Editorial

Type B intramural hematoma: thoracic endovascular aortic repair (TEVAR) or conservative approach?

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Introduction

An intramural hematoma (IMH) is the second most common presentation of acute aortic syndrome, with an estimated occurrence of 1.98/100,000 person-years (1). In the majority of cases, an IMH is located in the descending thoracic aorta. The clinical symptoms are similar to those of aortic dissection (AD), yet the population is older, the morphology different, and the mortality rate lower compared to AD. IMH was first described by Krukenberg in 1920 (2). Ever since, the pathophysiology of the disease is yet to be elucidated. We currently have only retrospective series available to describe the natural history. This is partly due to relatively low incidence of the disease itself. The onset of IMH has been associated with the occurrence of intimal micro-tears, vasa-vasorum hemorrhage and undiscovered penetrating atherosclerotic ulcers (PAU) (3,4).

Wolf in sheep's clothing?

Treatment of a type B IMH starts with medical management, by way of controlling blood pressure. Operative management is reserved for patients who present with complications such as frank rupture, visceral malperfusion, intractable pain, or other signs of impending aortic rupture. The majority of these complicated cases can be managed with thoracic endovascular aortic repair (TEVAR), with recent studies describing better outcomes compared to open surgical repair (5,6). The early-mortality rate of patients treated with open *vs.* endovascular repair has been shown to be 16% *vs.* 5% and the 3-year mortality rate was 23% *vs.* 7% (7).

Clinical decision-making for uncomplicated type B IMHs is more complex, as patients with uncomplicated IMHs have either complete regression during follow-up, or may develop adverse aortic remodeling or progression into classic AD. In a non-resolving IMH, most adverse events happen in the first year after onset (8). For now, the best radiographic predictor for adverse events in uncomplicated cases is the outer diameter of the aortic wall, similar to other acute aortic syndromes (9). IMH-specific predictors of adverse events include the presence of ulcer like projections (ULP) and local wall thickness >11 mm (*Figure 1*) (5,10,11). The latter predictor, however, was found in a study from 2003 that analyzed a cohort of 25 patients with type A IMH (10). The nomenclature in the literature may be confusing, as IMH and PAU can be considered points on a spectrum of intramural aortic pathologies. Originally PAU was described as a pathologic diagnosis (12). Contemporary clinical diagnosis is based upon computed tomography (CT) and therefore the term ulcer-like projection (ULP) better reflects radiographic diagnosis. For IMH treatment, it is important to understand the etiologic differences between ULPs. As described previously by Williams *et al.* the branch artery pseudoaneurysm may be benign and could be treated with optimal medical treatment (13) (*Figure 2*). Acute focal intimal disruptions, whether or not due to atherosclerotic ulcers, should be considered for TEVAR (14,15). Subacute or chronic focal intimal disruptions may be treated more conservatively (14). If CT quality allows, tiny intimal disruptions (<3 mm) should be distinguished from focal intimal disruptions (>3 mm), as the risk for adverse events differ significantly (16). An IMH with tiny intimal disruption should, however, be followed closely in the first year after onset as they may progress into focal intimal disruptions (16).

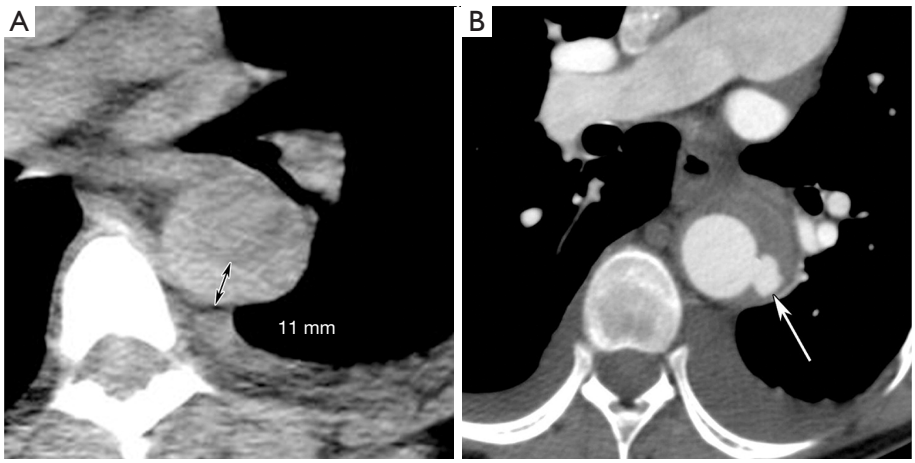

Figure 1 IMH predicting factors for adverse events. (A) Axial nonenhanced CT image shows an IMH thickness of 11 mm (double-headed arrow). The patient is at increased risk for progression and mortality. (B) Axial contrast-enhanced CT image shows an ulcer like projection (arrow) with localized contrast enhancement extending from the aortic lumen into the IMH and a visible communication. Reprinted with permission from Gutschow *et al.* (3). IMH, intramural hematoma; CT, computed tomography.

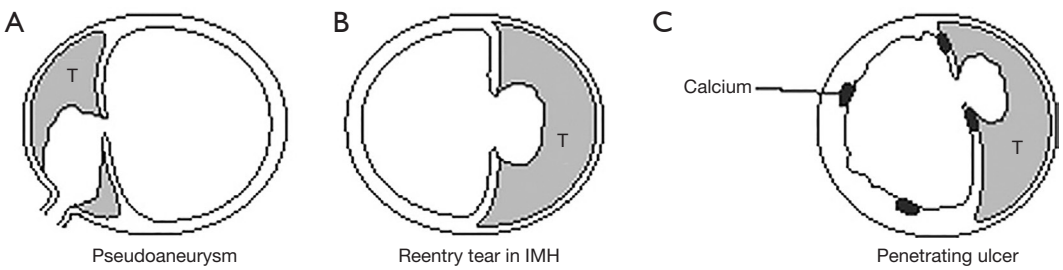

Figure 2 Schematic representation of aortic cross section in different pathologies. As these diagrams show, the radiographic signs of ulcer like projection can encompass three potential pathologic entities. (A) The first represents a typical branch artery pseudoaneurysm, the natural history of which is benign (T = thrombus). (B) The middle schema shows a re-entry tear developing in intramural hematoma (IMH), with local excavation of the mural thrombus. As the hematoma resolves, it can be left with an appearance of a penetrating ulcer. The diagnosis of entry tear would have been possible only if the original imaging study, in which the IMH was "fresh", was available. (C) The final schema represents the original PAU as described by Stanson *et al.* (12). Reprinted with permission from Williams *et al.* (13).

TEVAR challenges

Endovascular repair of type B IMHs pose challenges similar to those encountered when treating type B AD (17). Since the mechanical properties of the IMH change during follow-up, timing of TEVAR has implications on the environment in which the stent-graft is deployed. In the acute phase, the hematoma expands and the aortic lumen is compromised most. During follow-up, the hematoma thromboses and resolves, increasing the aortic lumen over time (18). This makes stent-graft sizing challenging. In the acute phase, the stent-graft can potentially be undersized, increasing the risk of stent-graft migration and type 1 endoleak. Measuring the total aortic radius including the IMH, on the other hand, comes with the risk of excessive oversizing and related complications. The elevated stress distributions across the wall may cause collapse of the stent-graft (*Figure 3*). Furthermore, apical bare metal stents and excessive balloon dilation can induce new entry-tears in friable aortic tissue (6,20). Therefore, if the aortic dimension is not increasing over time, delaying treatment is advised (15). After the subacute phase, the intimal layer stiffens and favorable aortic remodeling can be harder to achieve (21). In this phase, stent-graft sizing will rely more on the true lumen diameter, as the stiffened intima may not

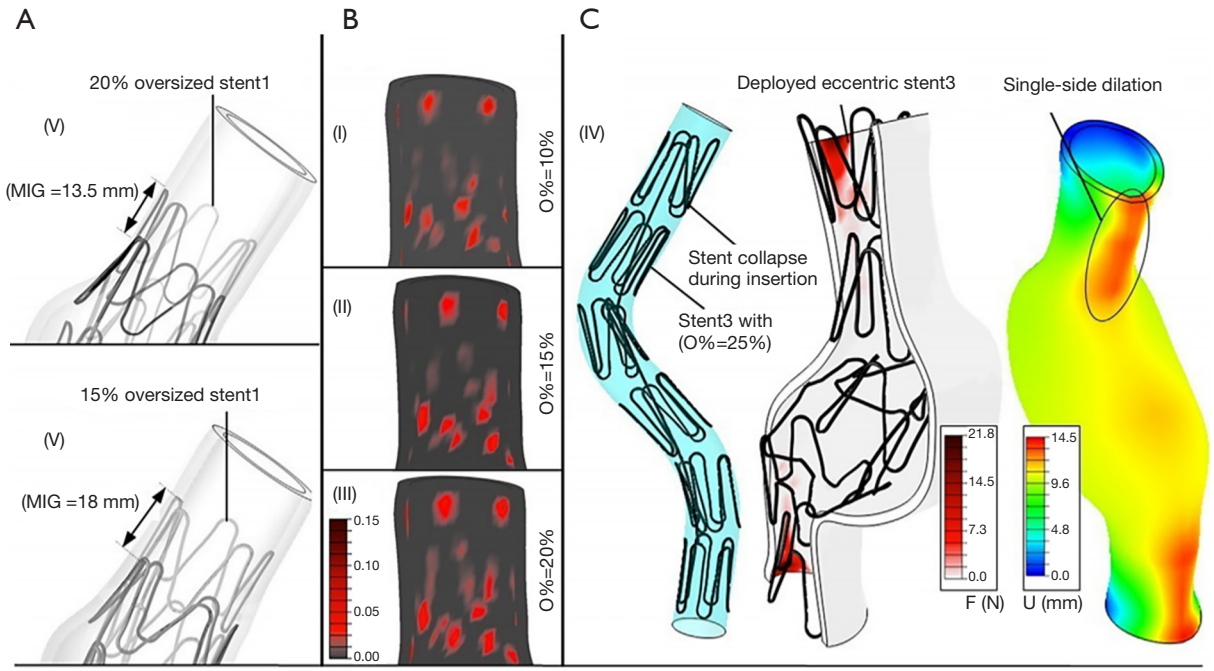

Figure 3 Schematic depiction of stent sizing and the associated forces calculated using a computational model. (A) Impact of 20% oversizing on proximal landing zone stent migration; (B) contact pressure stresses (MPa) in the proximal landing zone for different percentages of oversizing; (C) excessive oversizing with stent-graft collapse and increased dilatation (U) in the landing zones. Reprinted with permission from Altnji *et al.* (19). MIG, migration; O%, percentage of oversizing; F, force (Newton); U, displacement (millimeter).

allow much radial oversizing.

The adverse aortic remodeling, as seen in older patients, can result in angulation and tortuosity of the diseased aorta, which increases hemodynamic displacement forces acting on the surface of the stent-graft (22). In this anatomic configuration, an extended length of repair is often necessary to achieve appropriate sealing in the proximal and distal landing zones (22,23). Extended repair also increases the risks of stroke, spinal cord ischemia and type 3 endoleak at the intermodular junctions (24,25). Finally, current stent-grafts for TEVAR are 50–200 times stiffer than healthy aortic tissue (26,27). This increased stiffness of the aorta following stent-graft deployment can increase stresses in the untreated segments of the aortic wall (28). Furthermore, left ventricular stroke work is estimated to increase up to 26% as a consequence of stent-graft induced descending aortic stiffening, resulting in adverse left ventricular remodeling (29).

Conclusions

Uncomplicated type B IMHs are optimally treated with medical management. TEVAR is reserved for patients with complications and requires an experienced surgeon who can identify patient-specific challenges that increase the risk of the procedure. Factors such as the presence of ULPs should be taken into consideration for the decision to operate and reduce the long-term risk of aortic rupture.

However, large prospective or retrospective cohort data, generating IMH-specific outcomes are lacking. Contemporary decision-making is based on: (I) retrospective cohorts from >10 years ago, comprising no more than 66 patients with isolated IMHs; (II) clinical experience; and (III) acute aortic syndrome reports with a majority describing classic double barrel ADs.

Further research is necessary and should focus on providing a stratified risk model coupled with a natural history assessment in a large cohort and should be contrasted to that seen with classic AD.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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