

# **Case Report**

# Entry tear hemodynamics using 4D flow MRI in a patient with acute type B aortic dissection $^{x,xx}$

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## ABSTRACT

Stanford type B aortic dissection (TBAD) is a potentially fatal condition involving a tear in the descending aorta. As TBAD can be managed with medical therapy or surgical repair, identifying predictors of adverse outcomes is important to risk-stratify patients for preemptive surgical procedures. 4D flow magnetic resonance imaging (MRI) has shown to be useful in characterizing the complex hemodynamics seen in TBAD patients and correlating flow patterns with adverse outcomes. We report a case of a 58-year-old man who presented to the hospital with acute TBAD and a large primary entry tear. He was initially managed with medical therapy due to his stable clinical status and computed tomographic angiography showing a stable dissection. However, 4D flow MRI showed high velocity flow through the entry tear, which foreshadowed the later clinical decompensation of the patient. Our case demonstrates that performing 4D flow MRI on TBAD patients is feasible and can provide valuable information in the decision to pursue medical or surgical management.

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Abbreviations: TBAD, Type B aortic dissection; FL, false lumen; TL, true lumen; 4D, 4-dimensional; MRI, magnetic resonance imaging; CTA, computed tomography angiography; MRA, magnetic resonance angiography.

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#### Introduction

Stanford Type B aortic dissection (TBAD) occurs when a tear develops in the intimal layer of the aorta, distal to the aortic arch, creating a flap between the medial layers of the descending aorta. Complication rates can approach 30% within 14 days of presentation [1]. Based on presenting symptoms and clinical course, TBAD can be managed either medically with antiimpulse therapy or with open surgical or endovascular repair. Rapid aneurysmal expansion of the false lumen (FL) is associated with poor outcomes in TBAD patients. Identifying predictors of adverse outcomes may aid in risk-stratifying patients for preemptive surgical procedures. One marker of interest is flow through the primary entry tear from the native true lumen (TL) into the FL. 4-dimensional (4D) flow magnetic resonance imaging (MRI) is a useful diagnostic tool to characterize the complex hemodynamics and flow patterns seen in TBAD patients [2,3]. Recent studies have shown that larger maximum aortic diameter, higher FL forward flow, and greater FL peak velocity are associated with adverse outcomes in TBAD [4-6]. Here, we present 4D flow MRI of a patient acutely presenting with TBAD and a large primary entry tear.

#### **Case Report**

A 58-year-old man (BMI 36) was admitted to the emergency department 4 hours after the onset of left-sided chest pain. He had a 38 pack-year smoking history and uncontrolled hypertension. The patient described the chest pain as sharp and constant, radiating into his left jaw and arm, with shortness of breath. On physical examination, his blood pressure was 209/97 mmHg and heart rate was 73 bpm. Electrocardiography showed nonspecific changes, and other laboratory results were as follows: troponin I, 23 ng/mL; D-dimer, 671 ng/mL; WBC 15.3  $\times$  10<sup>9</sup>/L. Computed tomography angiography (CTA) of the chest, abdomen, and pelvis showed dissection of the thoracic aorta from the innominate trunk to the iliac arteries (Fig. 1). In the context of uncontrolled hypertension, this clinical presentation was highly suggestive of acute TBAD. Medical therapy was started with blood pressure and heart rate control, and significant improvement in chest pain and blood pressure was observed within 24 hours with eventual stabilization. Forty-eight hours after admission, repeat CTA showed no significant change from prior imaging, supporting continuation of medical management. At this time, 4D flow MR with chest MR angiography (MRA) also showed dissection involving the proximal portions of the left brachiocephalic and common carotid arteries extending beyond the field of view. A maximum aortic diameter of 4.3 cm was observed in the proximal arch. The visualized portion of the dissection appeared stable compared to the prior study. However, 4D flow MRI showed high flow and velocity through a primary entry tear located 11.8 mm from the left subclavian artery with a diameter of 10.5 mm (Fig. 2). Analysis of flow through the entry tear into the FL was as follows: forward volume, 58.370 mL; peak velocity, 1.78 m/s; max pressure gradient 1279.083 mmHg; maximum flow, 244.714 mL/s. These values are all very elevated compared to others from our institutional TBAD cohort. Within 48 hours of the 4D flow MRI scan, the patient began experiencing chest pain again with blood pressure rising to 161/110 mmHg, consistent with worsening dissection and rupture. He was taken for emergent surgical repair within a few hours of clinical decompensation, but the further rupture in the distal descending aorta was found to be unrepairable, and the patient expired.

### Discussion

In monitoring acute presentation of TBAD, MRA or CTA can be performed with adequate spatial resolution to follow aortic diameter and extent of dissection [7]. MRA requires longer scan times than CTA, which can be prohibitive in the acute setting due to the often unstable clinical condition of TBAD patients. However, neither of these methods provides hemodynamic profiles of flow alterations in the FL in TBAD, which can be as-



Fig. 1 – Axial (A), sagittal (B), and coronal (C), CT angiography demonstrating the borders of the true (TL) and false lumens (FL) and primary entry tear (ET) location. Maximum aortic diameter = 5.2 cm. Maximum FL diameter = 3.2 cm.



Fig. 2 – (A) 3D velocity streamlines generated from 4D flow in the thoracic aorta. (B) Flow rate through the primary entry tear over the cardiac cycle.

sociated with adverse outcomes [4]. Prior to this patient's rapid decompensation, his vitals and physical exam were stable for 24 hours, suggesting the patient could be managed with antiimpulse therapy alone without any surgical intervention. 4D flow MRI acquired concurrently exhibited high velocity flow through the proximal entry tear into the FL. The entry tear flow and velocity in this patient were the highest values we have seen so far in our institutional TBAD cohort [4]. We hypothesize that this high velocity, high volume flow ultimately led to increased pressurization in the FL and eventual rupture, as has been suggested in other studies [7]. This case demonstrates that performing 4D flow MRI in acute TBAD is feasible and can also capture the complex hemodynamics present. While 4D flow MRI lengthens scan times by approximately ten minutes, it may provide important additional risk stratification metrics in patients with otherwise equivocal clinical and CTA/MRA findings.

#### **Patient consent**

The patient provided written informed consent for publication of his case.

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