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# Management of Stanford Type B Aortic Dissection in Pregnancy Following Successful Aortic Root Replacement

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**Abstract**

**Background:** Up to 90% of individuals with Marfan syndrome have underlying cardiovascular abnormalities, the most common of which affect the aorta. As the physiologic changes of pregnancy and the puerperium contribute to increased cardiovascular stress, pregnant persons with Marfan syndrome are at significant risk of complications such as aortic dissection. However, there is little consensus on the ideal management of patients with Marfan syndrome who experience this outcome during pregnancy.

**Case Presentation:** We present a case of Stanford Type B aortic dissection secondary to Marfan syndrome in a 31-year-old female at 29 weeks' gestation who had undergone an aortic valve and root replacement prior to conception. Although her aortic dissection was initially stable with medical management, she later required urgent cesarean delivery for fetal decelerations attributed to resultant changes in uteroplacental circulation. Her immediate post-operative course was complicated by bilateral rectus sheath hematomas due to the need for therapeutic anticoagulation in the perioperative period, which necessitated re-operation on post-operative day 1. By post-operative day 5, she was stable for discharge from the cardiovascular intensive care unit and, by post-operative day 7, she was deemed appropriate for outpatient management on a medication regimen including carvedilol, amlodipine, and enoxaparin.

**Conclusion:** While the maternal and neonatal outcomes were ultimately favorable, the patient's course highlights the need for clear preconception counseling in patients with genetic aortopathy regardless of prior surgical history. Additionally, it underscores the need for close multidisciplinary collaboration between maternal-fetal medicine, neonatology, cardiothoracic and vascular surgery, and anesthesiology when pregnancy complications arise particularly at an extremely premature gestational age.

**Keywords:** Aortic dissection in pregnancy; Aortopathy in pregnancy; Marfan syndrome; Cardio-obstetrics

## Introduction

Marfan syndrome (MFS) is an inherited connective tissue disorder with a prevalence of 6.5 per 100,000 [1]. Up to 90% of affected individuals have cardiovascular abnormalities, including aortic root enlargement, aortic branch abnormalities, aortic regurgitation, pulmonary arterial root dilatation, atrioventricular valve abnormalities, and cardiomyopathy [2]. In the MFS population, aortic dissection resulting from an enlarged aortic root is a leading cause of morbidity and mortality. Antenatally and postpartum, patients with MFS are at an even greater risk of aortic dissection, largely due to the physiologic cardiovascular changes of pregnancy. The incidence of cardiovascular complications in pregnant persons with MFS is estimated to be as high as 27-40% [3]. Nonetheless, there is limited evidence regarding the optimal management of and outcomes resulting from MFS-associated aortopathy in pregnancy. To augment the literature, we present the course of an aortic dissection secondary to MFS in a third trimester twin gestation, emphasizing the importance of multidisciplinary collaboration in ensuring optimal patient outcomes.

## Case Presentation

A 31-year-old gravida 1 with known MFS and a dichorionic/diamniotic twin pregnancy presented at 29 weeks' gestation due to shortness of breath and back pain. History was significant for mechanical aortic valve and root replacement 2 years earlier as well as previous thoracic and lumbar level rod placement for scoliosis. During pregnancy, she received aspirin 81 mg daily, metoprolol 25 mg twice daily, and enoxaparin 75 mg twice daily. An echocardiogram performed 8-weeks prior was without regurgitation or gradient across the mechanical aortic valve.

Initial vital signs included a heart rate of 102 beats per minute, blood pressure of 149/93 mm Hg, and oxygen saturation of 97%. Chest x-ray (Figure 1) showed a prominent cardiomediastinal silhouette and tortuous thoracic aorta. Computed tomography angiogram (CTA, Figure 2) demonstrated a Stanford type B aortic dissection (TBAD) extending from the left subclavian to celiac artery. The fetal status was reassuring for both twins.

As the patient was hemodynamically stable, her twins were premature, and her severe scoliosis would have inhibited safe access of the thoracic aorta, the decision was made for medical management in the cardiovascular intensive care unit (CVICU). She was started on esmolol and nicardipine infusions to maintain her heart rate between 60-70 beats per minute and blood pressure below 120/80 mm Hg. She was given betamethasone 12 mg intramuscularly to accelerate fetal lung maturity and reduce the risk of neonatal morbidity and mortality. Anticoagulation was held with the intention to transition to an unfractionated heparin infusion the following morning in case of imminent delivery.

Within 20 hours, the patient experienced respiratory decompensation and required 40 L of supplemental oxygen via high-flow nasal cannula to maintain saturations sufficient for fetal perfusion (> 95%). Simultaneously, fetus B began having recurrent heart rate decelerations that progressed to a terminal bradycardia (Figure 3). Accordingly, emergent delivery under general anesthesia was advised. An uncomplicated low transverse cesarean section with a quantified blood loss of 650 mL was performed, resulting in delivery of viable twins with Apgar scores of 8/9 and 4/8, respectively.

She remained intubated through a repeat CTA, which confirmed a stable TBAD (Figure 4), before being returned to the CVICU and extubated on post-operative day (POD) 0. Esmolol and nicardipine infusions were continued to maintain the aforementioned perioperative vital sign parameters. An unfractionated heparin infusion was resumed 6-hours post operatively.

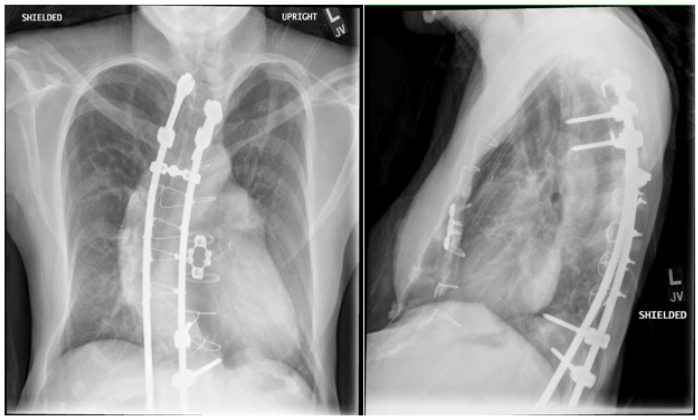
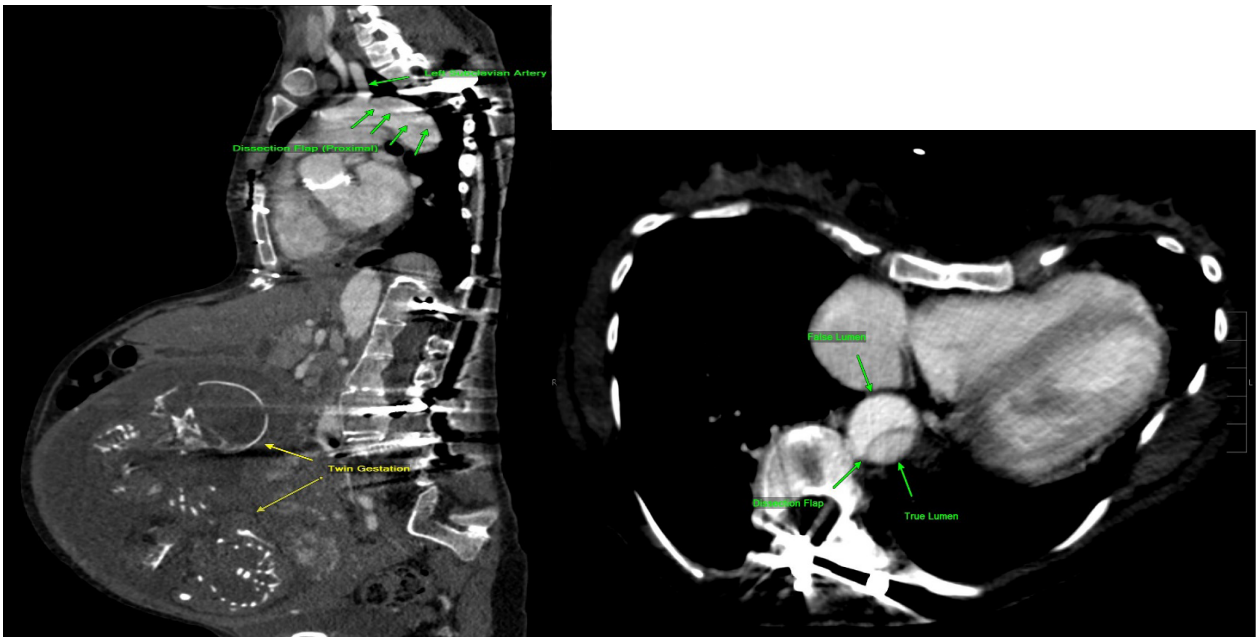



**Figure 1**: Admission chest x-ray demonstrating a prominent cardiomediastinal silhouette and tortuous thoracic aorta. The visualized sternotomy wires and screws are from the patient's aortic root and valve replacement two years prior.




**(a)** **(b)**

**Figure 2:** Admission computerized tomography angiogram (CTA) showing a Stanford type B aortic dissection in the **a)** sagittal plane and **b)** axial plane.


**Figure 3**: External fetal heart rate monitoring with a terminal bradycardia for fetus B.

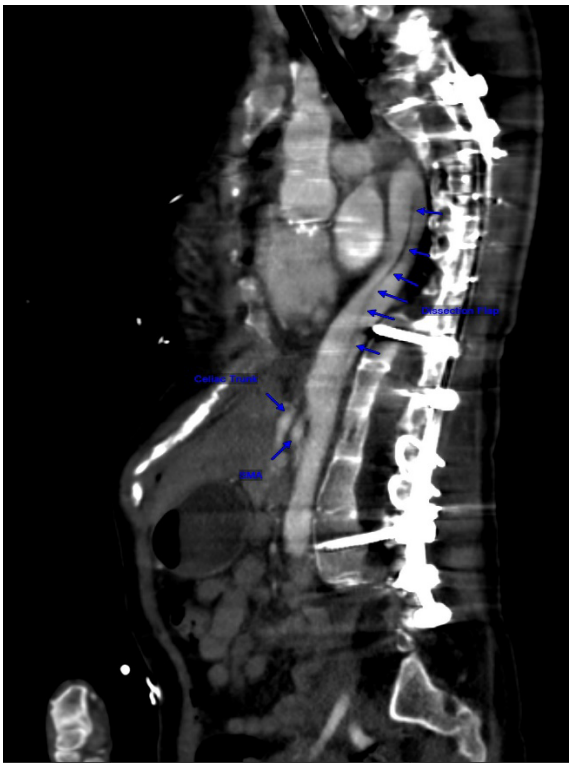



**Figure 4**: Post-operative day 0 computerized tomography angiogram (CTA) showing a stable Stanford type B aortic dissection.

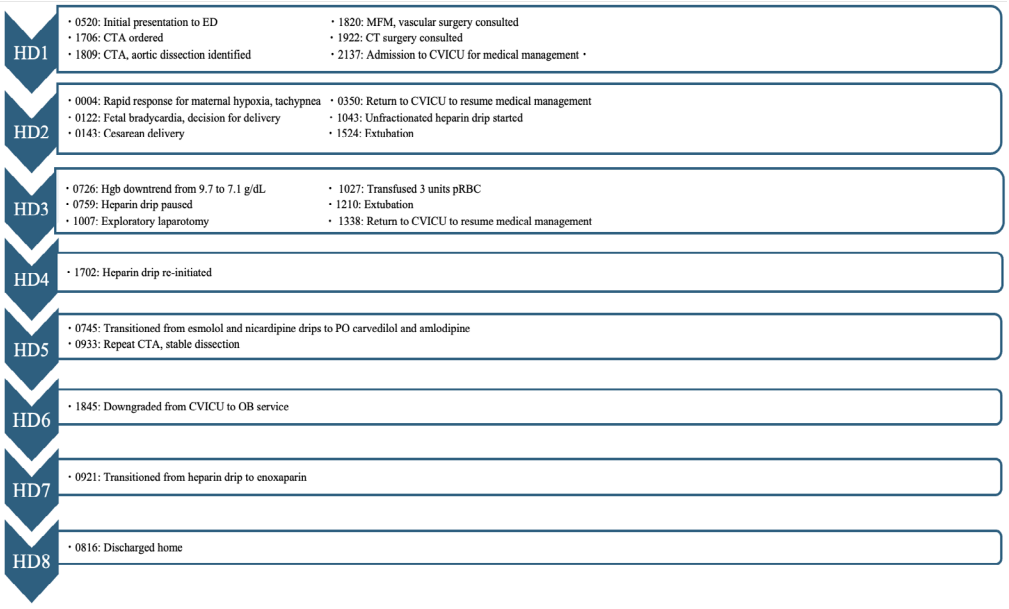



**Figure 5**: Graphical Summary.

**Figure Key:** ED: Emergency department; CTA: Computerized tomography angiogram; MFM: Maternal-fetal medicine; CT surgery: Cardiothoracic surgery; CVICU: Cardiovascular intensive care unit; Hgb: Hemoglobin; pRBC: Packed red blood cells; PO: by Mouth; OB: Obstetric.

On POD 1, her hemoglobin dropped from 9.7 to 7.1 g/dL. Physical exam revealed tachycardia with a heart rate over 110 beats per minute and significant abdominal distension. Bedside ultrasound found an intraperitoneal fluid collection. She was immediately taken for exploratory laparotomy, during which bilateral rectus sheath hematomas with 1500 mL of hemoperitoneum were noted. Hemoperitoneum was evacuated, the rectus muscles were made hemostatic, and she was transfused 3 units of packed red blood cells. Post-operatively, the patient was transferred back to the CVICU to continue medical management.

On POD 3, she was transitioned to carvedilol 12.5 mg twice daily for rate control and amlodipine 10 mg daily for hypertension management. She was downgraded from the CVICU to obstetric service on POD 5. On POD 7, she was stable for discharge and converted from he heparin infusion to enoxaparin 75 mg twice daily until she could be bridged to her pre-pregnancy warfarin. On discharge, routine postpartum visits, a 2-month post-operative CTA, and outpatient cardiothoracic surgery follow-up were planned.

## Discussion

This case illustrates the unique presentation and challenges facing a 31-year-old gravida 1 with known MFS who experienced an aortic dissection at 29 weeks' gestation in spite of having undergone aortic repair prior to conceiving. The increased risk of aortic dissection in patients like her with pre-existing aortopathy results from numerous physiologic changes of pregnancy. First, physiologic increases in heart rate and stroke volume contribute to a 30-40% rise in cardiac output over the course of pregnancy, with maximum cardiac output reached by 28 weeks' gestation [4]. At term, at least 12% of this cardiac output or 500 to 800 mL/minute is directed to the uterus. This rapid change in circulating blood flow, heart rate, and stroke volume intensifies shearing forces on the aorta. Additionally, compensatory increases in ventricular ejection occur as the gravid uterus expands and compresses the aorta, magnifying the risk of intimal tears. Beyond this, high estrogen levels are suggested to alter the structural integrity of the aorta, rendering it more susceptible to injury in the face of hemodynamic stressors [4].

As observed in our patient, maternal hypoxemia and uterine hypoperfusion are the leading causes of fetal insult related to aortic dissection [5]. Uterine hypoperfusion likely results from dissection extension into the internal iliac artery, from which the uterine arteries arise and perfuse the placenta [5]. A key difference between this patient's TBAD and a Stanford type A aortic dissection (TAAD) is that TAAD involves the ascending aorta, while TBAD involves the descending aorta and its tributaries. Because TBAD more often affects the internal iliac artery and compromises uteroplacental circulation, research suggests that rates of fetal mortality may be higher with TBAD compared with TAAD (35% vs 10%, respectively) [6].

Whereas a general consensus exists in favor of surgical management for TAAD, controversy remains regarding surgical versus medical management for TBAD, especially in pregnancy. Outside pregnancy, a 20-year analysis of the International Registry of Acute Aortic Dissection found the majority of TBAD cases were treated medically [5]. During pregnancy, in the absence of rupture or malperfusion, conservative management of TBAD continues to be preferred. However, a number of high risk anatomical and clinical features, including refractory pain, uncontrolled hypertension, initial aortic diameter greater than 4 centimeters, enlarging intramural hematoma, rapidly expanding false lumen diameter ( > 22 millimeters), and progression despite adequate medical therapy, have also been proposed as indications for surgery and necessitate repair in up to 20% of cases [5].

When surgery is required for TAAD or unstable TBAD in pregnancy, endovascular approaches may be favored as a bridge to more definitive open repair and timing is based on gestational age at diagnosis. Once viability has been achieved but before 28-30 weeks, urgent surgical repair with aggressive fetal monitoring is advised due to the risk of morbidity associated with delivery of an extremely premature neonate [7]. After 28-30 weeks' gestation, when complications of prematurity are less severe, urgent cesarean delivery followed by immediate aortic repair is advised [7]. Case series on aortic dissection repair during pregnancy document rates of maternal, fetal, and neonatal survival as high as 73-82% with either approach [4].

Anesthetic considerations are also paramount when determining appropriate interventions for aortic dissection in pregnancy, particularly in the context of MFS. The lumbar spine deformities and dural ectasia common to MFS may complicate epidural catheter placement and spinal anesthetic administration. When possible, pre-operative magnetic resonance imaging (MRI) should be obtained to assess for dural ectasia due to the associated risk of accidental dural puncture and ineffective spinal anesthesia [9].

When a cesarean delivery is planned, combined spinal-epidural anesthesia is preferred for its hemodynamic control, especially as general anesthesia may increase stress on the aortic root and worsen dissection. Nonetheless, when general anesthesia is

required for emergent cases such as ours, hemodynamic control can be maintained by inserting a radial artery catheter to allow for continuous blood pressure monitoring and administering opioids to blunt responses to laryngoscopy and tracheal intubation [8]. General anesthesia with volatile anesthetics or total intravenous anesthesia is favored to facilitate combined delivery and repair of the dissection, the latter of which is typically lengthy and requires heparinization that may increase the risk of epidural hematoma if neuraxial anesthesia is attempted [9].

Because pregnancy, postpartum, and the perioperative period are hypercoagulable states that increase the risk of valvular thrombosis in patients like ours with mechanical prostheses, the American College of Cardiology, American Heart Association, and American College of Obstetricians and Gynecologists recommend dose-adjusted (therapeutic) anticoagulation for this population [10,11]. No randomized-controlled trials have compared anticoagulant regimens in pregnant patients with mechanical heart valves. To balance the teratogenicity of warfarin in the first trimester against its improved efficacy in the non-pregnant population, expert guidance suggests two potential regimens: 1) low-molecular-weight (1 mg/kg every 12 hours) or unfractionated heparin (10,000 units or more twice daily) throughout pregnancy, or 2) low-molecular-weight or unfractionated heparin between 6 and 13 weeks followed by substitution with warfarin until early term when heparin-based agents can be resumed to reduce bleeding complications [10,11]. Low-dose aspirin is also recommended from the second trimester [11].

For planned delivery, dose-adjusted low-molecular-weight or unfractionated heparin should be discontinued 24 or 12 hours in advance, respectively; this reduces the risk of intra-operative hemorrhage and facilitates safe neuraxial anesthesia when clinically appropriate [11]. For unplanned delivery, patients with recent warfarin exposure will require cesarean delivery and neonatal treatment with vitamin K and fresh frozen plasma due to the risks of fetal hemorrhage with this medication [11]. In contrast, patients with recent low-molecular-weight or unfractionated heparin exposure may be candidates for reversal, though should still avoid neuraxial anesthesia due to the theoretical risk of epidural or spinal hematoma [11]. If cesarean delivery is required in therapeutically anticoagulated patients, a vertical midline incision should be performed to reduce the risk of hematomas resulting from inferior epigastric artery injury with a low transverse approach. Regardless of antenatal anticoagulation, postpartum patients with mechanical heart valves should be restarted on warfarin alongside a low-molecular-weight or unfractionated heparin bridge until the international normalized ratio is therapeutic (2.0 -3.0) [10].

Finally, preconception counseling for patients with known MFS and postpartum counseling for those who have experienced a related cardiovascular complication in pregnancy is critical. In accordance with the Modified World Health Organization Pregnancy Risk Classification for Women With Preexisting Cardiovascular Disease, patients with MFS and no aortopathy are counseled regarding a pregnancy-related risk of maternal cardiac events from 11-19%. As the aortic root dilation reaches 40 mm, the risk of such events increases to 20-27%, and prophylactic aortic root replacement is recommended in advance of potential conception [3]. Even when patients like ours pursue surgery, they should be advised that, while aortic root replacement significantly reduces the risk of TAAD, the risk of TBAD remains as high as 9% [12]. Regardless of surgical status, once aortic root dilation exceeds 45 mm, pregnancy is considered contraindicated for patients with MFS due to maternal event rates of 40-100% [3]. Appropriate contraception should be offered, with barrier and progestin-only methods preferred for patients with MFS aortopathy [12].

Presently, a single case report exists on the natural pregnancy history in a patient with known, chronic TBAD. This patient was managed with oral beta-blockers and hydralazine and ultimately had an uneventful, term cesarean delivery [13]. Nonetheless, as there is otherwise insufficient data on the long-term outcomes or safety of pregnancy in patients with a history of TBAD, we would advise patients such as ours to avoid future pregnancies. As this case illustrates, if subsequent pregnancies are still pursued, early multidisciplinary collaboration between maternal-fetal medicine, neonatology, cardiology, cardiothoracic and vascular surgery, and anesthesiology should be prioritized.

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**Patient Consent:** Informed consent was obtained from the patient for publication of this case report.

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