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References


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Discussion
Presenter: Rana-Armaghan Ahmad

Dr Bradley Leshnower (Atlanta, Ga). Thanks to Dr Kelly and the Association for the invitation to discuss this article. Great job, Armi, and thank you to Dr Yang for providing a well-written manuscript. Again, you guys are shedding light on this concept of a delayed central aortic repair and a vascular treatment of MPS followed by delayed aortic repair in selected MPS to improve outcomes. Certainly, I’ve adopted this slightly different approach for mesenteric malperfusion, but in this article, you address it with both visceral and iliac malperfusion. Essentially, what you’ve done is take a control group and compare it with an MPS group that you have converted to non-MPS and showed the safety of that and that there’s no difference in short- and long-term mortality reintervention, and slightly increased growth in the descending abdominal aorta, but fantastic work.

When I looked at the manuscript I extracted a couple of other important data points. This is another large series of type A dissections to Dr Girardi’s point that he eloquently made, only a 21% need for reintervention over the long term for distal reintervention, so that should be noted. Second, certainly, the delayed aortic repair concept is controversial. It’s always a big discussion when we talk about it. I want to point out that in your big algorithm, you did lose 10% of patients with aortic rupture when you delayed them, but that those patients likely would have died had you operated on them emergently, we’ll never know. I want to point out the safety of the delayed aortic approach, as Himanshu Patel actually put it the other day, that the Michigan group believes the bigger threat to life is the
MPS, not the risk of rupture. Although you showed that the mortality short term was about 5% in your MPS, you do have to consider those with aortic ruptures, it’s a strategy of an intent to treat, and when I did the numbers, the mortality was 33%. But again, if you’re dealing with mesenteric malperfusion, those patients have a 60% to 70% mortality, so it’s still better.

On to my questions. First, in your arch measurement, you showed the descending and abdominal group, but in the arch measurements, I think it’s skewed because 40% of your malperfusion patients actually received a zone 1, 2, or 3 arch. Did you think about just looking at the hemiarches? Because when you sew a completely circular anastomosis, we all know you have a better chance of closing the false lumen, which will reduce it. Did you look at just the hemiarches in each group and compare measurements?

Dr Leshnower. Okay. If the MPS was resolved, why did you add TEVARs to 10% of the MPS groups?

Dr Ahmad. Are you saying the patients who didn’t receive open aortic repair?

Dr Leshnower. No. In your data, in the MPS group, 10% of those, when I looked at the operative data, received frozen elephant trunks.

Dr Ahmad. Okay.

Dr Leshnower. What was the need for that?

Dr Ahmad. So, that was not to treat the MPS, but more so to treat the proximal tear and prevent downstream aortic pathology. Maybe Dr Yang can further clarify that.

Dr Leshnower. This gets into a bit of a deeper thought; 89% of your MPS cases had both aortic and branch vessel stenting. Now, when I do a TEVAR first for a mesenteric malperfusion, the malperfusion is usually dynamic, so a TEVAR will completely resolve it. But you’re saying in almost 90% of patients, you’re also having to do branch vessel stenting, which would mean it’s static. So can you comment on the reason why and the differences you perceive?

Dr Ahmad. So, with the Michigan Protocol, once they go to interventional radiology for that, the way it’s done is that Dr Williams, the interventional radiologist, measures the difference in the systolic blood pressure between the aorta and each branch vessel. If it’s greater than 15, then they perform stenting.

Dr Leshnower. The way I do it is I shoot a picture in the operating room, and if there’s robust filling, I don’t do the physiologic measurements, so that explains it. The last question gets to the technique. You’ve shown that the descending and abdominal aortic growth rates are higher in your fenestration and stenting group. In your article, you offer the hypothesis that this is likely related to the aortopathy or vasculopathy of the patient, so it’s inherent to the aorta. But remember, you’re creating a 16-mm fenestration, and when we look at, for instance, stenting chronic type Bs, retrograde false lumen perfusion is a big reason why aneurysms grow. So, I would submit it’s not inherent to the aorta. It’s inherent to your technique. You are creating a large tear in the aorta and increasing blood flow to that false lumen and probably increasing pressure. It’s just something to think about. It’s not necessarily a question, but it’s a difference in the technique, whereas if I do a stent graft, I’m covering tears and eliminating blood flow into the false lumen. But anyways, very good job.

Dr Bo Yang (Ann Arbor, Mich). Just a clarification. Number one is that the rupture, yes, when you observe those patients, there’s always risk of aortic rupture. The most rupture happened in the first decade in our circulation paper. It’s about 16% ruptured in the first decade. In the second decade, 4% of patients had rupture because we’re gaining experience on how to manage those patients to control the blood pressure. It’s really low, below 90, and on waking them up, well, you keep them in the intensive care unit tightly controlled, and intensive care units are more confident with those patients in the second decade. This is number one. Those patients, if we take them to the operating room, their mortality is probably 30% to 40% operative mortality. But yes, when we do this, there is a risk of rupture. We found out that limb malperfusion has more risk of rupture than mesenteric malperfusion. Number 2 is the tear. We cover the tear with TEVAR during the open repair because we see the tear in the proximal descending aorta and we cover it. That’s why it’s not for malperfusion per se. Number 3, the stenting of the branch vessels. David always measured the blood pressure of branch vessels. If it is 15 mm Hg lower than the ascending aorta, he will stent it just to prevent all of these things being malperfused. That’s why he does more.

Dr Marek Deja (Katowice, Poland). Maybe I missed it, but can you clarify what was the time frame? I mean, in the group that you stented or stent-grafted, between the presentation and the operation on the ascending part, how long was the delay and what do you guide the delay with? This is one question. And another, you excluded the patients in the group without stent-grafting who had malperfusion. Can you give us a hint of what was the fate of those patients who directly went to aortic surgery rather than performing stent-grafting on them?

Dr Ahmad. I can answer the first question, or I guess the second question first. For the patients who had MPS and were excluded, who didn’t have fenestration stenting, it was because they had cerebral, coronary, or visceral with
cardiac tamponade. We excluded them because it didn’t make sense to compare them directly because they were never corrected for MPS. Our point was that we take the MPS patients, return them to the same baseline as patients who didn’t have MPS, with fenestration and stenting, and then it’s a direct comparison. If we included those patients, then it’s not really analyzing the efficacy of fenestration and stenting before open repair.

Dr Yang. But the fate of those patients, that’s a good question. We do not have the data right now. We didn’t look into it, but we will. I don’t think the outcome is good, but we’ll look into it. Cerebral malperfusion and coronary malperfusion are treated the way of open aortic repair. I had a discussion before about this issue.

Dr Deja. And the delay of the—?

Dr Yang. Oh, delay, yeah. The delay.

Dr Deja. What was the strategy? How long do you wait?

Dr Yang. The median time used to be 4 days. Now, it’s about 1 to 2 days, where it will be more active. We operate those patients earlier now. The criteria for waiting for operation are if the acidosis is corrected, a shock is corrected, they are not on multiple pressors, acute respiratory distress syndrome is better, they’re not on 100% oxygen but instead just 50, and you think the patient can’t tolerate the cardiopulmonary bypass and hypothermia circulatory arrest, then we’ll take the patient to the operating room. Kidney function takes a long time to recover, so I don’t wait for renal failure to recover.

Dr Deja. So, on average, this is what, 2 days?

Dr Yang. Yes. Maybe it’s 1 to 2 days now. In the first decade, it was 4 days.