DOI: 10.1111/jocs.14592

ORIGINAL ARTICLE

JOURNAL OF CARDIAC SURGERY WILEY

Aortic root valve-sparing repair and dissections in Marfans syndrome during pregnancy: A case series

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Funding information
Mississippi Center for Clinical and Translational Research (MCCTR), Grant/Award Number: NIGMS U54 GM-115428

Abstract

Introduction: Marfan syndrome is a connective tissue disorder caused by mutations in the fibrillar FBN-1 gene. Aortic dissection and rupture are major causes of morbidity and mortality and are of special concern during pregnancy.

Materials and Methods: The authors report four cases of aortic root repair with preservation of the native aortic valve that have has created a discussion between cardiothoracic surgeons, obstetricians, and gynecologists regarding the best care for Marfan syndrome patients. We present these cases here with a review of the literature.

Results: Surgery of the aorta and valves in Marfan syndrome is less risky than in previous eras and surgical management guidelines are generally accepted. Yet, we may be unnecessarily referring women to terminate pregnancies or to avoid pregnancy. We believe there may be alternative options for these patients.

Conclusions: Marfan syndrome during pregnancy can be navigated with preconception counseling, antepartum care, and close postpartum follow-up involving an appropriate multidisciplinary team.

1 | INTRODUCTION

Marfan syndrome is a connective tissue disorder caused by mutations in the fibrillar FBN-1 gene, with an estimated prevalence of 2 to 3 of 10 000.[1-3] The diagnosis is clinical, relying on the Ghent criteria[4-6] and often in combination with genetic testing. Transmission is autosomal dominant with variable penetrance but about 25% of cases are sporadic.[7] Patients experience skeletal, ocular, and serious cardiovascular complications. Aortic dissection and rupture are major causes of morbidity and mortality and contribute to the reduced life expectancy for patients with Marfan syndrome.[2-4]

Marfan syndrome presents serious concerns related to pregnancy. As well as the risk of transmission to offspring, the connective tissue abnormalities present in Marfan syndrome put patients at risk for obstetric complications including preterm premature rupture of membranes, cervical insufficiency, and a 15% incidence of preterm birth.[1,5]

The most serious pregnancy concerns are related to the increased risk of cardiovascular complications. Aortic root aneurysm and dissection are a significant cause of morbidity and mortality in patients with Marfan syndrome.[3] Concerns regarding these risks in the setting of the physiologic changes of pregnancy have led to recommendations that women with Marfan syndrome should avoid pregnancy. Medical literature has suggested that pregnancy increases the risk of aortic dilation and dissection but the effect of pregnancy on aortic disease has been debated.[1] It has been suggested that progressive dilation of the aorta during pregnancy more than 5 mm necessitates urgent surgery either after therapeutic termination of pregnancy or during pregnancy.[8]

Pregnancy induces profound physiologic and hormonal alterations. Increases in blood volume, heart rate, stroke volume, cardiac

output, LV mass, and cardiac diastolic dimensions occur beginning in the first trimester.[5] Estrogen and progesterone lead to structural changes of the vascular walls with the fragmentation of reticulum fibers, decreased elastic fibers, and decreased smooth muscle. Smith et al[9] prospectively examined the arterial pulse wave in 60 pregnant women enrolled in three stages of pregnancy; 17 to 20, 25 to 28, and 33 to 36 weeks. In the 53 women that remained normotensive throughout the pregnancy, the data showed a significantly shorter time from the start of the arterial waveform to the second peak/shoulder as well as a significant shortening in the rapid ejection time, and the augmentation pressure and augmentation index were significantly lower in pregnancy. A study by Macedo et al[10] matched 193 women with normal singleton pregnancies at 11 to 41 weeks of gestation to 23 nonpregnant controls. Their study demonstrated no significant change in pulse wave velocity (carotid-radial and carotid-femoral) during gestation, and only marginal differences between pregnant and nonpregnant women ($P = .03$ and $P = .05$ for carotid-radial and carotid-femoral, respectively). These studies confirm the known clinical findings associated with the cardiovascular changes of pregnancy, namely that of vasodilation of peripheral vessels and expansion of blood volume.[9,11]

The highest demand physiologically occurs during the third trimester, labor, and in the immediate postpartum period. During labor, there is an increase in sympathetic tone and systemic vascular resistance, which are further increased by pain stimuli. Each contraction forces 300 to 500 mL of blood back to the central venous system. The second stage of labor is associated with further increases in blood pressure and heart rate. Delivery of the placenta sends approximately 500 mL of blood back to the maternal circulation, increasing the central venous pressure, preload and cardiac output.

Women with underlying cardiovascular pathology often have a diminished ability to adapt to the cardiovascular changes of pregnancy.[7] Increases in vasculature wall stress, myocardial contractility, shear forces, and pulse pressure; coupled with changes to the vasculature walls are believed to render the aortic wall more susceptible to injury by hemodynamic forces.[2] Maximal hemodynamic changes are seen in the third trimester, intrapartum and postpartum, which coincides with an increased risk of aortic complications during this time.

2 | MATERIALS AND METHODS

We retrospectively analyzed four of our institution's cases involving the peripartum care of women with Marfan syndrome. To further understand this process, we performed a thorough literature search identifying articles that addressed work up, perioperative care, and complications involving pregnant women with Marfan syndrome. This review sought to identify similarities between our cases and, in conjunction with existing literature, offer recommendations for cardiac surgeons and obstetricians who may encounter this patient population. A brief review of our cases is presented below.

2.1 | Case 1

A 31-year-old female presented to her obstetrician/gynecologist (OB/GYN) for prenatal care. The patient was known to have Marfan syndrome since birth but was lost to follow up until presentation for obstetric care. The patient had a dilated aortic root measuring 4.5 cm. The patient noted occasional chest pain, shortness of breath, and lower extremity edema. The echocardiogram demonstrated mild concentric left ventricular (LV) hypertrophy, with a normal ejection fraction of 65%. The sinotubular junction measured 3.5 cm with the sinuses measuring 5 cm with an ascending aorta of 3.4 cm. The echocardiogram noted mild aortic regurgitation. Per the OB/GYN, the patient was advised to have an abortion to avoid the risk of aortic dissection, possible death, and loss of the pregnancy. After the abortion, the patient was referred to cardiac surgery for further evaluation. The patient was scheduled for an aortic valve-sparing root replacement. The patient tolerated the procedure well and was discharged home a few days after surgery.

2.2 | Case 2

A 27-year-old female with a known history of Marfan syndrome had an aortic root aneurysm and a family history (father) with the disease. One year prior, the transthoracic echocardiogram, an aortic root diameter was at 4.2 cm. She underwent a forceps-assisted vaginal delivery of her first child 7 months before the visit with the cardiothoracic surgeon. On magnetic resonance imaging (MRI), the aortic root was found to have a focal dissection and was now enlarged to 5.8 × 5.6 cm. By transthoracic echocardiogram, the patient now had moderate central aortic insufficiency of a trileaflet valve. She underwent an elective valve-sparing aortic root replacement (with the David reimplantation technique) and replacement of the ascending aorta with a hemiarch repair under deep hypothermic circulatory arrest. She had an uneventful recovery. Four years later, the transthoracic echocardiogram has minimal aortic insufficiency and normal LV function.

2.3 | Case 3

A 29-year-old female with Marfan syndrome (genetically confirmed) developed severe shortness of breath with minimal exertion several weeks after cesarean section delivery of her second child. She was found to have severe mitral regurgitation from a flail P2 segment and preserved LV systolic function. By computed tomography angiography, she had a 4.5 × 5.0 cm aortic root aneurysm. She had mild aortic insufficiency and an LV end-diastolic dimension of 75 mm. She underwent a valve-sparing aortic root replacement (using the David reimplantation technique) and mitral valve repair with a quadrangular P2 resection and complete rigid ring annuloplasty. She had an uneventful recovery. A transthoracic echo 18 months later showed minimal aortic insufficiency, minimal mitral regurgitation, preserved LV systolic function, and an LV end-diastolic dimension of 6.2 cm.

2.4 | Case 4

A 29-year-old female with a known history of Marfan (diagnosed at age 5) and a strong family history including her father, brother, and uncle was interested in getting pregnant a second time. During her initial pregnancy 19 months prior her aortic root measured 3.8 cm, and with close follow up and guideline-directed care she delivered without complication via cesarean section. At her presentation, cardiac MRI revealed an aortic root measuring 4.3 cm. After counseling, she elected to undergo a valve-sparing aortic root replacement. Her operative course was complicated by iatrogenic transection of an anomalous right coronary artery arising from the left coronary sinus. In addition to the root replacement, she underwent a successful bypass of the right coronary artery using the right internal mammary artery. Her postoperative course was complicated by atrial fibrillation for which she was successfully cardioverted. She was started on dabigatran and was discharged home on postoperative day 5. She proceeded to become pregnant and delivered another healthy child via cesarean section 2 years following her surgery. Four days after discharge home she returned to the emergency department with chest pain radiating to her back and was diagnosed with segmental and subsegmental pulmonary emboli and a type B aortic dissection originating distal to the subclavian and continued into bilateral iliac arteries. She was treated conservatively initially, however ultimately, she underwent endovascular repair of her thoracic aorta and left carotid subclavian bypass from which she recovered well. She has returned to work and is caring for her two healthy children.

Existing literature is composed largely of case reports and retrospective studies. Case reports have described serious cardiovascular risks to Marfan syndrome patients in pregnancy and have led to recommendations for these women to avoid conception. Case reports are subject to bias, as uncomplicated pregnancies have gone unreported.[3] Recent studies have sought to study aortic changes in pregnancy and to clarify risks, which may be lower today than was described in previous studies. In addition, there is considerable phenotypic heterogeneity among Marfan syndrome patients, and women who opt to conceive may represent a different phenotypic population than those who do not. An event-free pregnancy cannot be guaranteed and aortic dissection can occur even at normal diameters. However, the risk may be acceptable and comparable to nonpregnant state in a select low-risk group of women.

A retrospective study by Pacini et al[3] compared the risk of severe aortic complications in 85 women with Marfan syndrome (who had a total of 160 pregnancies) to 68 women with Marfan syndrome who had never been pregnant. All Marfan syndrome patients from their clinic were eligible for analysis. They noted a total of seven aortic complications (including six dissections) for risk of 4.4% per pregnancy, compared to 14 events (10 dissections) for risk of 0.9% per year while not pregnant, in the absence of specific care. Consistent with previous reports of aortic dissection, these events mainly occurred in the third trimester or after delivery. They determined that the relative risk of aortic complication in pregnancy was 5.5 (95% confidence interval, 2.0-15.5; $P = .001$), and that the lifetime risk of aortic complication was similar among the parous and nulliparous groups. Marfan syndrome diagnosis was made later in women who had never been pregnant (33 ± 15 vs 22 ± 14; $P < .05$), therefore pregnancy appears to unmask aortopathy but is not a risk factor in increasing aortic wall diameter.[3]

Lipscomb et al[1] provided a retrospective report of 91 pregnancies in 36 asymptomatic patients with Marfan syndrome listed in a regional genetic register in the United Kingdom. They noted six aortic complications; four dissections with one immediately fatal, and two progressive root dilations without dissection requiring aortic surgeries after delivery. Of note, in two of the women, the diagnosis of Marfan syndrome was not known before aortic dissection. Dissection did occur in two normotensive women without a dilated root (40 and 42 mm). The remaining 22 women had uncomplicated pregnancies and were found not to have the aortic disease after 10.4 years of follow up. All of the patients had an aortic root measurement of less than or equal to 43 mm. They concluded that the risk is low at diameters less than 40 mm, but that aortic diameter may change during pregnancy. The risk of obstetric complications did not exceed expectations in their population.[1]

Rossiter et al[7] reported pregnancy and long-term outcomes of 45 pregnancies in 21 patients with Marfan syndrome. Aortic dissection occurred in two patients, both noted to be at increased risk before pregnancy (due to diagnosis of chronic dissection, drug abuse, and nonuse of beta-blockade). Aortic complications occurred in two out of four women with a root measurement of 40 to 43 mm. No adverse outcomes or dissections occurred when the aortic diameter was less than 40 mm. They observed the change in aortic root diameter during pregnancy and the postpartum period and noted little change. Long-term follow up (6 years) showed no worsening in long term cardiovascular prognosis or acceleration of dilation of the aortic root in 18 parous compared with 18 nulliparous women with Marfan syndrome, who were followed for up to 13 years. Obstetric complication rates were similar to the general population. There was a comparable degree of disease severity between pregnant and nonpregnant groups. They concluded that patients without cardiovascular compromise and with aortic root less than 40 mm usually tolerate pregnancy well.[4]

Meijboom et al[2] investigated the influence of pregnancy on the growth of the aortic root and sought to clarify the diameter above which pregnancy should be discouraged. They prospectively studied aortic diameter in 127 women with Marfan syndrome (61 became pregnant and 66 did not, analyzed 22 matched women from each group) with serial echocardiogram exams. Before, during, and after pregnancy, the aortic root diameter changes were not significant ($P = .77$). No aortic dissections occurred in patients without previous dissection and with an aortic root diameter less than or equal to 45 mm. During a median follow up of 6.4 years, there was no significant difference in aortic root growth between the pregnancy and control group, but root growth was significantly increased in the pregnancy compared with the nonpregnancy group in women with a diameter of greater than or equal to 40 mm. Women with an aortic

root diameter greater than 40 mm had a slightly accelerated growth in the long term. Aortic root enlargement was minimal during pregnancy when the aortic root diameter was less than 45 mm before conception, but when the diagnosis of Marfan syndrome was known before pregnancy (0.28 vs 0.19 mm/y; $P = .08$). They concluded that pregnancy may be safe up to 45 mm.[2] The type of aortic dissection (A or B) was examined by Mulder and Meijboom by combining three prospective studies.[2,7,12,13] They found that in 145 pregnancies there were no type A dissections reported in 78 nonoperated women with Marfan syndrome.[12]

3 | RESULTS

Here we present our recommendations for the evaluation of this patient group. Preconception evaluation and counseling is of critical importance for individuals with Marfan syndrome desiring pregnancy.[14] The preconception counseling should include a detailed discussion of the risk of cardiovascular compromise and complications, the transmission of Marfan syndrome to offspring, and thorough clinical evaluation including an echocardiogram. Risk stratification can be performed, encompassing the patient's hemodynamic state, functional capacity, addressing of other comorbidities, and genetic counseling.[5]

3.1 | Antepartum

A multidisciplinary team involving the patient's primary OB/GYN, cardiology, cardiothoracic surgery, maternal-fetal medicine, obstetric anesthesiology, and neonatology should be assembled and consulted. Genetic testing via chorionic villus sampling or amniocentesis should be offered, as well as a targeted fetal anatomic survey at 18 to 20 weeks and a detailed fetal echocardiogram. Maternal echocardiogram remains the mainstay of monitoring, with significant risk demonstrated if the aortic diameter is greater than equals to 4.0 cm or demonstrating a steady increase in aortic root diameter over interval echocardiograms.[1] Using echocardiogram and clinical parameters to determine maternal cardiac status will assist in the optimization of cardiac function before the early second trimester when hemodynamic changes become more pronounced. It is recommended to repeat transthoracic echocardiogram every 6 to 10 weeks, with a decreased interval if echocardiogram findings on the initial ultrasound are concerning for aortic dissection.[1,5,8] Typically, during the third-trimester serial fetal growth ultrasounds and antenatal fetal testing, has been employed.[5] Routine use of β-adrenergic receptor blockade from the mid-trimester onward has also been recommended.[7] Regarding the route of delivery, vaginal delivery is recommended with stable aortic measurements (<40-45 mm). With a vaginal delivery, a dense, slow-onset epidural and assisted second stage of labor with either vacuum assistance or forceps is recommended. Cesarean delivery is preferred for aortic diameter greater than 40 mm, aortic dissection, severe aortic regurgitation, or heart failure.[5] Aggressive treatment of hypertension with a β-blocker or vasodilator during labor and delivery is theorized to decrease the chance of aortic dissection and is therefore recommended.

3.2 | Postpartum

The risks of aortic dissection extend beyond the immediate postpartum period. There is an increased risk of dissection that persists until up to 8 weeks postpartum.[7] It is speculated that as the physiologic and hormonal effects of pregnancy began to devolve and normal hemodynamic physiology ensues after 6 to 8 weeks and risk for aortic dissection returns to baseline.

Special consideration should be given to this patient population by cardiac and vascular surgeons. While medical therapy remains the primary management for Marfan syndrome in pregnancy, a subset of patients will require invasive surgical procedures. Recognizing that neonatal outcomes are inherently tied to maternal outcomes, maternal cardiac surgery mortality has been reported to be near 10% with fetal mortality as high as 30%.[15,16] The World Health Organization pregnancy classification risk for Marfan syndrome is dependent on the degree of aortic dilation[17]: class 2 ≤ 40 mm, class 3 ≥ 40 mm, and class 4 ≥ 40 mm with prior dissection or mechanical valve. Avila et al[18] described 1000 pregnant patients with heart disease from 1989 to 1999. Of the women studied 765 (76.5%) experienced no cardiovascular events and 235 (23.5%) had cardiovascular complications. Of those patients with cardiovascular complications, the risk of maternal mortality was highest with surgeries performed for aortic dissection (22%). Barth reports a mortality rate of 1% per hour until treated among patients with dissection of the thoracic aorta.[16] The highest risk to the fetus during cardiothoracic surgery is during procedures that require cardiopulmonary bypass.[16] Therefore, strong consideration must be given to delivery first followed by maternal cardiac surgery if the fetus is at a gestational age where postdelivery neonatal complications would be lower than intrauterine fetal morbidity.

A landmark article by Pyeritz[19] published in the *Annual Review of Medicine* reported no aortic complications in 105 pregnancies with an aortic root less than 42 mm. The recommendation was then made that with an aortic root less than 4 mm pregnancy was well tolerated. However, as exemplified by our fourth case, there is a real risk for postpartum aortopathy that necessitates close follow up to identify the progression of the disease and catch dissection early enough to intervene. The European and Canadian guidelines report an aortic root diameter of 45 mm to be considered safe during pregnancy.[20,21]

Our first patient underwent a recommended abortion before her surgical intervention. This is a controversial recommendation. Pregnancy in a patient with Marfan syndrome requires a clear discussion between the patient and her care team regarding the risk of continuing the pregnancy as well as the risk of requiring an operation while pregnant or in the immediate postpartum period. A shared decision-making model can assist in introducing the patient's choice, describing options given the available data, and helping the patient make a decision.[22] As always, each case should be individualized.

4 | CONCLUSIONS

Marfan syndrome during pregnancy can be successfully managed with appropriate preconception counseling, antepartum care, and close postpartum follow-up. The variability regarding maternal and fetal outcomes is due in part to the wide spectrum of disease and associated comorbidities. While some standardization has been achieved regarding maternal and fetal care for those with Marfan syndrome, further research is warranted in regard to risk stratification and outcomes of those who require surgery for aortopathy during pregnancy. Utilizing a multidisciplinary approach, the risk of aortopathy can be mitigated to improve both maternal and fetal outcomes. An aortic root diameter of less than 45 mm is considered safe during pregnancy, and, the patient should be monitored with routine echocardiograms throughout the pregnancy.

ACKNOWLEDGMENTS

One of the authors, Hannah Copeland is partially supported by the National Institutes of General Medical Sciences under the National Institutes of Health under Award Number 1U54GM115428. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. Hannah Copeland is supported by the Mississippi Center for Clinical and Translational Research (NIGMS U54 GM-115428). She is a consultant for Bridge to Life. Aortic Root Valve-Sparing Repair and Risk of Dissections in Marfan Disease during pregnancy: a case series and review article.

CONFLICT OF INTERESTS

Dr Copeland is a consultant for Bridge to Life and her husband is a consultant for Syncardia Total Artificial Heart Systems.

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How to cite this article: Cottrell J, Calhoun J, Szczepanski J, et al. Aortic root valve-sparing repair and dissections in Marfans syndrome during pregnancy: A case series. *J Card Surg*. 2020;35:1439–1443. https://doi.org/10.1111/jocs.14592