Take the bull by its horn: 'Prophylactic aortic intervention' in uncomplicated type B aortic dissection

Mohammed Idhrees[1]

[1]SRM Institutes for Medical Science Vadapalani

May 18, 2022

Take the bull by its horn: 'Prophylactic aortic intervention' in uncomplicated type B aortic dissection

Running title: Prophylactic intervention in uncomplicated TBAD

Dr. A. Mohammed Idhrees MCh, FIAS

ORCID ID : 0000-0001-5981-9705

Consultant,

Institute of Cardiac and Aortic Disorders (ICAD),

SRM Institutes for Medical Science (SIMS Hospital), Chennai.

Contact Address:

Dr. A. Mohammed Idhrees MCh, FAIS

Cardio Thoracic and Vascular Surgeon

Institute of Cardiac and Aortic Disorders

SRM Institutes for Medical Science (SIMS Hospitals)

Chennai, India – 600 026

Phone: +919962268787

E-Mail:a.m.idhrees@gmail.com

Key Words: Aortic dissection, false lumen, TEVAR, complicated

Uncomplicated Stanford type B aortic dissection (UnTBAD) constitutes more than two-thirds of the type B aortic dissection. The present recommended management of UnTBAD is optimum medical management. On the other hand, intervention is warranted in patients with complicated Stanford type B aortic dissection (CoTBAD). It is documented that nearly one-fourth of the patients who were initially diagnosed with UnTBAD may progress to CoTBAD (1,2). Further, there is evidence that the aorta in 40% of UnTBAD develop dilatation within a period of 18 months (3,4). To avoid these complications and progression of the natural disease, it has been suggested to identify the 'high risk' subset of patients. Alexander et al (5) in their review have enumerated a battery of risk factors to identify these 'high risk' patients. They strongly recommended *'timely prophylactic TEVAR intervention'* in these patients.

This brings us to a juncture where many questions are left unanswered with limited literature. 'How to categorise high and low-risk patients?'. 'What are the flow dynamics and pathophysiology of the Type B

aortic dissection?'. 'What patient factors contribute to the progression of the disease?'. 'Is there a possible way to stop the progression of the disease?'.

The progression of the pathology in these patients is dependent on the flow dynamics involving the intimo-medial dissection flap and entry tear. Unfortunately, this factor is not entirely understood. The intimo-medial dissection flap is thin and fragile in acute conditions while it is more mature and fibrosed in chronic aortic dissection. The margins of entry tears in acute dissection are thin and free-floating fragile, while that in chronic dissections are well defined with a fibrosed rim. To complicate the flow dynamics further, the aorta lies in three different axes with varying degrees of concavity and convexity at the level of the aortic arch. The elastic property of the aorta in varying between patients and between different segments in a patient. Apart from these, patient factors like age, ethnicity, gender, genetic association, and comorbidities like hypertension, and diabetes mellitus also influence the flow dynamics (7). All these factors have a complex interaction and influence the progression of the disease from UnTBAD to CoTBAD.

We have incontrovertible evidence that patent false lumen (FL) is an important factor for future complications including dilated false lumen compressing the true lumen, aortic dilation, malperfusion and rupture. At the turn of this century, Fattori et al in their research has shown that the growth rate of the descending aorta is substantially higher with a patent false lumen (3.7mm/year as compared to 1.1mm/year). Further, it is demonstrated that the growth rate of the aorta is significantly reduced in patients where complete false lumen thrombosis is achieved (8,9). The patency of the FL is directly related to the size of the entry tear and its location. A larger entry tear will direct more blood to the FL keeping it patent. This eventually may lead to true lumen collapse; higher wall stresses in FL causing aortic dilatation. Additionally, the proximal location of the entry tear tends to raise the diastolic pressure in FL, a tendency toward aortic dilatation (10).

Hence all our efforts would be directed to achieving false lumen thrombosis in type B aortic dissection. Optimum medical management may achieve this in most of these UnTBAD. But unfortunately, it is not in 'all' patients. A proper subset of patients does not respond to this medical management. In other words, these patients are 'allowed' to progress from UnTBAD to CoTBAD under the banner of 'medical management'. It is imperative to take the bull by its horn. Recently, the long-term clinical benefits of thoracic endovascular aortic repair in UnTBAD have been enumerated (11). Though larger randomised trials are warranted, with the limited evidence available, these patients have to be categorised based on laboratory, radiological and clinical parameter. The identified 'high-risk patients' have to have a lower threshold for prophylactic aortic intervention

Reference

1. A multicentre clinical trial of endovascular stent graft repair of acute catastrophies of the descending thoracic aorta. Cambria RP, Crawford RS, Cho JS et al. J Vasc Surg 2009;50:1255-64:e1251-54.

2. The incidence of delayed complications in acute type B aortic dissections is underestimated. Reutersberg B, Trenner M, Haller B, Geisbusch S, Reeps C & Eckstein HH. J Vasc Surg 2018;68:356-63.

3The natural history of medically managed acute type B aortic dissection. Durham CA, Cambria RP, Wang LJ, Ergul EA, Aranson NJ, Patel VI & Conrad MF. J Vasc Surg 2015;61:1192-9.

4. TEVAR for chronic aortic dissection – is covering the primary entry tear enough?. Kolbel T, Tsilimparis N, Wipper S et al. J Cardiovasc Surg (Torino) 2014;55:519-527.

5. Alexander et al. Risk Profile Analysis of Uncomplicated Type B Aortic Dissection Patients Undergoing Thoracic Endovascular Aortic Repair: Laboratory & Radiographic Predictors. Journal of Cardiac Surgery. JOCS-2022-REV-679

6. Fattori R, Bacchi-Reggiani L, Bertaccini P, et al. Evolution of aortic dissection after surgical repair. Am J Cardiol 2000;86:868-72.

7. Al-Tawil M, Geragotellis A, Jubouri M, Tan SZ, Mohammed I, Williams I, Bashir M. Population risk profile analysis of acute uncomplicated type B aortic dissection patients undergoing thoracic endovascular aortic repair. Asian Cardiovasc Thorac Ann. 2022 May 9:2184923221099771.

8. Kunishige H, Myojin K, Ishibashi Y, Ishii K, Kawasaki M, Oka J. Predictors of surgical indications for acute type B aortic dissection based on enlargement of aortic diameter during the chronic phase. Jpn J Thorac Cardiovasc Surg. 2006;54(11):477-82

9. Sueyoshi E, Sakamoto I, Hayashi K, Yamaguchi T, Imada T. Growth rate of aortic diameter in patients with type B aortic dissection during the chronic phase. Circulation. 2004;110(11 Suppl 1):Ii256-61.

10. Dziodzio T, Juraszek A, Reineke D, Jenni H, Zermatte E, Zimpfer D, Stoiber M, Scheikl V, Czerny M. Experimental acute type B aortic dissection: different sites of primary entry tears cause different ways of propagation. **Ann Thorac Surg** . 2011; *91* : 724– 727.

11. Jubouri M, Al-Tawil M, Yip HCA, Bashir A, Tan SZCP, Bashir M, Anderson R, Bailey D, Nienaber CA, Coselli JS, Williams I. Mid- and long-term outcomes of thoracic endovascular aortic repair in acute and subacute uncomplicated type B aortic dissection. J Card Surg. 2022 May;37(5):1328-1339.