Dr Czerny: What were your criteria for convexity and concavity? You didn't mention that

Dr Kitamura: We just retrospectively reviewed the CT scan. For the initial cases, it was approximately 2.5-mm slices, so we just reconstructed it.

Dr Czerny: Let's say, it's never 90 and 270 degrees. So you draw a line?

Dr Kitamura: Yes.

Dr Czerny: And everything above was convexity and everything below was concavity?

Dr Kitamura: Yes.

Dr J. Bavaria (Philadelphia, PA, USA): So regarding the fact that your outer or greater curvature had more of a problem with TEVAR, in that your TEVAR results were worse in your last slide than it was when the entry site was the inner curve; why? Is that just because you had a more difficult time covering the primary tear site? Did you have more type IA endoleaks? Why do you think the greater curve is more problematic?

Dr Kitamura: Well, for our previous study, the stent grafts used were mostly handmade ones rather than a commercially available one. So probably with

the latest generation of stent grafts the outcome would be better, but one needs a longer time to say anything for sure.

Dr M. Grimm (Innsbruck, Austria): How does this impact on your daily doings now? When you have a patient presenting with an uncomplicated type B dissection and the entry tear is in the inner curvature, you rather go for a conservative observation, and when the entry tear is at the outer curvature, you rather tend towards liberal TEVAR in these patients? Because this would be the message I understand out of this.

Dr Kitamura: Well, at the moment the indication for intervention is only the diameter because, looking into the data, it seemed that all the rupture cases had aortic expansion before rupture. So probably for outer curvature entry cases, we might as well check CT or MRI more often. If the patient develops symptoms, including indefinite ones, we might as well check CT or MRI to exclude rapid expansion. But the indication for intervention being a maximum aortic diameter of 55 to 60 mm would be reasonable for all cases, I think.

Dr Grimm: So rather close monitoring than more aggressive treatment? **Dr Kitamura:** Yes.

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Re: Impact of the entry site on late outcome in acute Stanford type B aortic dissection

Martin Czerny*

University Clinic for Cardiovascular Surgery, University Hospital Zurich, Zurich, Switzerland

* Corresponding author. University Clinic for Cardiovascular Surgery, University Hospital Zurich, Rämistrasse 100, CH-8091 Zurich, Switzerland. Tel: +41-442-551111; fax: +41-442-554467; e-mail: martin.czerny@usz.ch (M. Czerny).

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Acute type B aortic dissection is regarded as the 'nice' dissection as it is usually not a cardiac surgical emergency and the acute phase responds well to medical therapy in a large number of patients. Therefore, the term 'uncomplicated' was attributed to this group when compared with the 'complicated' ones presenting with malperfusion, rupture or retrograde type A aortic dissection. Primary TEVAR for patients presenting with complications has already been established and remains the treatment of choice in the vast majority of patients with complicated type B aortic dissection. However, 'uncomplicated' should not be confused with 'harmless' as many patients will develop the need for any kind of treatment—early or late. 'Uncomplicated' should be used as a synonym to 'currently not in need of intervention' and this may change sooner or later as shown by the present study [1].

Recently, a better understanding of the natural course of the disease has been gained and thereby a much better anticipation of the need for treatment is available. Every physician treating patients with acute type B aortic dissection knows the clinical scenario of an initially very stable patient being found dead soon afterwards. Recent research has identified a new sub-group at risk for early adverse events and thereby the location of the primary entry tear comes into the focus of attention. It was

demonstrated that patients with a primary entry tear located at the concavity of the distal aortic arch have a substantially higher incidence of primary present or secondary developing complications such as malperfusion or retrograde type A aortic dissection/type A intramural haematoma [2]. Also the distance of the primary entry tear to the left subclavian artery is decisive; the closer the distance, the higher is the likelihood of already having or developing complications [3]. Consequently, these patients qualify for 'early' therapy to close the primary entry tear, aimed at prevention of these complications.

Kitamura and his group now share with us their experience, identifying a primary entry tear at the convexity of the aortic arch (which is new) as well as aortic diameter (which is known) as independent predictors of 'late' adverse outcome. What is not reported in their study is the time from the onset of symptoms to the time of referral and diagnosis as patients with a primary entry tear at the concavity of the distal aortic arch may either never have reached a hospital as limiting complications such as severe malperfusion or retrograde type A aortic dissection might have already occurred and thereby a natural selection process or a triage process (retrograde type A directly to cardiac surgery) may have already taken place.

Each phase of the disease process in acute type B aortic dissection has its inherent complications as new onset of malperfusion or retrograde type A aortic dissection is rare after the first 2 weeks after the event. After the acute phase, the subacute and chronic phases may also challenge both patient and physician as diameter increase in the 'subacute' phase is frequently observed. However, there is still a lack of evidence to show that 'physiological' diameter increase is inherent to the disease process itself and that 'pathological' diameter increase warrants treatment to prevent rupture. There is ongoing research aimed at clarifying these questions. What is already known is that patients with an entry tear >10 mm or a false lumen diameter of >22 mm at the time of diagnosis are at an increased risk for aneurysm development and therefore may be considered for prophylactic TEVAR [3, 4].

In the chronic phase, regular visits to a dedicated aortic outpatient clinic taking care of clinical as well as imaging follow-up remain decisive as late aneurysmal dilatation remains a clinical reality in a certain percentage of patients, who have to be monitored and eventually treated appropriately, as was confirmed here.

In summary, the lessons from this study support the fact that uncomplicated type B aortic dissection should not be confused with 'harmless' type B aortic dissection and clinical alertness in each phase of the disease process is decisive. In many patients, it

is not the question whether complications develop, but rather when they develop. Although the incidence of severe malperfusion and retrograde type A aortic dissection decreases over time, there is an increasing incidence of aneurysmal degeneration.

Morphological predictors of complications such as the location of the primary entry tear, the distance to the left subclavian artery, initial aortic diameter and the size of the primary entry tear should be actively implemented into decision-making to either remain with medical therapy or to advance to treatment.

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