assess the following three a priori established MRI criteria (Fig. 1): (i) the

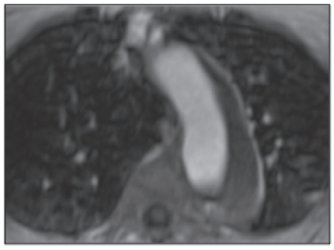
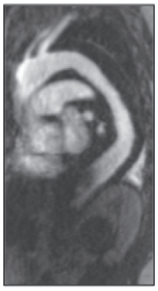
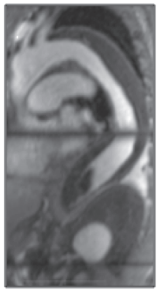
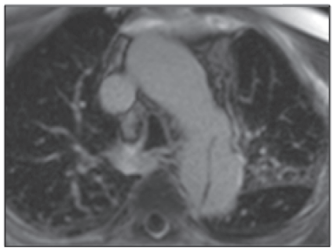
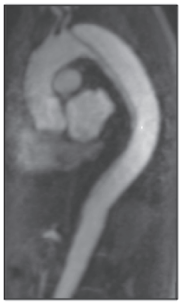
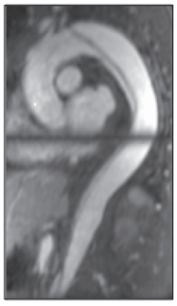
MRI criteria	Absence of aortic entry tear or communication between the true lumen and hematoma/false lumen on any of the MRI sequences	Absence of contrast uptake in the aortic wall on a first angiographic run	Absence of contrast uptake in the aortic wall on equilibrium T1 weighted sequence
Positive			
Negative			

Figure 1. Detailed representation of the three different magnetic resonance imaging (MRI) criteria used to define intramural haematoma.

absence of an aortic entry tear or communication between the true lumen and haematoma/false lumen on any of the MRI sequences; (ii) the absence of contrast uptake in the aortic wall on a first angiographic run; (iii) the absence of contrast uptake in the aortic wall on equilibrium T1 weighted sequence (T1-VIBE or Ti 600 sequence).

Patients were classified in two groups regarding the aforementioned MRI criteria: (i) the IMH group included patients with the presence of all MRI criteria; (ii) the AD group included patients with the absence of one or more MRI criteria

For patients included in the IMH group, the presence or absence of additional imaging criteria that were found to be predictors of complications in the literature, were recorded: maximum IMH thickness (measured perpendicular to the centreline using the software Osirix, including the aortic wall), ulcer like projections, intramural blood pools, and intimal irregularities.[9–13]

Aortic remodelling evaluation

The aortic remodelling for each patient was assessed by comparing aortic volumes between the diagnostic MRI and the last available follow up imaging modality (MRI or CT).

On both diagnostic and follow up imaging scans, the same region of interest was defined and thus the same aortic segment to be analysed for volume measurements was used. On axial views, the contour of the entire aorta and of the haematoma/false lumen was manually drawn by two of the authors (AS and MK) on each slide of the region of interest (slice thickness varying from 4 to 6 mm depending on the imaging modality and sequence). The entire aortic volume and the haematoma/false lumen volume were then computed automatically using the software Osirix (Fig. 2). The true lumen volume was calculated by subtracting the haematoma/false lumen volume from the whole aortic volume on both diagnostic and follow up imaging modalities.

Data analysis

Aortic remodelling was assessed by comparing the evolution of the true lumen volume, the haematoma/false lumen volume, and the total aortic volume in the two groups, using a Wilcoxon signed rank test.

RESULTS

One hundred and sixty-seven patients underwent an MRI for aortic dissection or intramural haematoma between December 2008 and May 2015. Of these, 85 had a type B lesion and 42 underwent a diagnostic MRI in the acute phase. Thirty-nine patients underwent all MRI sequences needed for criteria evaluation and were thus included in the analysis (Fig. 3). The median delay between the onset of symptoms and MRI acquisition was one day (range 0—7 days).

Regarding the presence or absence of the aforementioned diagnostic MRI criteria, seven patients (18%) were

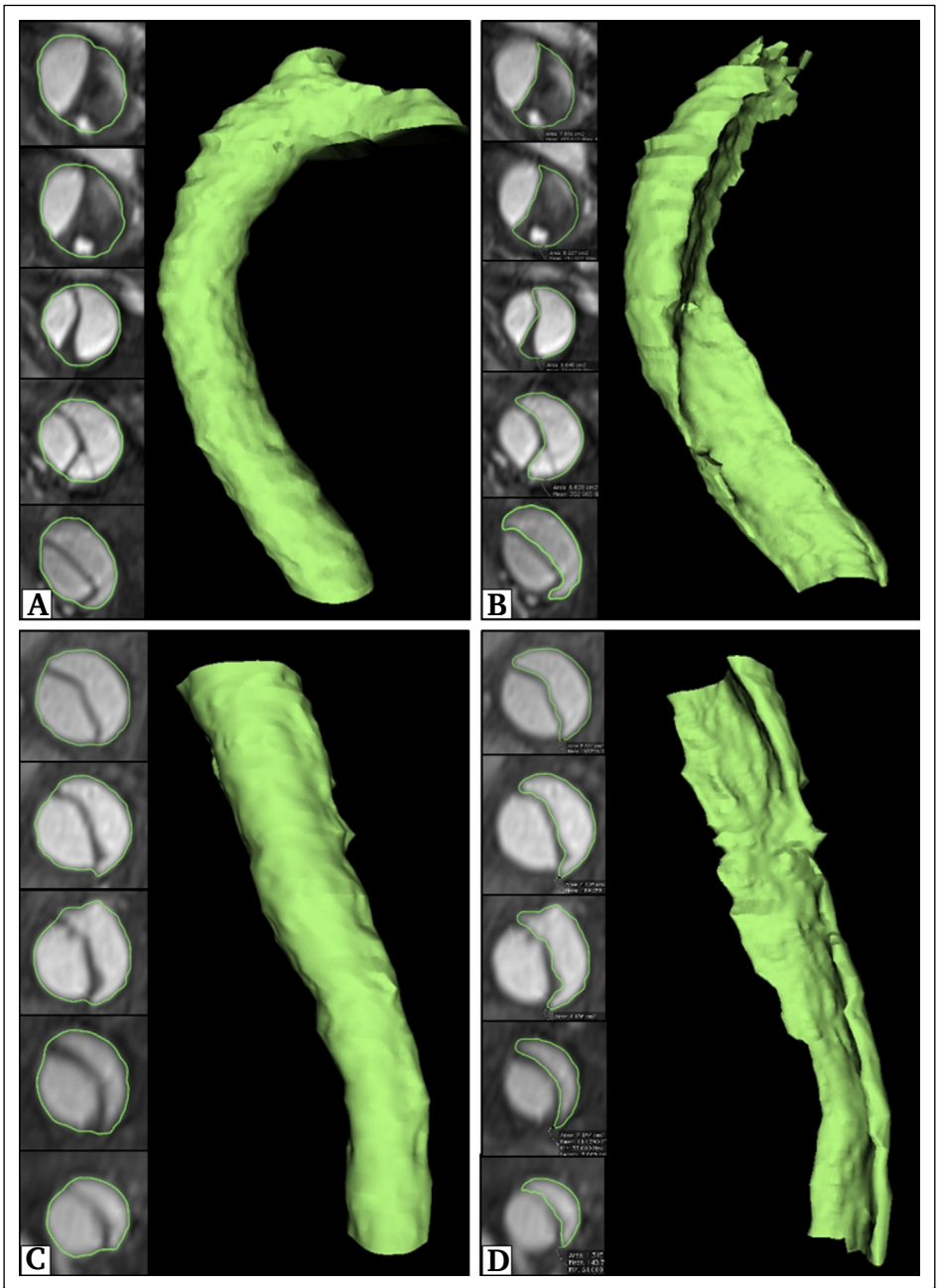


Figure 2. Axial view of the segmentation and three dimensional volume representation (Osirix) of the (A, C) whole thoracic aorta and (B, D) thoracic false lumen of a type B aortic dissection.

put into the IMH group and 32 (82%) into the AD group. Patients with IMH and AD were similar regarding patient demographics, except for the mean ± SD creatinine level, which was higher in the AD group (1.6 ± 0.27 and 1.0 ± 0.09, respectively; $p = .036$).

Patients with IMH and AD were also similar regarding the maximum aortic diameters at the level of the ascending aorta, arch, isthmus, and descending thoracic aorta. Detailed results regarding patient's demographics and diagnostic MRI analysis are summarised in Table 1.

Eighteen patients had follow up imaging performed: six patients in the IMH group and 12 patients in the AD group. Of the 21 patients for whom there was no follow up imaging study (one in the IMH group and 20 in the AD group), four patients died (all in the AD group), nine were lost of follow up (all in the AD group), and eight underwent thoracic endovascular aortic repair (TEVAR) in the acute phase (one in the IMH group and seven in the AD group). Indications for TEVAR were visceral ischaemia in two patients with AD, persistent severe chest pain in one patient with AD and one with IMH, haemothorax in two patients with AD, aortic diameter increase >7 cm diameter in one patient with AD, and acute limb ischaemia in one patient with AD. Patient demographics were similar for patients with imaging follow up and those without.

Mean ± SD follow up imaging was similar for the two groups (326 ± 109 days for the IMH group and 341 ± 152 days for the AD group; $p = .53$).

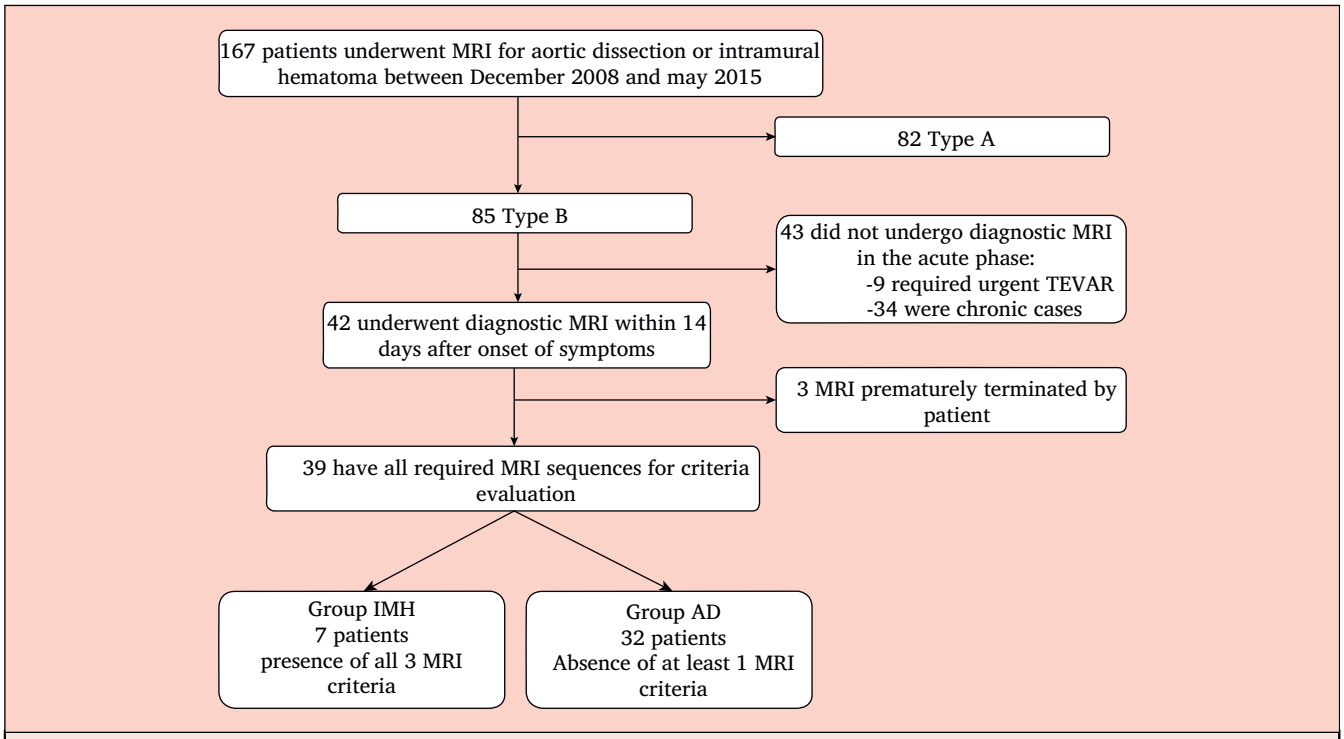


Figure 3. Inclusion criteria. MRI = magnetic resonance imaging; TEVAR = thoracic endovascular aortic repair; IMH = intramural haematoma; AD = aortic dissection.

In the IMH group, the evaluation of the two luminal volumes over time showed a significant *decrease* of the mean ± SD aortic lesion volume (93 ± 26 mm^3 on diagnostic imaging vs. 13 ± 11 mm^3 on follow up imaging; $p = .046$), a significant increase of the mean ± SD true lumen volume (170 ± 48 mm^3 vs. 263 ± 69 mm^3; $p = .046$) and no significant changes for the volume of the total aorta ($p = .46$) (Figs. 4 and 5).

In the AD group, results showed a significant increase of the mean ± SD aortic lesion volume over time (134 ± 17 mm^3 vs. 194 ± 32 mm^3 for the diagnostic and follow up imaging; $p = .008$), no significant changes in the true lumen volume ($p = .75$) and a significant *increase* in the mean ± SD volume of the total aorta (281 ± 35 mm^3 vs. 341 ± 47 mm^3; $p = .003$) (Figs. 4 and 5). Detailed results regarding aortic volumes remodelling are summarised in Table 2.

Regarding the assessment of the additional imaging criteria that were found in the literature to be predictors of complications of IMH, in seven patients of the IMH group, the mean ± SD maximum IMH thickness was 13.6 ± 1.78 mm (range 7.3–19.8 mm), mean ± SD maximal aortic diameter was 4.4 ± 0.49 cm (range 2.9–6.2 cm), four patients had intramural blood pools, and two patients had intimal irregularities on their diagnostic MRI.

DISCUSSION

The findings of this pilot study suggest that the combination of three specific diagnostic MRI criteria may differentiate acute type B IMH from AD, as patients assumed to have IMH showed the expected aortic remodeling over time, whereas patients with AD did not.

Despite an abundance of research and publications, controversies persist regarding the natural history of IMH, and even regarding its definition as a distinct pathology. Some groups believe all AD originate from an IMH;[4] others advocate the abolition of the IMH terminology as they believe all IMH are actually thrombosed AD.[5] The present results showing a different evolution between the two groups favour the belief that IMH and AD may actually be two distinct pathologies.

Despite rising awareness within the cardiovascular community, the imaging work up of IMH is inadequately defined. Based on clinical experience acquired over the years in cardiac MRI with regard to AAS imaging, the three MRI criteria that might identify IMH were selected a priori. The MRI protocol involves standard acquisitions that are available on every scanner, and require a mean total acquisition time of 30 min. The absence of an entry tear correlates with the initial definition of IMH. The assessment of contrast uptake in the haematoma or false lumen, on both first pass angiography and equilibrium phase sequences, allows for specific screening of low perfused regions and ensures the complete absence of perfusion of the aortic lesion in the IMH group.

The current definition of IMH mostly relies on diagnostic CT criteria.[1,7] The rate of evolution of IMH towards regression, stabilisation, or progression varies significantly in the literature, especially as a result of the large variety of study designs and the mix of type A and B IMH results. In recent systematic reviews, Mussa et al. reported a resolution rate of

Table 1. Patient demographics, magnetic resonance imaging delays, and initial aortic diameters

	IMH group (n = 7)	AD group (n = 32)	p-value
Age, years	60.6 ± 5.9	61.3 ± 2.7	0.9
Men	3 (43)	21 (66)	0.4
Hypertension	7 (100)	27 (84)	0.56
Dyslipidaemia	1 (14)	15 (47)	0.21
Diabetes	1 (14)	4 (13)	1
Smoking	3 (43)	9 (28)	0.65
CAD	2 (29)	4 (13)	0.29
Beta blocker use	4 (57)	14 (44)	0.68
ACE inhibitor use	3 (43)	10 (31)	0.67
Serum creatinine (mg/dL)	1.0 ± 0.09	1.6 ± 0.27	**0.036**
GFR (mL/min/1.73 m^2)	76.3 ± 6.12	70.4 ± 7.2	0.53
Median (range) delay from onset (d)	1 (0–7)	1 (0–7)	0.85
Time to follow up (d)	326 ± 109	341 ± 152	0.93
Diameter, ascending aorta (cm)	4.0 ± 0.29	3.7 ± 0.11	0.36
Diameter, aortic arch (cm)	3.3 ± 0.2	3.2 ± 0.12	0.63
Diameter, aortic isthmus (cm)	4.4 ± 0.49	4.1 ± 0.17	0.66
Diameter, mid-descending aorta (cm)	4.2 ± 0.42	3.8 ± 0.15	0.46
Diameter, distal descending aorta (cm)	3.5 ± 0.26	3.5 ± 0.12	0.91

Data are presented as absolute number (%) or mean ± standard deviation, unless indicated otherwise. Bold denotes significance. IMH = intramural haematoma; AD = aortic dissection; CAD = coronary artery disease; ACE = angiotensin converting enzyme; GFR = glomerular filtration rate.

10% with medical treatment and a progression rate towards aortic dissection between 16% and 47%,[14] whereas Evangelista et al., who focused on type B IMH, reported stabilisation or regression rates greater than progression rates at one year (58.5% vs. 48.7%).[9] Yet, knowing how the disease will evolve over time is the key factor that will influence initial management and surveillance protocols. One main limitation of the current IMH literature is the heterogeneity of their CT imaging datasets. Most studies are based on CT scans of various acquisition techniques and imaging qualities: variability of the region of interest, of slice thickness, and above all, variability of the delay between contrast injection and

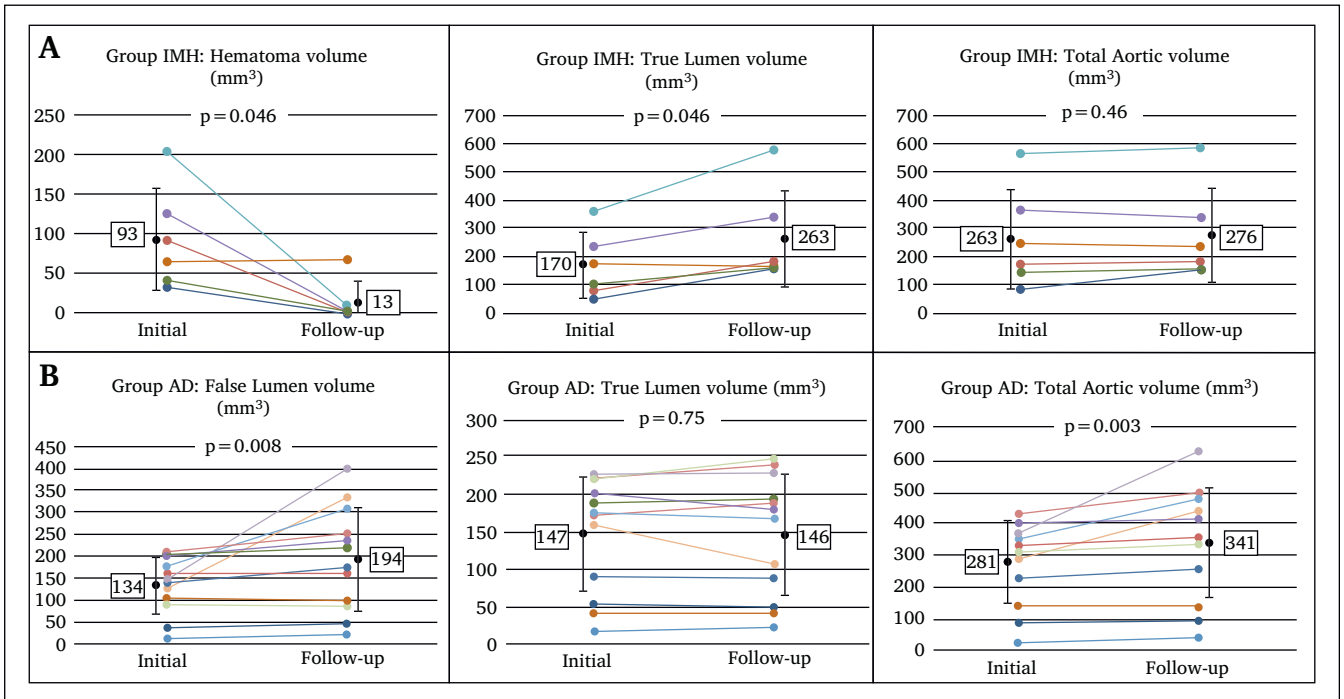


Figure 4. (A) Inverse relationship between true lumen and haematoma volume evolution over time in the intramural haematoma (IMH) group, showing significant true lumen volume increase, haematoma volume decrease, and stable whole aortic volume (B) For the aortic dissection (AD) group, significant false lumen volume increase and stable true lumen volume contribute to significant increase in the whole aortic volume over time.

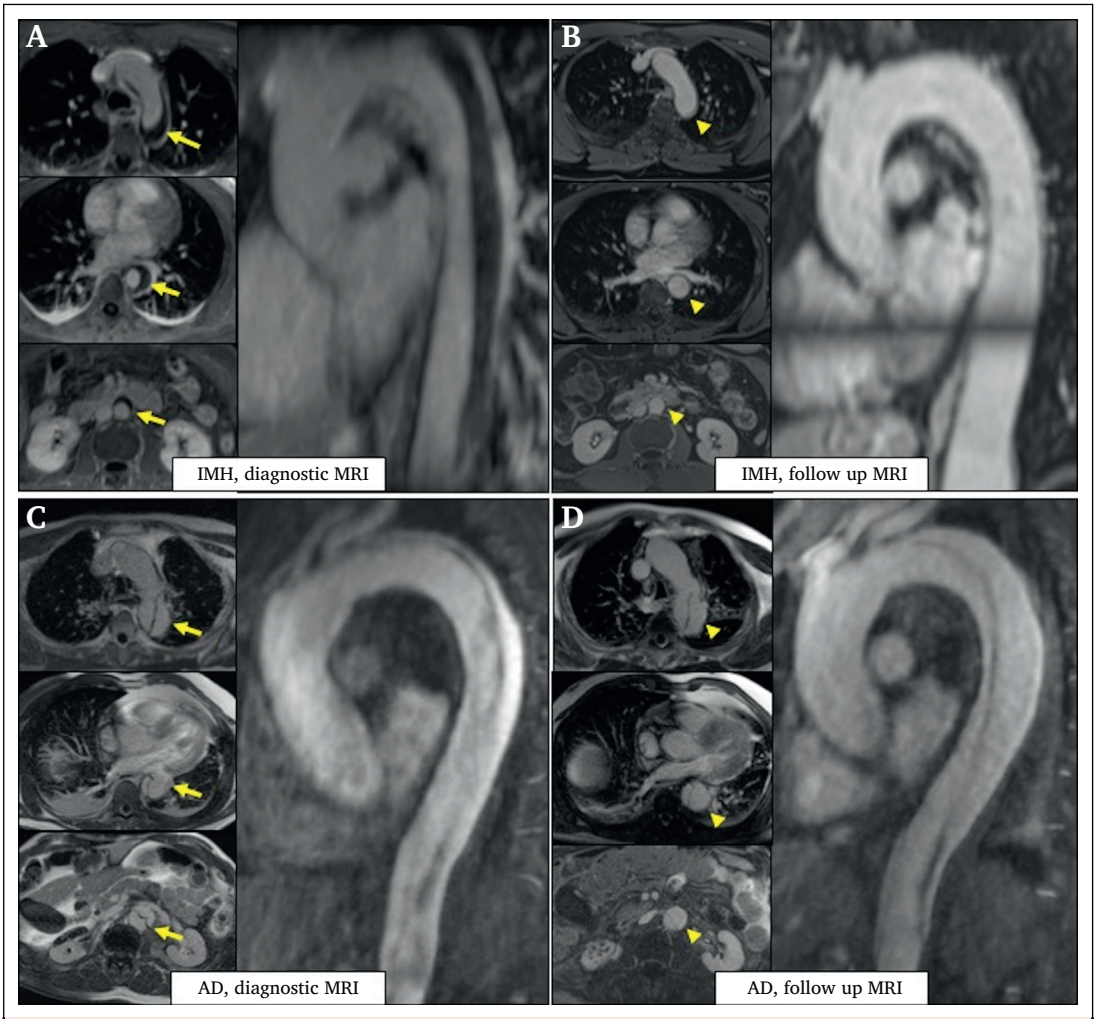


Figure 5. Diagnostic T1-VIBE sequence in a plane following the aortic arch, as well as three axial views at the level of the arch, mid-descending thoracic aorta, and visceral aorta showing the extent of (A) a type B intramural haematoma and (C) a type B aortic dissection, both in the acute phase (arrows). T1-VIBE sequence of the same patients, same locations on the last follow up imaging study, showing (B) optimum aortic remodelling: complete resorption of the intramural haematoma and re-expansion of the aortic lumen at each level and (D) the aortic dissection with persistent perfused false lumen and absence of aortic remodelling at each level (arrow heads). MRI = magnetic resonance imaging; IMH = intramural haematoma; AD = aortic dissection.

acquisition, which is believed to be a major reason that may lead to misappropriation between IMH and AD. Contrast enhanced CT scans are routinely done at an early arterial phase where the image acquisition is timed to the arrival of contrast in the proximal aorta. When studying AD, these first pass acquisition techniques have already been shown to overestimate the volume of thrombus in the false lumen when compared with the actual thrombus volume measured using equilibrium MRI sequences.[15] A low flow perfused false lumen resulting in late contrast uptake can thus be missed on initial CT and an aortic dissection may erroneously be diagnosed as IMH (Fig. 6). This misdiagnosis may explain why many authors report such high rates of progression of IMH towards AD. Late venous phase acquisition should add valuable information, however firstly, it is not performed routinely and not mentioned in the multi-society clinical guidelines in the diagnostic imaging workflow,[1,16] and secondly, the venous phase usually acquired 70 s after contrast injection may still miss low flow perfused areas, whereas the equilibrium MRI sequence acquired after 10 min will not.

In the literature, several imaging criteria have been established as “strong” predictors of IMH progression, and might thus be seen as indicators for aggressive treatment in the acute phase: (i) a maximum aortic diameter $>$ 40–45 mm in the acute phase is described as one of the major predictors of progression, especially towards aortic dissection;[9–11] (ii) a maximum aortic wall thickness $>$ 10 mm in the acute phase has also been described as a predictor of progression of IMH;[9–13] (iii) the presence of ulcer like projections, defined as localised blood filled pouches protruding into the haematoma with a wide communicating orifice of $>$3 mm, are also frequently described at risk factors for IMH progression.[9,12]

Table 2. Total aortic, true lumen, and intramural haematoma/false lumen volumes for 18 patients on both diagnostic and follow up imaging studies

Aortic pathology	Patient n°	Baseline MRI							Follow up imaging study				
		Interval since presentation (d)	MRI Criteria			TA volume	TL volume	IMH/FL volume	Imaging modality	Interval since baseline (d)	TA volume	TL volume	IMH/FL volume
			Entry tear	First pass CU	Equilibrium CU								
IMH													
	1	0	not vis	Absent	Absent	173.6	82.0	91.6	MRI	246	183.1	183.1	0.0
	2	1	not vis	Absent	Absent	365.1	239.4	125.7	MRI	401	337.6	337.6	0.0
	3	2	not vis	Absent	Absent	565.7	361.7	204.0	MRI	756	587.2	578.1	9.2
	4	0	not vis	Absent	Absent	83.7	52.4	31.4	MRI	115	153.9	153.9	0.0
	5	7	not vis	Absent	Absent	142.9	102.9	40.0	MRI	431	157.5	154.3	3.2
	6	1	not vis	Absent	Absent	245.5	179.9	65.6	MRI	6	235.7	168.1	67.6
Aortic Dissection													
	7	0	vis	Present	Present	28.4	13.8	14.5	MRI	4	41.9	21.0	20.8
	8	1	vis	Present	Present	331.6	172.1	159.5	MRI	10	351.8	187.8	164.0
	9	0	vis	Present	Present	88.8	53.1	35.6	MRI	630	95.0	48.8	46.2
	10	1	vis	Present	Present	403.1	190.0	213.1	MRI	380	412.7	195.0	217.7
	11	2	vis	Present	Present	401.8	201.5	200.2	MRI	10	416.9	179.6	237.3
	12	7	vis	Present	Present	142.3	39.6	102.7	CTA	227	138.6	40.7	97.9
	13	1	vis	Present	Present	354.3	176.8	177.5	MRI	384	477.3	168.7	308.6
	14	0	vis	Present	Present	430.4	221.2	209.2	MRI	14	495.7	241.4	254.2
	15	2	vis	Present	Present	289.8	160.4	129.4	MRI	1966	440.7	106.8	334.0
	16	2	vis	Present	Present	227.9	89.8	138.1	MRI	7	259.4	87.7	171.6
	17	7	vis	Present	Present	310.4	221.4	89.0	MRI	19	335.6	248.5	87.1
	18	0	vis	Present	Present	368.6	226.9	141.7	MRI	1113	628.2	229.2	399.0

MRI = Magnetic resonance imaging; IMH = intramural hematoma; CU = contrast uptake; not vis = not visualized; vis = visualized; CTA computed tomography angiography; TA volume = total aortic volume; TL volume = true lumen volume; FL volume = false lumen volume. All volumes are presented as mm^3.

Finally, the presence of intimal irregularities or defects and intramural blood pools, defined as a very small contrast uptake in the wall, without visible communication with the aortic lumen, have also both been recorded as specific imaging findings related to IMH, but their role in the occurrence of complications after IMH has not been clearly established.

In the present study, the presence of an intimal flap or communication between the true lumen and the haematoma or false lumen was a criterion for inclusion in the AD patient group. Thus, patients presenting with ulcer like projections on their diagnostic MRI were not diagnosed as having IMH.

In the authors' database, of the seven patients classified into the IMH group, five had a maximum aortic wall thickness between 12.2 and 19.8 mm, and four patients had a maximal aortic diameter >4 cm. Yet, comparison of aortic volumes between their diagnostic and follow up imaging datasets showed a significant decrease in haematoma volume and a significant increase of the true lumen volume, defined as optimal aortic remodelling; three patients actually achieved complete healing of the IMH on their follow up imaging. The results show that optimum aortic remodelling occurs after type B IMH, even in the presence of the previously described high predictors of progression, for example maximum aortic diameter >4 cm and IMH thickness >11 mm.

One danger of IMH management is to perform unnecessary and potentially harmful surgical procedures. Today's gold standard treatment for complicated IMH is the endovascular approach (TEVAR), owing to its lower short-term mortality and morbidity rates when compared with open repair. Nevertheless, in a recent retrospective study comparing endovascular treatment with best medical treatment, Bischoff et al. reported a 21.4% 30 day re-intervention rate after TEVAR vs. no invasive treatment at all in the medically treated group.[17] In addition to the usual possible post-operative complications following TEVAR (groin haematoma/infection, stroke, spinal cord ischaemia, iatrogenic dissection, or rupture of iliac arteries), one specific complication associated with IMH stenting is the occurrence of iatrogenic aortic dissection at the level of the stent graft landing zones. In order to avoid such outcomes, both proximal and distal landing zones should lie in healthy aortic tissue, resulting in an increased length of aortic coverage and thus an increased risk of spinal cord ischaemia. The ability of the three criteria described in the present study to identify IMH and predict favourable aortic remodelling may prevent unnecessary endovascular treatment and secondary complications.

These findings are in accordance with the current clinical practice guidelines of the European Society for Vascular Surgery (ESVS), which recommend the treatment of uncomplicated type B IMH medically with follow up imaging surveillance.[7] Moreover, as acknowledged by the ESVS guidelines, discrimination between IMH and AD may be difficult, and the role of endovascular repair is debatable, with critical identification of appropriate indications for treatment. This paper aimed to further explore the imaging workup of IMH, focusing on MRI capabilities, and to help fill the current knowledge gap regarding IMH identification and management.

The main limitation of the study is the lack of comparison to a reference diagnostic standard. For future research, the authors propose following patients prospectively with optimised CTA and MRI protocols, allowing for an

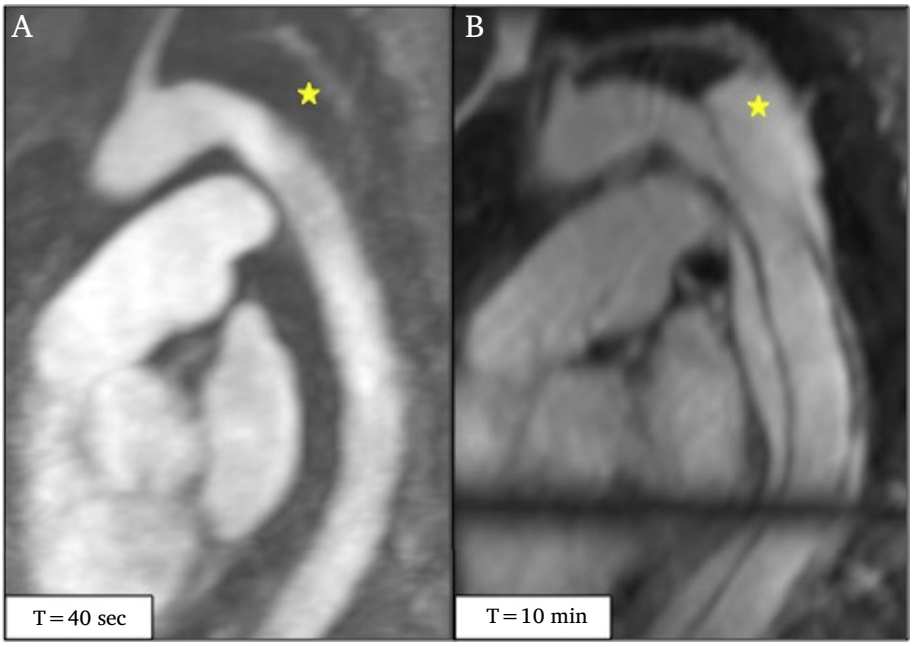


Figure 6. (A) First angiographic run sequence, acquired 40 s after contrast injection, showing the absence of contrast uptake within the haematoma/false lumen (star) (B) T1-VIBE equilibrium phase sequence of the same patient, acquired 10 min after contrast injection, showing contrast uptake in the false lumen (star). Based on the analysis of the first angiographic run (either magnetic resonance imaging or computed tomography), this aortic dissection would have been misdiagnosed as intramural haematoma.

exhaustive comparison. A one year follow up seems to be sufficient as different evolution patterns were found after a mean follow up of 11.2 months. The retrospective design of the study is a second limitation, which is also the case for all current literature on the subject. In addition, the number of patients in each group is small. However, as opposed to current literature's weakness with regard to the heterogeneity of CT imaging datasets, the systematic MRI protocol, gathering static, dynamic, early phase, and equilibrium phase acquisitions, guarantees the homogeneity of the studied imaging datasets and strengthens the results. Finally, of the 39 initial patients, only 18 had follow up imaging at the authors' institution. Nevertheless, of the 21 patients that were lost to follow up, only one was classified as having an IMH with regard to the initial criteria. Therefore, it is very unlikely that additional data would have changed the principle finding, namely that patients identified as having an IMH by the established MRI criteria demonstrate a high probability of healing.

CONCLUSION

This pilot study proposed three simple MRI criteria to differentiate between AD and IMH. The results show the value of diagnostic MRI for patients with type B AAS and encourage broader interest and use of this imaging modality, as it may have an impact on therapeutic decisions and imaging follow up protocols, as IMH are likely to heal spontaneously. These results will need to be substantiated in larger prospective studies before they can be validated and applied to clinical practice. Once prospectively and clinically validated, this could have substantial therapeutic benefits as IMH are likely to heal spontaneously.

CONFLICT OF INTEREST

None.

FUNDING

None.

REFERENCES

1 Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. *Eur Heart J* 2014;**35**:2873–926.
2 Evangelista A, Mukherjee D, Mehta RH, O'Gara PT, Fattori R, Cooper JV, et al. Acute intramural hematoma of the aorta: a mystery in evolution. *Circulation* 2005;**111**:1063–70.
3 Krukenberg E. Beiträge zur Frage des Aneurysma dissecans. *Beitr Pathol Anat Allg Pathol* 1920;**67**:329–51.
4 Tsai TT. Acute aortic syndromes. *Circulation* 2005;**112**:3802–13.
5 Uchida K, Imoto K, Karube N, Minami T, Cho T, Goda M, et al. Intramural haematoma should be referred to as thrombosed-type aortic dissection. *Eur J Cardiothorac Surg* 2013;**44**:366–9.
6 Song J-K, Yim JH, Ahn J-M, Kim D-H, Kang J-W, Lee TY, et al. Outcomes of patients with acute type a aortic intramural hematoma. *Circulation* 2009;**120**:2046–52.
7 Riambau V, Böckler D, Brunkwall J, Cao P, Chiesa R, Coppi G, et al. Editor's choice – management of descending thoracic aorta diseases: clinical practice guidelines of the European society for vascular Surgery (ESVS). *Eur J Vasc Endovasc Surg* 2017;**53**:4–52.

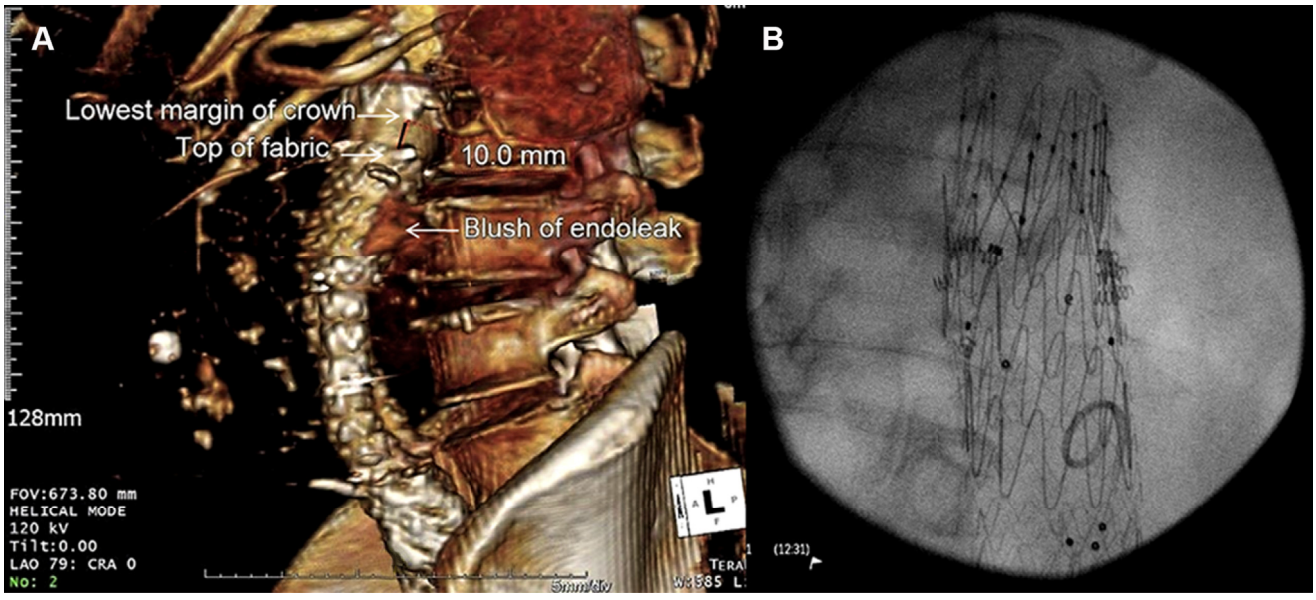
8 Nienaber CA, Von Kdolitsch Y, Nicolas V, Siglow V, Piepho A, Brockhoff C, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993;**328**:1–9.
9 Evangelista A, Czerny M, Nienaber C, Schepens M, Rousseau H, Cao P, et al. Interdisciplinary expert consensus on management of type B intramural haematoma and penetrating aortic ulcer. *Eur J Cardiothorac Surg* 2015;**47**:209–17.
10 Sueyoshi E, Imada T, Sakamoto I, Matsuoka Y, Hayashi K. Analysis of predictive factors for progression of type B aortic intramural hematoma with computed tomography. *J Vasc Surg* 2002;**35**:1179–83.
11 Gutschow SE, Walker CM, Martínez-Jiménez S, Rosado-de-Christenson ML, Stowell J, Kunin JR. Emerging concepts in intramural hematoma imaging. *Radiographics* 2016;**36**:660–74.
12 Lee YK, Seo JB, Jang YM, Do KH, Kim SS, Lee JS, et al. Acute and chronic complications of aortic intramural hematoma on follow-up computed tomography: incidence and predictor analysis. *J Comput Assist Tomogr* 2007;**31**:435–40.
13 Song J-M, Kim H-S, Song J-K, Kang D-H, Hong M-K, Kim J-J, et al. Usefulness of the initial noninvasive imaging study to predict the adverse outcomes in the medical treatment of acute type A aortic intramural hematoma. *Circulation* 2003;**108**(Suppl):II324–8.
14 Mussa FF, Horton JD, Moridzadeh R, Nicholson J, Trimarchi S, Eagle KA. Acute aortic dissection and intramural hematoma: a systematic review. *JAMA* 2016;**316**:754–63.
15 Clough RE, Hussain T, Uribe S, Greil GF, Razavi R, Taylor PR, et al. A new method for quantification of false lumen thrombosis in aortic dissection using magnetic resonance imaging and a blood pool contrast agent. *J Vasc Surg* 2011;**54**:1251–8.
16 Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, et al. Guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the American college of cardiology foundation/American heart association task force on practice guidelines. *Circulation* 2010;**121**:e266–369.
17 Bischoff MS, Meisenbacher K, Wehrmeister M, Böckler D, Kotelis D. Treatment indications for and outcome of endovascular repair of type B intramural aortic hematoma. *J Vasc Surg* 2016;**64**:1569–79.

Eur J Vasc Endovasc Surg (2019) 57, 359

COUP D'OEIL

Uneasy Lies the Graft that No Longer Wears the Crown

Arindam Chaudhuri
Bedfordshire- Milton Keynes Vascular Centre, Bedford Hospital NHS Trust, Kempston Road, Bedford MK42 9DJ, United Kingdom



An 81 year old male presented with a symptomatic abdominal aortic aneurysm after prior endovascular aneurysm repair in 2010 using an Endurant endograft (Medtronic, Santa Rosa, CA, USA). The aortic body had completely detached and migrated caudally from the suprarenal stent (panel A), resulting in a type Ia endoleak with sac size increase from 85mm to 115mm. Deployment of a 36 mm aortic cuff (Zenith, Cook Aortic Interventions, Bloomington, IN, USA) with additional reinforcement using 8 Heli-Fx EndoAnchors (Medtronic; panel B) re-established the proximal seal. The aneurysm remains stable without recurrent type Ia endoleakage beyond 12 months. This was reported to Medtronic/MHRA for investigation.

E-mail address: a.chaudhuri@ntlworld.com (Arindam Chaudhuri).
Twitter: @vascularis

https://doi.org/10.1016/j.ejvs.2018.12.009