The primary entry tear location in acute type B aortic dissection as an adjunct in therapeutic decision-making

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In the management of type B acute aortic dissection, most recent benchmarking has been proposed by the investigation of Stent Grafts in Aortic Dissection (INSTEAD) trial [1]. In the case of uncomplicated dissection with optimal medical therapy alone, only ~22% of these particular patients had to undergo secondary intervention or surgery at 2 years. This indicates that, in a benchmark trial, 78% of patients did well without developing the need for secondary reinterventions. However, per definition, the INSTEAD trial deals with chronic dissection, since the time delay from event to randomization was ~6 weeks in average and >2 weeks in all patients. So, there is still a window of uncertainty about treatment modality in the early, acute period of dissection. For this early time frame, the data from the International Registry of Acute Aortic Dissection (IRAD) indicate that 24% of 515 patients with acute Type B dissection require emergent intervention for early complications within the acute phase of 14 days [2]. However, we still miss clear predictive parameters identifying patients at risk of a complicated course.

The merit of the manuscript by Weiss et al. [3] in this issue is the fact that they provide a potential tool for further classification of patients into a higher risk group in the acute phase of dissection. They report that a primary entry tear at the concavity of the aortic arch and short distance between the primary entry tear and the left subclavian artery are frequently associated with the development of acute complications. Most promising is the fact that the authors postulate that this discrimination can be done at the first, diagnostic imaging study. When the entry tear was located at the convexity of the aortic arch, subsequent complications, needing intervention, developed in 20% of the patients. In contrast, when entry tear was located at the concavity of the arch, an impressive 89% of the patients required acute intervention. This potentially important information is somewhat confused by the fact that in total 56% of all studied patients finally developed complicated acute dissection. Remarkably, under the assumption of comparable definitions of complications, this is twice the incidence when compared with IRAD and by this weakens the broader applicability of the given decisive algorithm in daily routine.

The pathophysiological background is that the supra-aortic branches on the convexity of the aortic arch serve as a natural anatomic barrier against retrograde propagation of the dissection process. A similar protective structure is missing at the concavity of the arch. In a porcine model of aortic dissection, this theory has been confirmed: when the entry was at the concavity of the aortic arch the retrograde propagation reached the ascending aorta in 16%, whereas the dissection stopped at the origin of the left subclavian artery when the entry was at the convexity of the aortic arch [4]. One might speculate, together with the authors, to deliberately go for an intervention in patients with an entry localized at the concavity of the aorta. Nevertheless, in the present paper, there remain two major concerns. First, if we start with endovascular intervention in otherwise uncomplicated Type B aortic dissections, in this specific high-risk group with an entry close to the left subclavian artery, we inevitably end up in the landing zones 1 or 2 of the aortic arch. This in turn carries a certain risk of stentgraft-induced retrograde type A aortic dissection [5]. For this particular mechanism, it will be a matter for future studies to show whether completely covered stentgrafts might reduce the risk of retrograde aortic dissection by avoiding bare springs. Secondly, the entry localization was analysed retrospectively when the patients were already classified as complicated or uncomplicated course, which carries the risk of bias in retrospective analysis. Despite a relatively large number of acute aortic syndromes referred to our interdisciplinary aortic centre and despite sophisticated imaging techniques, in many cases we are still uncertain about the exact localization of the primary entry tear. This decision, to accurately allocate the entry tear and, by this, guide acute therapeutic strategy, may become even more difficult in the setting of clinical routine for less-experienced radiologists/surgeons on call.

In conclusion, we appreciate the effort by Weiss et al. to identify the entry localization at the concavity of the aorta as a new risk factor for complication of acute type B aortic dissection. It initiates structured decision-making in the very early phase of a potentially devastating disease. Nevertheless, two questions have to be answered in the future. First, is prospective detection of the primary entry tear localization accurate and reproducible in broad clinical application? Secondly, is the risk of aggressive endovascular treatment of the distal aortic arch in otherwise uncomplicated dissections really justified?
REFERENCES


