# News in December 2023

## 1. Effect of Mavacamten in Women Compared With Men With Obstructive Hypertrophic Cardiomyopathy

A variety of cardiovascular diseases and their related therapies exhibit important and, sometimes, surprising sex-specific differences in clinical manifestations, disease progression, treatment efficacy, and adverse effects.[1] Women with hypertrophic cardiomyopathy (HCM) are one such group who tend to present with worse heart failure symptoms, cardiovascular outcomes, left ventricular outflow tract obstruction, and diastolic dysfunction at baseline compared with men.[2] While treatment with mavacamten, a first-in-class myosin ATPase inhibitor, has been shown to improve symptom burden, functional status, cardiac biomarker level, and degree of left ventricular outflow tract obstruction among symptomatic patients with obstructive HCM compared with placebo,[3] the potential differences in treatment effect between women and men had not been examined.

In this prespecified post hoc analysis of the blinded, randomized, placebo-controlled trial EXPLORER-HCM, the investigators compared the treatment responses to mavacamten versus placebo from baseline to 30 weeks between men and women.[4] Commensurate with previous observations, women at baseline were older and had lower peak oxygen consumption, higher NT-proBNP levels, and greater symptom burden and functional limitation as measured by the KCCQ-CSS and NYHA functional class. With respect to EXPLORER trial's composite primary outcome (≥1.5–mL/kg/min increase in peak oxygen consumption and at least one NYHA class reduction or a ≥3.0–mL/kg/min increase in peak oxygen consumption with no worsening of NYHA class), after 30 weeks of treatment, women demonstrated a treatment response similar to that in men (percentage difference on mavacamten vs placebo, 22% vs 19%, respectively; P = .759). Interestingly, although both women and men had a significant improvement in health status in response to mavacamten as measured by the KCCQ-CSS, women derived a statistically and clinically

significant greater improvement compared with men (+14.8 vs +6.1; P = .026). Women also had greater improvements in NT-proBNP levels (−1322 vs −649 ng/L; P = .0008). The rate of serious adverse events was not different between the two groups.

Overall, women with obstructive HCM treated with mavacamten in the EXPLORER-HCM trial had favorable health status outcomes. Importantly, despite consisting of a sicker cohort of patients at baseline, women demonstrated improvements in symptom burden and functional class comparable to those in men, without significantly greater adverse medication events.

**2. Neighborhood Disadvantage Tied to Higher Postpartum Depression Risk**

The researchers found that higher neighborhood disadvantage and race and ethnicity were associated with PPD in an adjusted analysis. Black individuals were more likely to have PPD, while Asian and Hispanic individuals were less likely to have PPD, compared with White individuals. There were significant associations between Neighborhood Deprivation Index and PPD that differed by race and ethnicity. The risk for PPD was the greatest overall in Black individuals and increased with neighborhood disadvantage in a dose-response manner (adjusted relative risks: quartile [Q] 2, 1.39; Q3, 1.50; Q4, 1.60). While the magnitude of the risk was lower, neighborhood disadvantage was associated with PPD among Asian and White individuals but not Hispanic individuals.

**3. Is Migraine Really Gender Specific?**

Migraine is widely considered a predominantly female disorder. Its frequency, duration, and severity tend to be higher in women, and women are also more likely than men to receive a migraine diagnosis. However, gender expectations, differences in the likelihood of self-reporting, and problems with how migraine is classified make it difficult to estimate its true prevalence in men and women.

Epidemiologists and migraine specialists discussed these apparent sex differences and the difficulties in obtaining accurate estimates of migraine prevalence in a debate session at the 17th European Headache Congress in Barcelona.

**Different Symptoms**

Headache disorders are estimated to affect 50% of the general population ; tension-type headache and migraine are the two most common. According to epidemiologic studies, migraine is more prevalent in women, with a female-to-male ratio of 3:1. There are numerous studies of why this might be, most of which focus largely on female-related factors, such as hormones and the menstrual cycle.

"Despite many years of research, there isn't one clear factor explaining this substantial difference between women and men," said Tobias Kurth of Charité – Universitätsmedizin Berlin, Germany. "So the question is: Are we missing something else?"

One factor in these perceived sex differences in migraine is that women seem to report their migraines differently from men, and they also have different symptoms. For example, women are more likely to than men to report severe pain , and their migraine attacks are more often accompanied by photophobia, phonophobia, and nausea, whereas men's migraines are more often accompanied by aura.

"By favoring female symptoms, the classification system may not be picking up male symptoms because they're not being classified in the right way," Kurth said, with one consequence being that migraine is underdiagnosed in men. "Before trying to understand the biological and behavioral reasons for these sex differences, we first need to consider these methodological challenges that we all apply knowingly or unknowingly."

Christian Lampl, professor of neurology at Konventhospital der Barmherzigen Brüder Linz, Austria, and president of the European Headache Federation,

told Medscape Medical News, "I'm convinced that this 3:1 ratio which has been stated for decades is wrong, but we still don't have the data. The criteria we have [for classifying migraine] are useful for clinical trials, but they are useless for determining the male-to-female ratio.

"We need a new definition of migraine," he added. "Migraine is an episode, not an attack. Attacks have a sudden onset, and migraine onset is not sudden — it is an episode with a headache attack."

**Inadequate Menopause Services**

Professor Anne MacGregor of St. Bartholomew's Hospital in London, United Kingdom, specializes in migraine and women's health. She presented data showing that migraine is underdiagnosed in women; one reason being that the disorder receives inadequate attention from healthcare professionals at specialist menopause services.

Menopause is associated with an increased prevalence of migraine, but women do not discuss headache symptoms at specialist menopause services, MacGregor said.

She then described unpublished results from a survey of 117 women attending the specialist menopause service at St. Bartholomew's Hospital. Among the respondents, 34% reported experiencing episodic migraine and an additional 8% reported having chronic migraine.

"Within this population of women who were not reporting headache as a symptom [to the menopause service until asked in the survey], 42% of them were positive for a diagnosis of migraine," said MacGregor. "They were mostly relying on prescribed paracetamol and codeine, or buying it over the counter, and only 22% of them were receiving triptans.

"They are clearly being undertreated," she added. "Part of this issue is that they didn't spontaneously report headache as a menopause symptom, so they weren't consulting for headache to their primary care physicians."

Correct diagnosis by a consultant is a prerequisite for receiving appropriate migraine treatment. Yet, according to a US study published in 2012, only 45.5% of women with episodic migraine consulted a prescribing healthcare professional. Of those who consulted, 89% were diagnosed correctly, and only 68% of those received the appropriate treatment.

A larger, more recent study confirmed that there is a massive unmet need for improving care in this patient population. The Chronic Migraine Epidemiology and Outcomes (CaMEO) Study, which analyzed data from nearly 90,000 participants, showed that just 4.8% of people with chronic migraine received consultation, correct diagnosis, and treatment, with 89% of women with chronic migraine left undiagnosed.

The OVERCOME Study further revealed that although many people with migraine were repeat consulters, they were consulting their physicians for other health problems.

"This makes it very clear that people in other specialties need to be more aware about picking up and diagnosing headache," said MacGregor. "That's where the real need is in managing headache. We have the treatments, but if the patients can't access them, they're not much good to them."

**4. Earlier Age at Menarche Raises Type 2 Diabetes Risk**

Earlier age at menarche is associated with type 2 diabetes risk among women younger than 65 years, according to a study published online Dec. 5 in BMJ Nutrition Prevention & Health.

Maria P. Santos, from the Tulane University School of Public Health and Tropical Medicine in New Orleans, and colleagues used data from 17,377 women (aged 20 to 65 years) participating in the National Health and Nutrition Examination Survey (1999 to 2018) to examine associations of age at menarche with type 2 diabetes and with cardiovascular disease (CVD) complications among women with diabetes.

The researchers found that 10.2 percent of women reported having type 2 diabetes. When adjusting for age, race/ethnicity, education, parity, menopause status, family history of diabetes, smoking status, physical activity, alcohol consumption, and body mass index, there was an association seen between earlier age at menarche and type 2 diabetes compared with a median age at menarche of 13 years. Earlier age at menarche among women with diabetes, was associated with stroke, but not with total CVD, in an adjusted analysis. Similarly, extremely early age at menarche (10 years and younger) was significantly associated with stroke (adjusted odds ratio, 2.66).

**5. Metabolic Disorders Mediate the Relation of Miscarriage With CVD**

AIMS

The extent to which the contribution of pregnancy loss to cardiovascular diseases can be explained by metabolic disorders is poorly elucidated but holds insights for reducing long-term cardiovascular risk. To investigate the mediating effects of hypertension, diabetes mellitus (DM), and lipoprotein metabolism disorders on the association of miscarriage and stillbirth with coronary heart disease (CHD), stroke, heart failure, atrial fibrillation, and composite outcomes.

METHODS

A total of 163 283 ever-gravid women (age 55.3 ± 7.9 years) from the UK Biobank cohort without established metabolic disorders and cardiovascular diseases were included and followed from 2007-2010 baseline until December 2020. Causal mediation analyses were used to estimate the proportion mediated.

RESULTS

Hypertension mediated 11.1% (95%CI, 3.7% to 18.5%) of the association between a history of miscarriage and incident CHD. Approximately 9.5% (4.1% to 14.8%) of the effect of recurrent miscarriages on incident CHD was via hypertension, 8.4% (2.5% to 14.3%) of effect was via lipoprotein metabolism disorders, and 1.7% (0.5% to 2.9%) of effect was via DM. 10.7%

(0.2% to 21.1%) of the effect of recurrent miscarriages on incident stroke was via hypertension. Hypertension mediated the largest proportion of effect for the atherosclerotic cardiovascular event (15.5% for a history of miscarriage and 9.4% for recurrent miscarriages), followed by lipoprotein metabolism disorders and DM.

CONCLUSION

Hypertension, DM, and lipoprotein metabolism disorders mediated the association between miscarriage and various cardiovascular outcomes in later life. In particular, hypertension mediated a large proportion of the relation between miscarriage and atherosclerotic cardiovascular disease.

**6. Sex Differences in the Clinical Presentation and Natural History of Dilated Cardiomyopathy**

BACKGROUND

Biological sex has a diverse impact on the cardiovascular system. Its influence on dilated cardiomyopathy (DCM) remains unresolved.

OBJECTIVES

This study aims to investigate sex-specific differences in DCM presentation, natural history, and prognostic factors.

METHODS

We conducted a prospective observational cohort study of DCM patients assessing baseline characteristics, cardiac magnetic resonance imaging, biomarkers, and genotype. The composite outcome was cardiovascular mortality or major heart failure (HF) events.

RESULTS

Overall, 206 females and 398 males with DCM were followed for a median of 3.9 years. At baseline, female patients had higher left ventricular ejection fraction, smaller left ventricular volumes, less prevalent mid-wall myocardial fibrosis (23% vs 42%), and lower high-sensitivity cardiac troponin I than

males (all P < 0.05) with no difference in time from diagnosis, age at enrollment, N-terminal pro-B-type natriuretic peptide levels, pathogenic DCM genetic variants, myocardial fibrosis extent, or medications used for HF. Despite a more favorable profile, the risk of the primary outcome at 2 years was higher in females than males (8.6% vs 4.4%, adjusted HR: 3.14; 95% CI: 1.55-6.35; P = 0.001). Between 2 and 5 years, the effect of sex as a prognostic modifier attenuated. Age, mid-wall myocardial fibrosis, left ventricular ejection fraction, left atrial volume, N-terminal pro-B-type natriuretic peptide, high-sensitivity cardiac troponin I, left bundle branch block, and NYHA functional class were not sex-specific prognostic factors.

CONCLUSIONS

We identify a novel paradox in prognosis for females with DCM. Female DCM patients have a paradoxical early increase in major HF events despite less prevalent myocardial fibrosis and a milder phenotype at presentation. Future studies should interrogate the mechanistic basis for these sex differences.

**7. Experiencing Racism May Increase Stroke Risk in Black Women**

Shanshan Sheehy, Sc.D., from the Slone Epidemiology Center at Boston University, and colleagues examined the association of perceived interpersonal racism with incident stroke among U.S. Black women. The analysis included 48,375 participants in the Black Women's Health Study with follow-up from 1997 through 2019.

The researchers identified 1,664 incident stroke cases, of which 550 were definite cases confirmed by neurologist review and/or National Death Index linkage. For those reporting experiences of racism in all three domains of employment, housing, and interactions with police, there was a 38 percent increase in incident stroke (95 percent confidence interval [CI], 1.14 to 1.67) and a 37 percent increase in definite cases (95 percent CI, 1.00 to 1.88) compared with that seen in women with no such racism experiences. A similar trend was seen for comparisons of women in the highest quartile of the everyday interpersonal racism score versus those in the lowest quartile

(hazard ratio, 1.14 [95 percent CI, 0.97 to 1.35] for all incident stroke; hazard ratio, 1.09 [95 percent CI, 0.83 to 1.45] for definite cases).

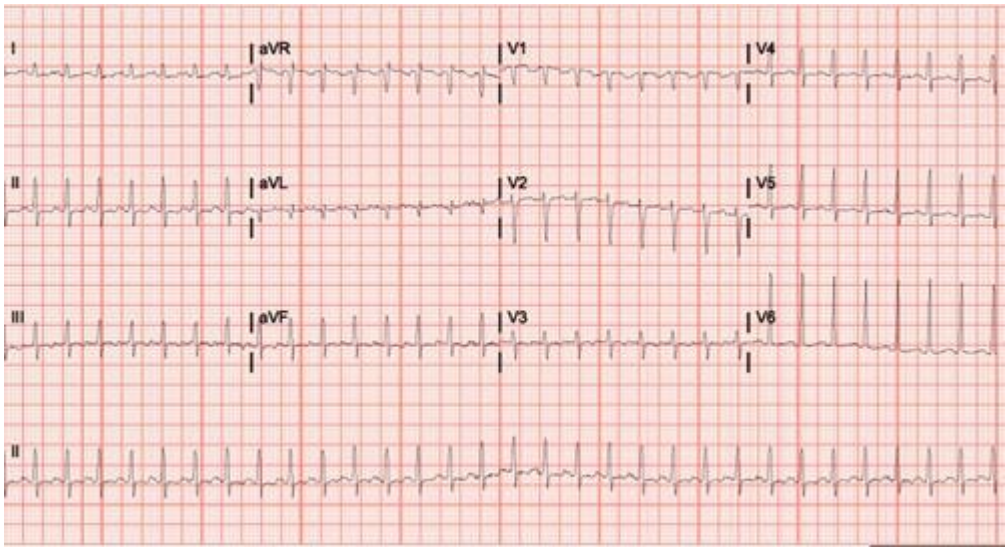
## 8. Atrial Tachycardia-Induced Cardiomyopathy in Late Pregnancy: It Is Not Always Peripartum Cardiomyopathy


### Abstract

Tachycardia-mediated cardiomyopathy is an established cause of left ventricular dysfunction. The development of cardiomyopathy depends on type, rate, and duration of tachyarrhythmia. Early recognition and treatment are critical in preventing left ventricular dysfunction and heart failure. Normal physiologic changes in pregnancy can complicate the early recognition and treatment of pathologic tachyarrhythmia.


### Case Presentation

A 26-year-old gravida 1, parity 0 woman with gestational anemia, but no personal or familial cardiac history presented to obstetric (OB) triage at 36.6 weeks complaining of emesis, dyspnea, and severe back pain. Heart rate was 193 beats/min, initial blood pressure was 122/78 mm Hg, and arterial oxygen saturation of 93% on 8-L nasal cannula. Initial respiratory rate was 42 breaths/min, but this improved to 18 breaths/min with supplemental oxygenation. On physical exam, she was gravid, in mild distress with normal mentation, and dyspneic with diminished breath sounds in bilateral bases and scattered rhonchi. Her extremities were warm with 1+ bilateral lower extremity edema. Initial laboratory evaluation was unremarkable aside from the anemia. A computed tomography angiography chest demonstrated pulmonary edema but was unremarkable for aortic dissection or pulmonary embolism. Her presenting electrocardiogram demonstrated a narrow complex supraventricular tachycardia (SVT) (**Figure 1**). Given stable blood pressure at presentation and electrocardiogram consistent with SVT, adenosine infusion was administered with no change in heart rate or rhythm. Cardiology was urgently consulted. A bedside point-of-care ultrasound revealed a severely reduced left ventricular ejection fraction (LVEF) of <10%.




**Learning Objectives**

- To clarify that CM diagnosed during pregnancy is not always peripartum and may be related to a tachyarrhythmia or other etiologies.
- To consider the use of mechanical circulatory supporting the peripartum period for cardiogenic shock as a strategy to decrease maternal mortality.
- To acknowledge that the elimination of ATs, with a combination of antiarrhythmic and radiofrequency ablation, can be curative for tachycardia-mediated CM.

## 9. **Successful Management of Fetal Torsades de Pointes and Long QT Syndrome by a Cardio-Obstetrical Team**

**Abstract**

A 32-week fetus with tachycardia and bradycardia, diagnosed with torsades de pointes, atrioventricular block, and sinus bradycardia due to a de

novo KCNH2 mutation was successfully managed by a cardio-obstetrical team. Maternal/fetal pharmacogenomic testing resulted in appropriate drug dosing without toxicity and delivery of a term infant in sinus rhythm.

**Case presentation**

A healthy 32-year-old G2P1001 pregnant subject with an unremarkable past medical history was referred at 32 weeks because of fetal hydrops and a complex fetal arrhythmia consisting of tachycardia (170-200 beats/min) and bradycardia with 2 distinct rates of 50 beats/min and 69 to 107 beats/min.

**Learning Objectives**

- To determine the differential diagnosis of tachycardia/bradycardia in the fetus.
- To recognize the fetal echo features of long QT syndrome.
- To understand how a multidisciplinary approach leads to successful management of fetal LQTS.

## 10. Intrapartum Management in Maternal Brugada Syndrome

Brugada syndrome is a genetic cardiac disease associated with increased risk of ventricular tachyarrhythmia and sudden cardiac arrest. Labor and delivery in this population poses management challenges of labor induction, analgesia, postpartum hemorrhage, and arrhythmic events. This case report describes a multidisciplinary approach to intrapartum management in maternal Brugada syndrome.

**History of presentation**

A 19-year-old G1P0 with Brugada syndrome presented to our university maternal-fetal medicine (MFM) office for transfer of care at 37 weeks 2 days gestation. Significant family history of early sudden cardiac death included

both her father, who experienced cardiac arrest around 40 years of age while driving, and her sister, deceased at 24 years of age while sleeping. Her father was diagnosed with Brugada syndrome following his accident, with genetic testing confirming SCN5A c.4172G>T (p.Gly1391Val) mutation, a variant of unknown significance (VUS). The patient herself never had any life-threatening arrhythmias. She was diagnosed with Brugada syndrome 1.5 years before pregnancy after presenting with fever due to infection and had a characteristic type I Brugada electrocardiogram (ECG) pattern (**Figure 1**). She declined implantable cardioverter-defibrillator (ICD) placement prior to pregnancy.

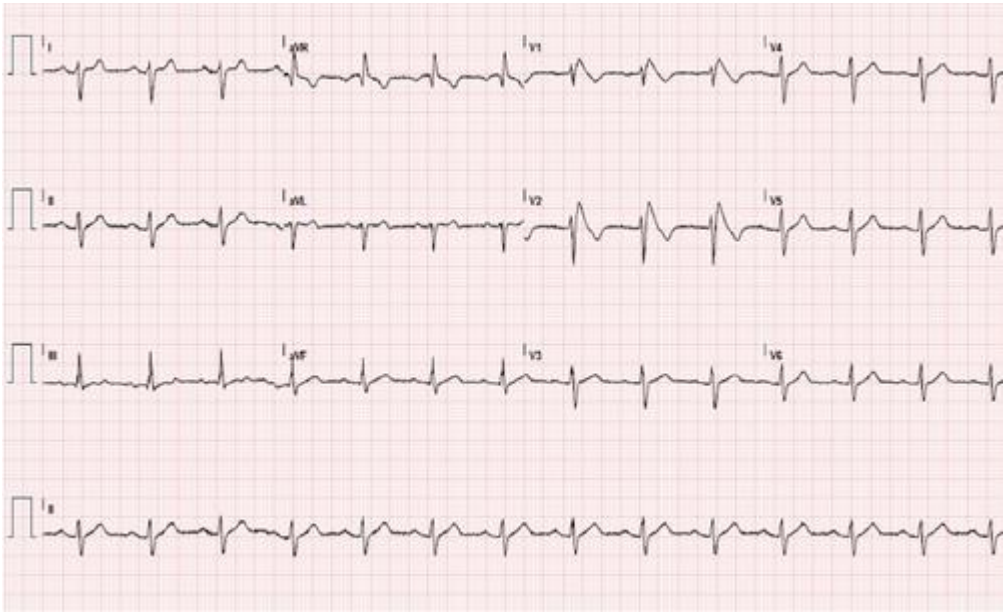


**Figure 1**

**Type I Brugada Pattern**

The patient's electrocardiogram showing characteristic type I Brugada pattern with coved ST-segments >2 mm in leads $V_1$ and $V_2$.

## Learning Objectives

- To be able to recognize triggers for ventricular arrhythmia in Brugada syndrome.

- To understand the interaction between these triggers and the needs of labor and delivery.
- To implement a plan of care to minimize risk in intrapartum management of maternal Brugada syndrome.

11. **Diagnosis and Management of Vasospastic Angina in a Young Woman Wishing to Become Pregnant**

Vasospastic angina can sometimes induce acute myocardial infarction in pregnant women, potentially endangering the lives of mother and child. We describe a case of a young woman with suspected vasospastic angina who wished to become pregnant. Vasospasm provocation testing revealed severe vasospasm, and subsequent appropriate management resulted in successful delivery.

**History of Presentation**

A 29-year-old woman presented with chest pain only at rest. The Holter electrocardiogram recorded ST-segment depression at the time of chest pain onset (**Figure 1**). A trial use of nitroglycerin sublingual tablets was effective. She was suspected to have vasospastic angina (VSA) and wished to become pregnant.

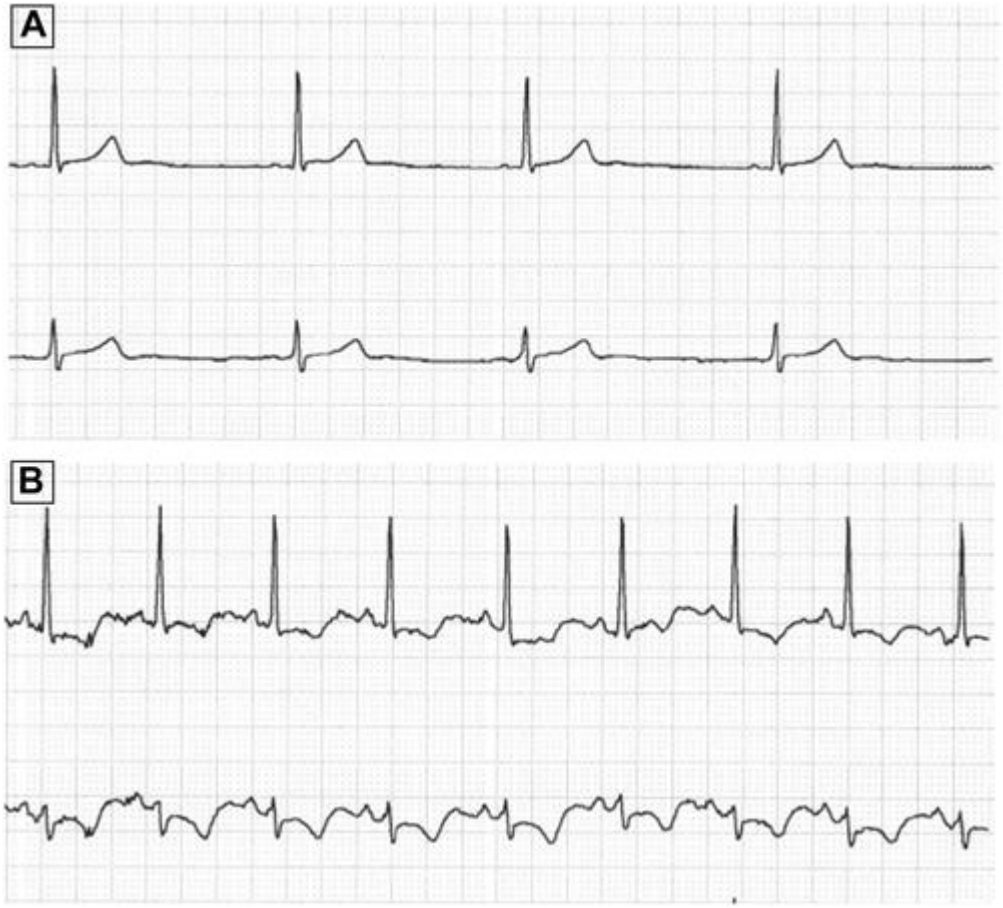



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**Figure 1**

**The Holter Electrocardiogram**

(A) Electrocardiogram under normal conditions. (B) Electrocardiogram during chest pain.

## Learning Objectives

- To understand the significance of proper diagnosis of vasospastic angina in women who wish to become pregnant.
- To learn that patients with vasospastic angina may require special management during the perinatal period.

## Past Medical History

She had no medical history.

**Differential Diagnosis**

The possible differential diagnoses included atherosclerotic coronary artery disease, coronary embolism, and spontaneous coronary artery dissection.

**Investigations**

Obstetricians and cardiologists discussed the case and decided to perform invasive coronary catheterization and vasospasm provocation testing to determine a definitive diagnosis and perinatal cardiovascular risk and, if the test was positive, to administer a calcium-channel blocker. Coronary angiography was performed via her right distal radial artery and revealed no stenosis (**Figure 2A**, **Video 1**). Vasospasm provocation testing with administration of 50 μg of acetylcholine into the left coronary artery resulted in subocclusion in the left main trunk (LMT) with chest pain, ST-segment depression in the $V_4$ to $V_6$ leads, and hypotension (**Figure 2B**, **Video 2**). An intracoronary infusion of isosorbide mononitrate and nicorandil improved the LMT occlusion, eliminated electrocardiogram changes, and increased blood pressure (**Figure 2C**, **Video 3**). She was confirmed to have severe VSA.

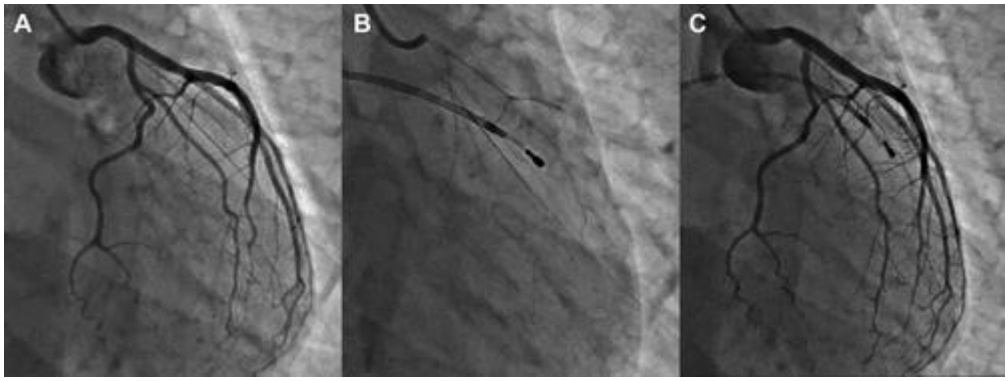


**Figure 2**

**Coronary Angiography**

(A) Baseline. (B) After administration of acetylcholine. (C) After administration of nitrate and nicorandil.

## Management

Diltiazem 200 mg daily was initiated. A few weeks later, she successfully conceived. The effectiveness of diltiazem was recognized when chest pain appeared shortly after the temporary discontinuation of diltiazem because of hyperemesis gravidarum. After the second trimester, her pregnancy progressed without chest pain, and the fetus developed well. Because spontaneous labor did not occur until full term, an induced labor was planned at the 40th week of gestation. At delivery, epidural analgesia was used to prevent vasospasm induction caused by pain stress or hyperventilation. She delivered a healthy girl without any anomaly. Oxytocin was used instead of methylergonovine for postpartum atonic hemorrhage. She had no chest pain or electrocardiogram abnormality in the peripartum period.

## Discussion

VSA is one of the important functional abnormalities of the coronary artery that can contribute to not only variant angina but also coronary microvascular disfunction, acute myocardial infarction, ventricular arrythmias, and sudden cardiac death.[1] Previous study has demonstrated that multivessel spasm is associated with poor cardiovascular prognosis, and VSA occurring in the LMT, as in this case, corresponds to multivessel spasm and is considered high risk.[2]

In pregnant women, it has been suggested that hormonal and nervous system changes that occur during pregnancy seem to trigger VSA.[3] Exposure to pain stress, hyperventilation-induced alkalosis, and administration of methylergometrine for flaccid hemostasis can further induce a vasospasm attack. Several studies have reported that VSA plays a role in acute myocardial infarction occurring in pregnant women.[4,5] Therefore, proper diagnosis and preventive management of VSA in pregnant women is considered highly significant for the safety of both mother and child.

First-line agents for VSA are calcium-channel blockers.[6,7] However, there are concerns about teratogenicity reported in animal studies.[8] Although the

safety of calcium-channel blockers for pregnant women is gradually being established, calcium-channel blockers remain unlicensed for use in pregnancy in many countries.[9] It is important to accurately assess the advantages and disadvantages of treatment in light of the certainty and severity of VSA when initiating the medication.

**Follow-Up**

After delivery, the patient continues to take diltiazem and has had no recurrence of angina attacks. The child is also growing without any disabilities. The amount of diltiazem ingested by the infant via breast milk is small and is unlikely to adversely affect breastfed infants.[10]

**Conclusions**

For women who wish to be pregnant, it is important to make a definitive diagnosis of VSA, determine its severity, and provide appropriate treatment. Under conditions of adequate prior consultation with the patient, family, obstetricians, and cardiologists, invasive catheterization including provocation test should be performed, and calcium-channel blocker administration should be considered for VSA.

## 12. Peripartum Lipid Apheresis: Novel Management of Familial Hyperlipidemia in Pregnancy

Heterozygous familial hyperlipidemia (FH) is an autosomal dominant genetic lipid disorder associated with an increased risk of early-onset coronary artery disease (CAD).[1] During pregnancy, total cholesterol (TC), low-density lipoprotein (LDL) cholesterol, and triglyceride (TG) levels increase dramatically to accommodate the necessary requirements for gestation, and in FH patients, there is a corresponding increase in their baseline abnormal lipid values. This poses a significant risk of cardiovascular events, particularly to patients with existing CAD. The most used therapies in pregnancy are bile acid sequestrants, and statins—although safe—are used less commonly. Lipid

apheresis (LA) is rarely used, and guidance surrounding apheresis protocols is limited.[2]

## Learning Objectives

- To understand the natural variation in lipid profiles during pregnancy and in patients with familial hyperlipidemia.
- To demonstrate the safety of concurrent statin, lipid apheresis, and fenofibrates during pregnancy.

## History of Presentation

A 32-year-old woman presented to the cardiology clinic for preconception counseling. She was managed on a PCSK9 inhibitor (evolocumab), rosuvastatin, and ezetimibe. Given her desire for pregnancy, she wanted to discuss therapeutic options and management of her FH during pregnancy. At the time of presentation, her vitals and examination findings were within normal limits, and she was maintaining an active lifestyle with no symptoms.

## Past Medical History

The patient's medical history was relevant for heterozygous FH, chronic hypertension, prediabetes, polycystic ovary syndrome, and depression. FH was diagnosed 3 years before presentation with routine screening after her 27-year-old brother experienced a myocardial infarction. A coronary computed tomography angiography demonstrated diffuse 3-vessel CAD. A subsequent treadmill stress test showed a blunted heart rate response and high-risk electrocardiogram features, which prompted left heart catheterization. Catheterization revealed mid-distal 90% left anterior descending artery and 80% proximal first obtuse marginal stenosis; she underwent percutaneous coronary intervention with stenting to both vessels with no residual stenosis.

Before catheterization, her lipid panel results were notable for LDL cholesterol of 350 mg/dL, TG of 235 mg/dL, and TC of 500 mg/dL. Postcatheterization, she was started on evolocumab 140 mg biweekly, ezetimibe 10 mg daily, and rosuvastatin 40 mg daily, with improvement in lipids to LDL cholesterol of 63 mg/dL, TG of 68 mg/dL, and TC of 150 mg/dL. She continued dual antiplatelet therapy for 1 year post–percutaneous coronary intervention and lisinopril 2.5 mg daily for hypertension.

**Investigations**

Before conception, she underwent repeat exercise stress treadmill test with above-average functional capacity and no evidence of active ischemia at peak heart rate.

**Management**

The case was discussed between the maternal-fetal medicine, cardiology, and transfusion medicine departments. After extensive preconception counseling, the decision was made to pursue LA to achieve an average LDL cholesterol of 150 to 200 mg/dL during pregnancy. Additionally, she was transitioned to moderate-intensity pravastatin at the time of a positive pregnancy test result.

LDL apheresis was performed using the Liposorber LA-15 system (Kaneka Pharma America LLC) device to selectively remove LDL cholesterol, very-low-density lipoprotein cholesterol, and lipoprotein(a) every 2 weeks, for a total of 15 sessions. In the first trimester of pregnancy, 3,600 mL of total plasma volume was processed (equal to 1.5 times the patient's plasma volume). During her second trimester, lipid levels elevated despite regular apheresis, likely because of increased placental production and a 30% to 50% increase in total plasma volume of pregnancy. Thus, total plasma volume was adjusted to a maximum of 5,000 mL of plasma processed in the third trimester with appropriate reduction in LDL levels (see **Figure 1**). Estriol, a placental hormone, was also assessed during the second trimester as a proxy for estrogen and progesterone levels to ensure appropriate levels in the setting of apheresis.

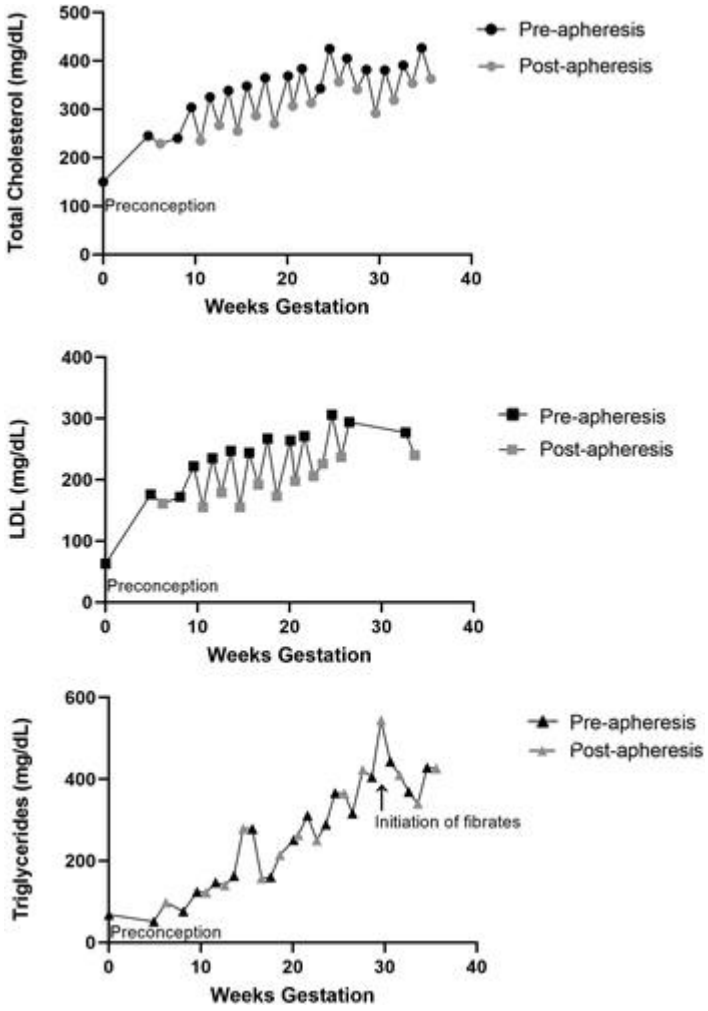


**Figure 1**

**Lipid Profile During Pregnancy**

The impact of lipid apheresis on (top) total cholesterol, (middle) low-density lipoprotein cholesterol, and (bottom) triglycerides.

At 29 weeks gestation, TG levels increased to 544 mg/dL. Given concern for the development of pancreatitis, she was initiated on fenofibrate with up-titration to 144 mg daily with improvement in TG levels (**Figure 1**).

Initially, the patient transitioned to labetalol before conception; however, it was ultimately discontinued in the first trimester because of lightheadedness. She was maintained on aspirin 81 mg daily for CAD and pre-eclampsia prophylaxis. She did not require initiation of antihypertensives during

pregnancy. Fetal status was monitored using serial growth ultrasounds and nonstress testing.

**Follow-Up**

In light of difficult-to-control LDL cholesterol despite apheresis and rising TG, the decision was made for early-term induction at 37 weeks. She underwent induction of labor with uncomplicated spontaneous vaginal delivery of a healthy baby weighing 3,490 g (89th percentile) with Apgar scores of 9 and 8 at 1 and 3 minutes, respectively. Postpartum, she was transitioned back to evolocumab and high-dose rosuvastatin with plans to pursue breastfeeding.

## 13. **Ultrasound-Guided Mechanical Thrombectomy Without Radiation for Deep Vein Thrombosis in Early Pregnancy**

**Patient 1**

A 30-year-old pregnant woman (9 weeks gestation) presented to the emergency department for severe hyperemesis (>20 episodes/day). The patient's obstetric history (gravida 5 para 2) included prior hyperemesis gravidarum, hemolysis, elevated liver enzymes and low platelets syndrome, and bilateral nephrolithiasis requiring nephrostomy tubes. She had no prior history of venous thromboembolism (VTE). On hospital day 3, the patient experienced severe left lower extremity (LLE) swelling and pain. At rest, she reported a pain level of 7 on a 10-point scale; however, pain escalated to 10 when ambulatory.

**Learning Objectives**

- To understand various considerations involved in DVT treatment during pregnancy.
- To recognize a method of interventional DVT treatment completed without radiation.

- To highlight the importance of collaboration with obstetrics and ultrasound departments.

Duplex ultrasound (DUS) of the LLE revealed noncompressible common femoral vein (CFV), femoral vein (FV), and popliteal vein (PV) consistent with acute deep vein thrombosis (DVT). After initiating anticoagulation, the patient's symptoms did not resolve as expected. The cardiology consultant discussed with the obstetrics team conservative management versus escalation of care. The head of the echocardiography department and lead sonographer reviewed the case to evaluate the feasibility of an interventional procedure without exposing the fetus to radiation. After discussing all options with the patient, the decision was made to proceed with mechanical thrombectomy (MT) for prompt obstruction reduction.

Ultrasound-guided access was obtained via the left PV. Using a 0.035-inch J wire with an intravascular ultrasound (IVUS) catheter, the wire was placed into the inferior cavoatrial junction. The placement of the catheter was confirmed with echocardiography. The wire was then exchanged, and the distal end was marked on the table to manage its position. The sheath at the PV was exchanged for the 16-F thrombectomy sheath. Under ultrasound guidance, the ClotTriever thrombectomy catheter (Inari Medical) was delivered to the confluence of the left common iliac vein (CIV). **Figure 1** exemplifies echocardiographic monitoring of the catheter during inferior vena cava (IVC) pullback. A total of 7 passes were made from the left CIV to PV with removal of thrombus (**Figure 2A**). IVUS was repeated after the fifth pass, and residual thrombus was detected within the external iliac vein. Subsequently, 2 additional passes were made with higher placement of the coring element, with minimal thrombus removed on the final pass. Agitated saline was injected into the sheath demonstrating antegrade flow on echocardiography in the FV, CIV, and IVC. No radiation or contrast was used throughout the case. Postprocedure treatment included enoxaparin 1.5 mg/kg/d, LLE compression, and limb elevation. The patient had improvements in pain and

swelling after the procedure (**Figure 3**). Once the hyperemesis was controlled, the patient was discharged home 10 days later.

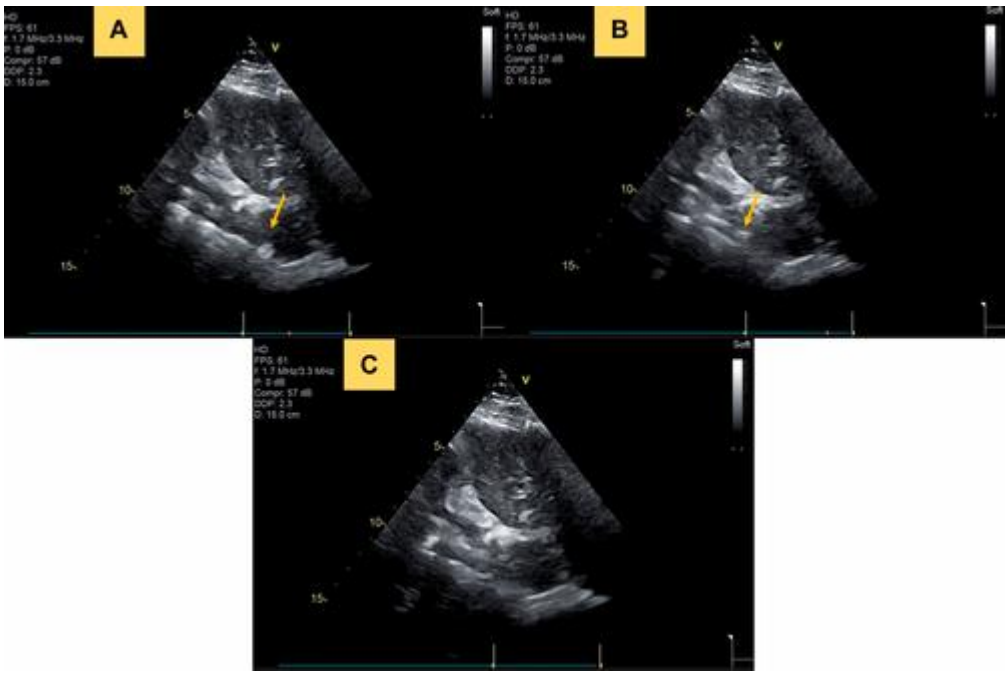


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**Figure 1**

**Echocardiographic Catheter Monitoring**

(A to C) Serial images show the pullback of the thrombectomy catheter through the inferior vena cava.

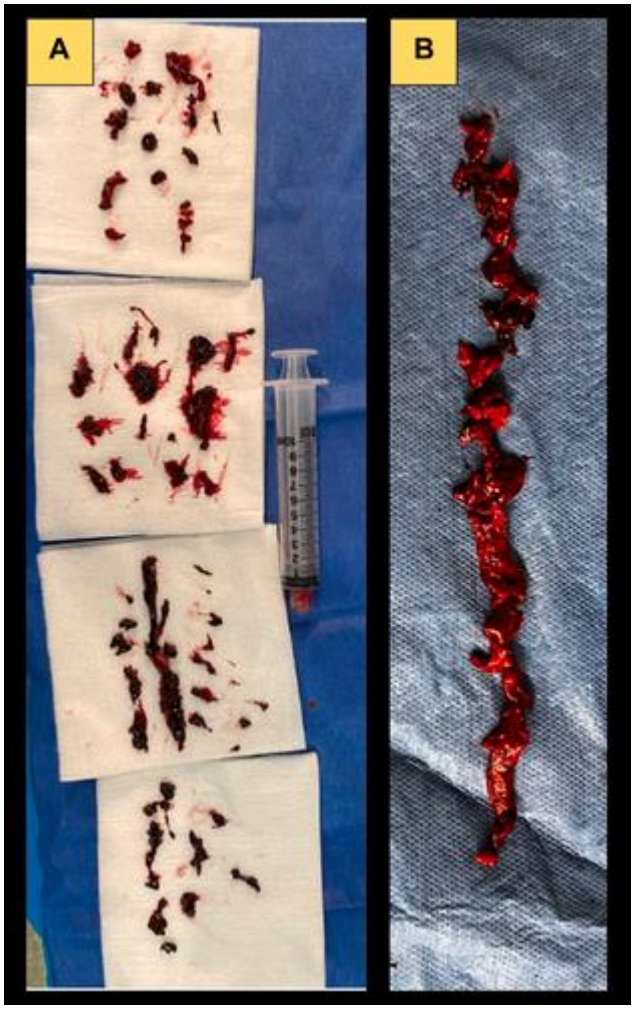


**Figure 2**

**Thrombi Extracted**

Images show the deep vein thrombi removed following mechanical thrombectomy procedures completed for (A) patient 1 and (B) patient 2.

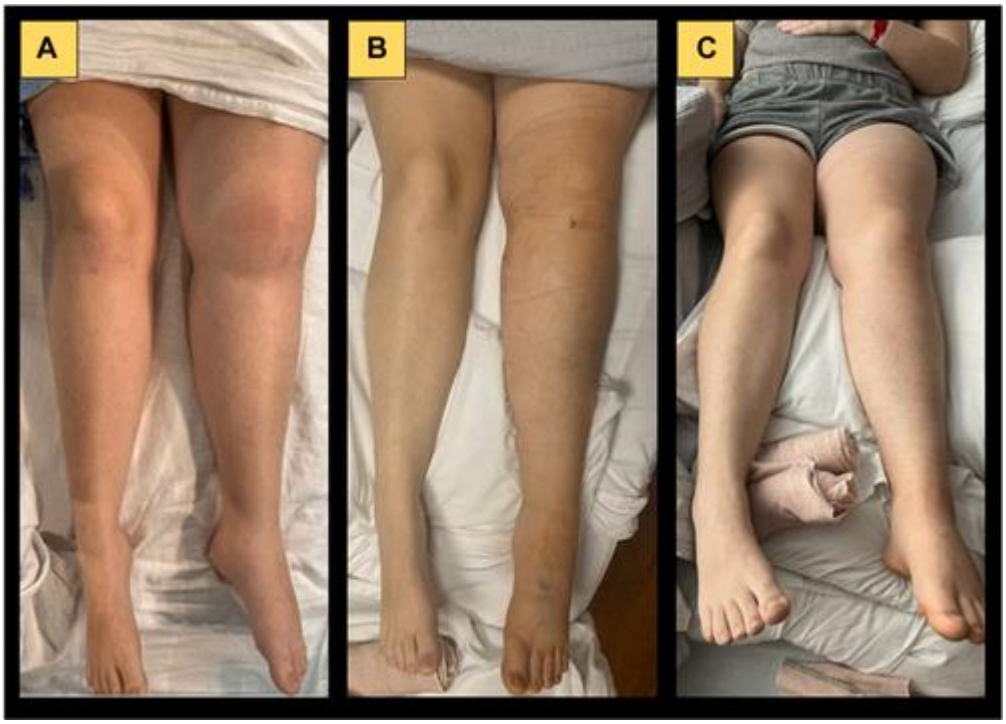


**Figure 3**

**Limb Images**

Photographs demonstrate an improvement in left limb swelling in a pregnant patient with deep vein thrombosis from (A) prethrombectomy to (B) 24 hours after the procedure and (C) postprocedural day 6.

Six months postprocedure, the patient developed preeclampsia and underwent a transverse c-section at 35 weeks. Partial compression of the CFV and FV was noted on DUS at the time of delivery. Shortly after birth, the limb returned to near normal, and reported symptoms were limited to a heavy sensation when walking. A repeat venous DUS was ordered at the 1-year follow-up and demonstrated nonocclusive chronic DVT in the left CFV and FV with no significant change from the time of delivery. The patient denied any LLE swelling or bulging varicose veins but had some LLE discomfort near the groin area, which could be related to mild post-thrombotic syndrome. The patient reported symptoms consistent with pelvic congestion syndrome, including recurrent hemorrhoids, dyspareunia, chronic pelvic discomfort, and pregnancy-related pelvic varicosity, which had not resolved.

**Patient 2**

A 27-year-old pregnant woman presented at 13 weeks gestation with a 2-day history of acute right lower extremity (RLE) edema extending from the proximal aspect through the level of the foot. The patient had no significant previous obstetric (gravida 2 para 1), medical, or surgical history. The obstetrics department consulted the vascular surgery department directly for further assessment.

Laboratory testing revealed severely elevated D-dimer. DUS imaging revealed compression of the right CFV and FV, with Doppler waveform analysis indicating a unilateral proximal occlusion in the right iliofemoral segment (**Figure 4**). Based on this imaging, IVC occlusion could be ruled out. Intervention was recommended using MT without adjunctive thrombolytics.

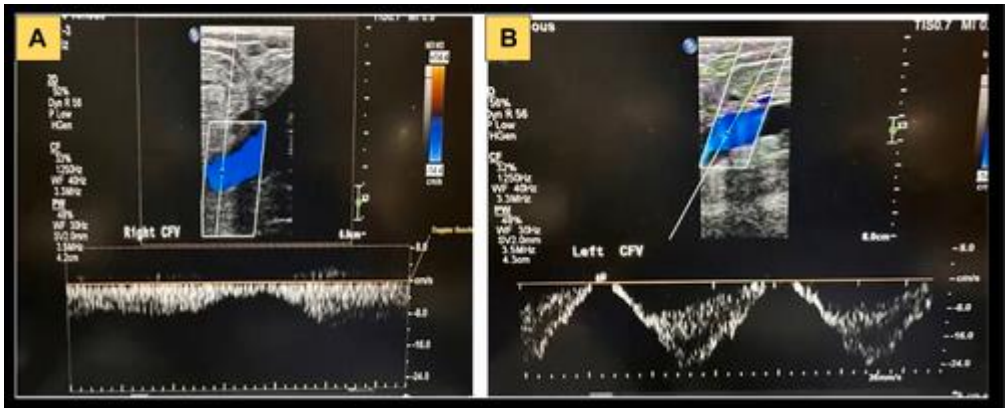


**Figure 4**

**Duplex Ultrasound**

Diagnostic images show the difference in lack of spontaneity and phasicity in (A) the right common femoral vein (CFV) vs (B) the left CFV, indicating proximal deep vein thrombosis of the right lower extremity in the iliofemoral segment.

Following the preferred technique, with the patient in prone position, ultrasound-guided access was obtained via the PV. A wire was then advanced into the IVC as confirmed per extravascular DUS. Next, as shown in **Video 1**,

IVUS investigation was completed before the thrombectomy. Serial dilation of the access site was performed for insertion of the 13-F thrombectomy sheath. The ClotTriever BOLD thrombectomy catheter was used from the IVC to the PV access site. Four passes of the device were conducted in total, with the coring element rotated 90° for each pass, extracting a significant amount of acute and subacute thrombus (**Figure 2B**). Thrombus was cleared off on the back table after each pass until there was a clean pass of the device. The postprocedural IVUS confirming the clearance of thrombus is shown in **Video 2**. No fluoroscopy or contrast was used during the procedure.

The patient was hospitalized for 2 days after the procedure for monitoring. There were no procedure-related complications. Anticoagulation with enoxaparin 1 mg/kg twice daily was initiated following the procedure and continued throughout the pregnancy. RLE compression stockings were recommended in addition to leg elevation whenever possible. The patient went on to deliver a healthy baby via spontaneous vaginal birth at 38 weeks gestation. RLE DUS completed 6 months postprocedure was negative for DVT or residual venous occlusion.

## Discussion

In both cases, collaboration with the obstetrics department and the ability to offer non–radiation-based intervention was critical to the care of each patient. Additionally, experienced sonographers with advanced understanding of DUS interrogation supported treatment.

Guidelines unanimously recommend low-molecular-weight heparin for VTE treatment during pregnancy.[1-3] Patient 1 had 2 young children at home, and resolution of symptoms caused by venous occlusion was prioritized. Similarly, patient 2 experienced bothersome symptoms caused by DVT with a more proximal iliac component based on waveform analysis. In each case, guideline-directed therapy was inadequate, and procedural risks were balanced with the patients' needs. The method of endovascular intervention

was selected to provide the lowest possible procedural and teratogenic risk given the unexpected nature of pregnancy.

Outcomes of the ClotTriever Outcomes registry demonstrated the safety and effectiveness of MT with the ClotTriever System.[4] The device-related serious adverse event rate through 30 days was 0.4%, and immediate significant improvements in pain and edema were sustained through 6 months.[4] MT was favored over catheter-directed or pharmacomechanical thrombolysis because it can be performed without thrombolytics, as guidelines recommend limiting the use of thrombolytics to cases of limb- or life-threatening VTE in pregnancy.[5]

Additionally, the period from 8 to 15 weeks postconception is the vulnerable period for cognitive development. During this period, radiation exposure may affect the growth of the fetus and also result in severe intellectual disability.[6] Although interventional DVT procedures can be done with low radiation, it was uncertain if the patients would require other procedures during pregnancy, especially given the complex obstetric history of patient 1. Further, the iodine-based contrast that is typically used for endovascular approach crosses the placenta, and well-controlled studies evaluating the teratogenicity are limited.[7] Generally, intravenous administration of low osmolar contrast has not been shown to adversely affect fetal thyroid function, and it is not routinely withheld during pregnancy.[7] Because the long-term effects of contrast administration during pregnancy are not fully understood, an abundance of caution was used in each case.

## Conclusions

Endovascular intervention in pregnancy is a complex clinical decision that requires careful consideration of patient-specific risks and benefits. In each case, intervention was appropriate based on the patient's clinical and social status. Mechanical thrombectomy was completed without exposing the mothers and fetuses to thrombolytics, contrast, or radiation. There were no related safety concerns for the patients or infants during follow-up.

## 14. **Peripartum Spontaneous Coronary Artery Dissection**

A 41-year-old Black female patient presented to the emergency department of Abidjan Heart Institute on March, 31, 2023, with acute chest pain and shortness of breath 1 week after her fourth uncomplicated spontaneous vaginal delivery of a healthy baby. She complained of 6 hours of crushing central chest pain with posterior radiation, 7 of 10 (visual analog scale). She had no significant past medical history or cardiovascular risk factors except for gestational hypertension. On exam, heart rate was 104 beats/min, blood pressure 165/100 mm Hg, and saturation 98% on room air. Pulmonary and cardiac auscultation was unremarkable. No pain or edema was found in her lower extremities.

### Learning Objectives

- To be able to make a differential diagnosis of chest pain syndromes during pregnancy and peripartum period.
- To plan and decide on the management strategies for peripartum SCAD.

## 15. **Managing High Cardiac Output Failure in a Patient With Hereditary Hemorrhagic Telangiectasias During Pregnancy**

### History of Presentation

A 39-year-old primigravida female patient with known hereditary hemorrhagic telangiectasia (HHT) presented at 32 weeks' gestation with progressive lower extremity swelling, shortness of breath, and epistaxis. The patient's prenatal course was complicated by increasing episodes and severity of epistaxis requiring packed red blood cell transfusions during her third trimester despite topical treatments. On clinical examination, the patient was tachycardic with

evidence of +3 pitting edema to the abdomen, jugular venous distention, and a new oxygen requirement.

**Learning Objectives**

- To recognize the signs of high CO failure in the setting of pregnancy.
- To understand how liver AVMs in HHT may result in high CO failure.
- To understand the treatment options for high CO failure in HHT.

16. **Successful Pregnancy After Cardiac Arrest in a Woman With Severe Coronary Vasospasm**

**Case Presentation**

A 37-year-old gravida 5, para 3 female presented to the cardio-obstetrics clinic with an unplanned pregnancy 6 weeks after hospitalization for severe coronary vasospasm complicated by ventricular fibrillation (VF) and cardiac arrest.

**Learning Objectives**

- To emphasize the importance of preconception counseling in women admitted with an acute coronary syndrome.
- To identify risk factors for maternal cardiovascular morbidity and mortality in pregnancy in women with acquired ischemic heart disease.
- To review delivery planning considerations in the case of prior maternal cardiac arrest from ventricular arrhythmia.

Before this event, her medical history was notable for smoking, chronic hypertension, and multiple psychiatric comorbidities. She had 3 prior

uncomplicated pregnancies with healthy children born by normal spontaneous vaginal deliveries. There was no family history of premature coronary disease, congenital cardiac conditions, or sudden cardiac death.

Approximately 6 weeks before presentation, she complained of acute burning chest pain and experienced a witnessed cardiac arrest at home requiring cardiopulmonary resuscitation, including intravenous amiodarone and defibrillation for VF. Her initial electrocardiogram (**Figure 1**) showed sinus tachycardia without diagnostic ST-segment changes, QTc prolongation, pre-excitation, or pathologic Q waves. An echocardiogram demonstrated normal biventricular size and function, with normal wall thickness and no segmental wall motion abnormalities or significant valve disease. Emergent coronary angiography was performed with immediate spasm of the right femoral artery access site. On engaging the left main artery, the patient went into ventricular tachycardia, which rapidly deteriorated into refractory VF requiring 8 defibrillations, amiodarone, and lidocaine. A single shot of the left coronary system was obtained showing sequential stenosis of the left anterior descending and left circumflex arteries (**Figure 2**). She was started on a nicardipine infusion for severe vasospasm. Angiography was aborted without administering intracoronary nitroglycerin or injecting the right coronary artery. Her mental status recovered, and a subcutaneous implantable cardioverter-defibrillator (ICD) was inserted. She was transitioned to amlodipine and had no further ventricular arrhythmia. She was discharged on aspirin, a high-intensity statin, lisinopril, amlodipine, and nitroglycerin as needed. Smoking cessation was strongly recommended, but contraception was not discussed before discharge.

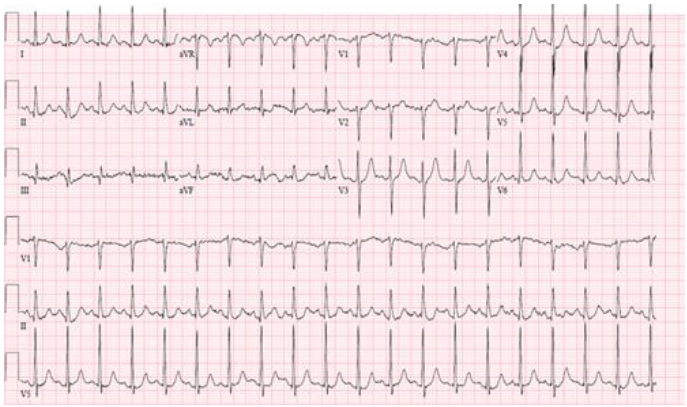

**Figure 1**

**Electrocardiogram on Initial Presentation for Acute Coronary Syndrome and Cardiac Arrest Showing No Evidence of an ST-Segment Elevation Myocardial Infarction**

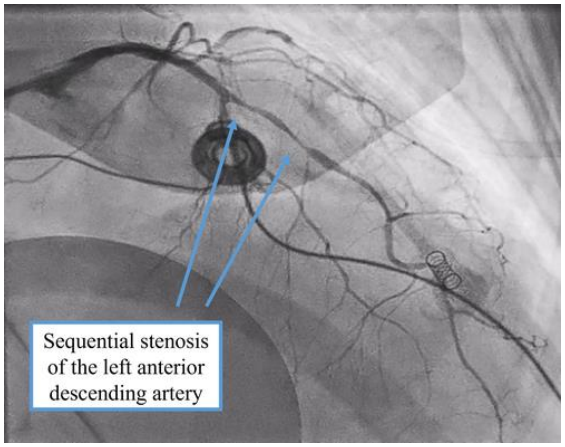


**Figure 2**

**Coronary Angiogram Showing Coronary Vasospasm**

Before her initial cardio-obstetrics visit, we reviewed her recent coronary angiogram with the interventional cardiologist who had performed her procedure and an outside expert for evidence of spontaneous coronary artery dissection (SCAD). Although coronary vasospasm was considered the most likely explanation for her presentation, our team could not exclude atherosclerosis because her right coronary artery had not been evaluated. Her vital signs were notable for pulse of 95 beats/min, blood pressure of 139/69 mm Hg, and oxygenation of 100% on room air. On physical examination, she was well kept and euvolemic, with normal cardiac examination findings. The

patient was counseled that pregnancy was not advisable because of a high risk for a maternal cardiac event, including ventricular arrhythmia and acute coronary syndrome (ACS). The estimated maternal cardiac event rate using the CARPREG II (Cardiac Disease in Pregnancy II) risk prediction score was at least 15% for a prior history of cardiac event or arrhythmia (3 points) and more than 40% if there was undiagnosed atherosclerosis (additional 2 points).[1] The patient did not want to consider termination. Her case was discussed in a multidisciplinary team, including cardiology, electrophysiology, anesthesia, and maternal-fetal medicine (MFM). Her lisinopril and statin were discontinued for potential teratogenicity. Her amlodipine was up-titrated, and nitroglycerin was continued. Despite these adjustments, she continued to report intermittent chest burning, fatigue, dyspnea, dizziness, and sweating. A coronary computed tomography angiogram was obtained and excluded obstructive atherosclerosis (**Figure 3**). There was no ventricular arrhythmia on weekly ICD interrogations through pregnancy.

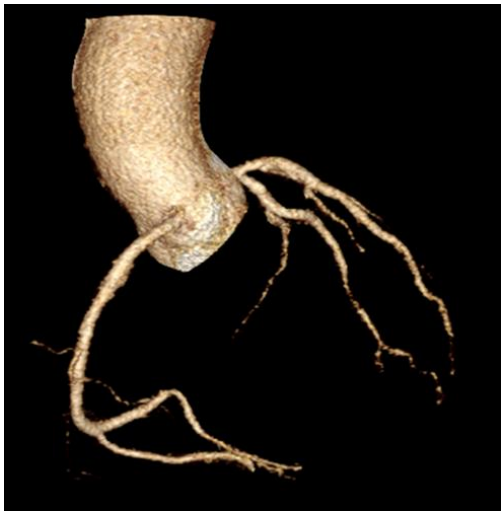

**Figure 3**

**Cardiac-Gated Coronary Computed Tomography Angiogram Confirming No Obstructive Coronary Artery Disease**

The patient was planned for induction of labor at 39 weeks but experienced escalating psychological distress regarding her physical fitness for pregnancy

toward the end of her third trimester and had multiple emergency visits for chest pain. After an extensive shared decision-making discussion with the patient, her family, and our multidisciplinary team, she was brought in for induction of labor at 37 weeks. She continued her amlodipine and long-acting nitrate through delivery. Her ICD was disabled during labor, and external defibrillator pads were placed. A healthy female infant was delivered via uncomplicated vaginal delivery with induction. She agreed to a progesterone-only contraceptive at discharge with plans for outpatient tubal ligation.

## 12. Cardio-Obstetrics: A Blueprint for Improving Maternal Cardiovascular Health Across the Life Course

### Background

Cardiovascular disease (CVD) is the leading cause of pregnancy-related death in the United States, and rates of maternal morbidity and mortality have been increasing over the past several decades.[1] Contributory factors to this alarming trend include advancing age as well as a higher burden of cardiovascular risk factors and medical comorbidities such as obesity in individuals who become pregnant. Additionally, advances in the care of patients with previously fatal congenital heart diseases and improvements in guideline-directed medical therapy of other cardiovascular (CV) conditions have led to an increasing population of child-bearing age individuals with overt CVD. Taken together, these events have led to an increasing need for clinicians who are knowledgeable and skilled in the management of cardio-obstetrics, the field at the intersection of pregnancy and CVD.

Whereas in the most severe CV conditions pregnancy may be associated with extremely high rates of morbidity, making it prohibitive, in the majority of cases individuals with CVD or CV risk factors can be safely guided through pregnancy with mitigation of risk for adverse maternal and fetal outcomes. Unfortunately, at the present time the need for access to cardio-obstetrics care teams outstrips the number of clinicians in cardiology and other specialties such as family medicine, obstetrics, and maternal-fetal medicine who have the requisite knowledge base and comfort level required to

appropriately manage CVD in pregnancy and beyond. As such, the field of cardio-obstetrics is evolving to foster improvements in maternal cardiovascular care across the spectrum of pregnancy, not only during pregnancy but also extending from preconception through the fourth trimester and across the lifespan to include broader aspects of reproductive health for individuals with CVD or CV risk factors and mitigation of long-term CVD risk associated with adverse pregnancy outcomes.

In line with the goals of the American College of Cardiology (ACC) to foster transformative cardiovascular care and improve heart health for diverse populations, in 2019 the ACC Cardio-Obstetrics Work Group developed as a grassroots, member-led initiative. This group established a tripartite mission: 1) to serve as a global cardio-obstetrics home for ACC members and the broader house of medicine; 2) to develop and disseminate educational opportunities for faculty, fellows-in-training (FITs), and CV team members; and 3) to support advocacy initiatives related to improving maternal health and the provision of equitable, high-quality care.

To inform educational initiatives and develop appropriately targeted programs, sentinel work from the cardio-obstetrics workgroup included surveying a diverse group of ACC members including practicing physicians, CV team members, and FITs to ascertain the current state of training, comfort level of care provision, and perceived needs related to cardio-obstetrics.[2] We found that 66% of practicing cardiologists and 94% of CV team members had received no formal didactics on cardio-obstetrics during their training. Additionally, significant gaps were noted between the level of comfort a clinician has managing the same CV condition in a nonpregnant patient compared with a pregnant patient. The largest gaps were noted in medication safety, management of aortopathies, acute coronary syndromes, and valvular heart diseases. These reported gaps were even more prominent among the responses from FITs and CV team members. Based on the results of this survey, a landmark 5-part focus seminar series in the Journal of the American College of Cardiology was published covering all aspects of cardio-obstetrics from preconception risk assessment, management of acquired and

congenital conditions, contraception, and diagnostic testing, among many other topics.[1,3-6] Additional educational offerings that have arisen to fill the gaps identified by the survey include the development and launch of a dedicated full-day ACC Cardio-Obstetrics Continuing Medical Education course, now an annual event, as well as quarterly Cardio-Obstetrics journal club webinars. Efforts to integrate cardio-obstetrics topics into subspecialty society conferences such as Society for Cardiovascular Angiography and Interventions and collaborations to provide cross-discipline webinars have also been well received.

In 2023, in recognition of the importance of this field and a desire to foster its advancement, the ACC Board of Trustees approved the transition from a cardio-obstetrics work group into the Reproductive Health and Cardio-Obstetrics Section. Expansion of the Section's name and scope beyond cardio-obstetrics was a deliberate recognition of the need to develop clinical and educational directives related to the intersection of CV and reproductive health issues in both men and women. The Reproductive Health and Cardio-Obstetrics Section now serves as a home for: 1) educational initiatives and resource development targeted to all levels of learners from introductory to advanced; 2) involvement in advocacy; and 3) cross-societal collaborations. Given the intersectional nature of the fields, a key component to furthering educational initiatives, in particular, is the promotion of integrated education for trainees both within and outside of cardiology in fields such as primary care, emergency medicine, anesthesia, obstetrics/gynecology, and maternal-fetal medicine. As a complement to clinician education, a primary directive of the section is to promote the dissemination of education to patients in continued conjunction with existing ACC resources such as CardioSmart, and the development of novel new programs.

Elevating these important focus areas of reproductive health and cardio-obstetrics to the Section level within the ACC is also an acknowledgement of the profound disparities in maternal outcomes experienced by Black and American Indian/Alaskan Native populations, and a commitment to improving health equity for these groups and others such as rural

populations. The medical risks faced by pregnant people with, and at risk for, CVD escalated rapidly in many areas after the Supreme Court ruling in Dobbs vs Jackson Women's Health Organization. This ruling has already exerted, and will continue to exert, profound effects on reproductive health care rights and adverse maternal CV health outcomes, heightening the need for education, advocacy, and protective legislation in this country. The Section has a dedicated Advocacy work group that liaises closely with ACC legislative staff to provide timely guidance for government groups on topics related to reproductive health and cardio-obstetrics. As the Reproductive Health and Cardio-Obstetrics Section continues to develop and expand, we encourage clinicians and researchers to join and participate in our efforts to further knowledge, education, and advocacy to improve outcomes for the diverse patient populations this Section represents.

13. **Successful Management of Pregnancies Complicated by Type B Aortic Dissections (Both Acute and Chronic)**

**Case Presentation**

**Case 1**

A 27-year-old patient (G3P2002) at 28 weeks presented to the emergency department with acute severe low back pain.

**Learning Objectives**

- To identify high-risk periods for aortic dissection through pregnancy.
- To manage chronic TBADs during pregnancy and delivery.

Her medical history consisted of genetically confirmed Marfan syndrome (FBN1 c.1090C>T pathogenic variant) and family history of mother and brother who had aortic dissections, the details of which were unknown. Her systemic signs included tall stature, arachnodactyly, and severe scoliosis requiring surgery. She did not have a history of ectopia lentis, and, notably,

her aortic dimensions were consistently noted to be normal on echocardiogram and cross-sectional imaging studies. Although the nature of the preconception counseling that she previously received was not known at the time of her presentation, her 2 prior pregnancies were well tolerated without recorded elevated blood pressures or changes in the aortic dimensions. She was treated with metoprolol, which she continued during the current pregnancy. A first-trimester echocardiogram demonstrated effacement of the sinotubular junction with normal aortic root, ascending aortic arch, and transverse aortic arch diameters measuring 3.2 cm ($z = 0.26$), 2.8 cm, and 2.9 cm, respectively, all unchanged from prior measurements. Her most recent aortic computed tomography with angiography (CTA) showed a maximal descending aortic diameter of 2.1 × 2.0 cm.

Her pregnancy was uncomplicated until 28 weeks when she was admitted to an outside hospital with acute severe back pain. A chest CTA was obtained, revealing a type B aortic dissection (TBAD), which was noted to extend into the left subclavian artery and both common iliac arteries (**Figure 1**). The measured descending thoracic aortic diameter was 2.6 × 2.5 cm. The left renal artery was noted to come off the true lumen, whereas the right renal artery was supplied by the false lumen. The aortic root and ascending aortic dimensions were again noted to be of normal diameters, which were unchanged from prior CTA. She was started on esmolol infusion and was transferred to our hospital.

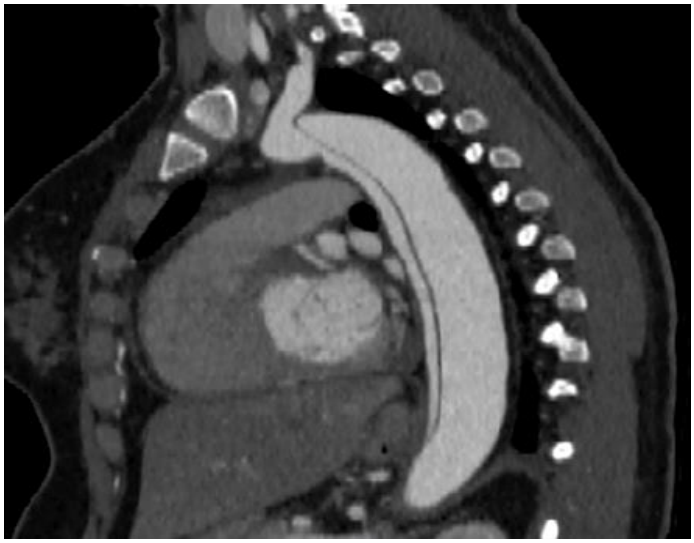

**Figure 1**

**Acute Type B Aortic Dissection**

Acute type B aortic dissection diagnosed by computed tomography angiogram after the patient presented at 28 weeks' pregnancy with acute chest pain radiating to her back.

On arrival at our hospital, she was admitted to the intensive care unit. Her hemodynamic status was normal with normotension and no laboratory or clinical evidence of end-organ malperfusion. A multidisciplinary team consisting of experts in the fields of cardio-obstetrics, maternal fetal medicine, obstetrical anesthesia, vascular and cardiac surgery, and intensive care unit was assembled and continued to provide her care throughout the hospitalization. Although her initial blood pressure was 113/77 mm Hg, values increased to >140/80 mm Hg within several hours and intravenous nifedipine was added, with subsequent need to add hydralazine, which provided better blood pressure control. Laboratory studies revealed proteinuria, prompting the diagnosis of preeclampsia.

In the following 2 weeks, she continued to require high dose of triple antihypertensive therapy with systolic blood pressure values ranging between 130 and 140 mm Hg. It was ultimately decided that delivery at 30 + 2 weeks would best balance the risks of fetal prematurity and the unique maternal medical needs. Due to the history of prior cesarean deliveries, it was decided to proceed with a repeat cesarean delivery under general anesthesia, which was performed during daytime in the middle of the week and was uncomplicated. Neuraxial anesthesia was not an option given patient's history of scoliosis and spinal fusion with Harrington rods from T10-S1. She also underwent elective bilateral salpingectomy because she did not desire any future pregnancies. Postpartum blood pressure was easier to control with down-titration of the blood pressure medications, and additional CTA of the aorta showed stable findings. She was observed for 7 days and was discharged home on losartan and metoprolol therapy.

Approximately 1 year after her discharge, an uncomplicated staged repair of the descending aortic dissection was performed, and she is currently followed regularly at the genetic aortopathy and vascular surgery clinics. Her blood pressure continues to be well controlled. Two of her 3 children were confirmed to have an identical FBN1 pathogenic mutation to hers.

**Case 2**

A 27-year-old patient (G1P1) with prior medical history consisting of postpartum types A and B aortic dissection presented for preconception counseling.

Prior to her presentation, she had completed an uncomplicated, well-tolerated pregnancy with vaginal delivery at 39 weeks. At that time her medical history was unremarkable, and she had no notable features or symptoms suggestive of connective tissue disorder and no family history of aortic dissection or surgeries. She had not had any prior imaging of her aorta.

Five days postpartum, she presented to an outside emergency department with severe chest and upper back pain. Her blood pressure was elevated at 150/61 mm Hg, and a chest CTA demonstrated an acute type A aortic dissection, extending to the ostium of the left common iliac artery without extension into the head and neck, renal, or femoral vessels (**Figure 2**). She was transferred to our institution for further management and underwent an emergent David-V surgery during which her aortic valve was not replaced in setting of a morphologically normal trileaflet aortic valve. Her postoperative course was uneventful, and she was started on labetalol to achieve a goal systolic blood pressure of <120 mm Hg. A full aortopathy genetic panel was obtained, which revealed no evidence of pathogenic mutation to explain her aortic dissection. Follow-up imaging studies demonstrated stable aortic root dimensions, with the largest aortic diameter seen at the sinotubular junction measuring 4.2 × 3.7 cm. The residual TBAD was unchanged.

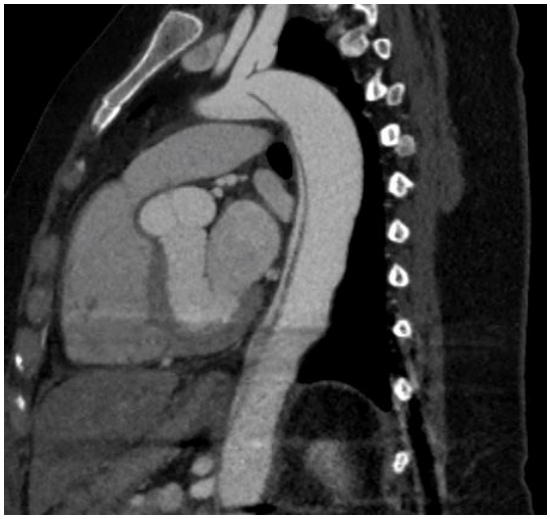

**Figure 2**

**Residual Unrepaired Aortic Dissection Following Type A Dissection Repair**

Baseline computed tomography demonstrating residual unrepaired type B dissection following ascending aorta replacement in a woman desiring to become pregnant.

Three years after this event, she presented to the cardio-obstetrics clinic for preconception counseling. Her blood pressure at that time was 110/69 mm Hg on metoprolol treatment. Her updated aortic CTA demonstrated a maximal descending aorta diameter of 3.4 × 3.4 cm in the mid-descending thoracic aorta without further extension of the residual descending aortic dissection. She was counseled that whereas data on the management of pregnancy with residual descending aortic dissection are scarce, a theoretical risk for adverse aortic events such as rupture and proximal progression of the dissection is of concern. The need to be followed closely throughout pregnancy and postpartum as well as meticulous blood pressure control were also emphasized. She expressed her full understanding of these risks and opted to proceed with another pregnancy. Once pregnancy was confirmed, aspirin therapy for preeclampsia prevention was initiated, and she was evaluated during each trimester at the cardio-obstetrics clinic. Whereas her echocardiograms showed stable ascending aortic diameter of ~3 cm, her blood

pressure values gradually increased from values of 105/60 mm Hg in the first trimester to 121/67 mm Hg in the early second trimester. She was transitioned from metoprolol to carvedilol at increasing doses, and hydralazine was later added for values >120/80 mm Hg. Urinalysis did not show proteinuria and preeclampsia labs were normal. During her third-trimester clinic visit, delivery options were reviewed, including vaginal delivery with an assisted second stage or a primary cesarean delivery. After careful deliberation, she elected to proceed with a primary cesarean delivery. An uncomplicated cesarean delivery was performed under spinal anesthesia at 37 + 0 weeks. Postpartum blood pressure values remained elevated at 134/62 mm Hg, and enalapril and amlodipine were added to the carvedilol and hydralazine therapy. She was monitored for 7 days and discharged to her home on as-needed hydralazine in addition to carvedilol, enalapril, and amlodipine. Repeat echocardiograms were again unchanged, showing ascending aorta measurement of 2.9 cm. At the time of the writing of this report, a repeat aortic CTA was not yet obtained. She was seen for a postpartum visit and was strongly encouraged to consider a highly effective method of birth control though ultimately, she opted to continue barrier methods. Her antihypertensives were adjusted to 50 mg of carvedilol twice daily, enalapril 20 mg daily, and spironolactone 25 mg daily.

## 14. **Severe Aortic Stenosis in a Pregnant Patient Displaced by the Ukrainian-Russian War**

Cardiac disease remains the most common cause of indirect maternal mortality in the United Kingdom[1] and in many other countries.[2] We present a case of a 32-year-old woman with severe aortic stenosis (AS) whose complex antenatal care was disrupted by her displacement because of war.

### Learning Objectives

- To recognize that access to multidisciplinary care is vital to optimize maternal outcomes in pregnant women with complex cardiovascular conditions.

- To understand the importance of postpartum contraception in women with pre-existing cardiac disease.

**History of Presentation**

A 32-year-old Ukrainian woman arrived in the United Kingdom in February 2023, having been displaced by the Ukrainian-Russian War. The rest of her family remains in Ukraine. At the time of arrival, she was 32 weeks' pregnant, in her second pregnancy. Her first pregnancy was 13 years prior, and she had an elective cesarean section at 39 weeks' gestation at the recommendation of her cardiologist in Ukraine.

She lived with a friend and spoke minimal English, requiring interpreting services to communicate. She was first seen in a routine antenatal outpatient setting at 34 weeks' gestation. She presented a single piece of paper stating she was pregnant, had severe bicuspid AS with a peak gradient of 70 mm Hg, and would require cardiac surgery postpartum. The only symptom she described was shortness of breath on moderate exertion (NYHA functional class II), giving her a modified World Health Organization classification of III. She was asked to attend the obstetric cardiac clinic on the same day but did not attend due to language and accessibility difficulties. Urgent echocardiography and cardiology review were arranged. On examination, her body mass index was normal. She was normotensive with a blood pressure of 117/69 mm Hg, pulse 82 beats/min, and no signs of heart failure; she had an ejection systolic murmur, with a quiet second heart sound.

**Past Medical History**

The patient had received regular cardiology follow-up for her bicuspid AS in Ukraine. She had been advised to have an aortic valve replacement following her previous pregnancy, but she had declined this intervention. She had an echocardiogram in Ukraine in 2022, but no detailed report was available. She had been asymptomatic aside from experiencing shortness of breath on moderate exertion.

**Differential Diagnosis**

The diagnosis of severe bicuspid AS was known from the limited medical information the patient provided. Any comorbidities, such as concomitant aortic regurgitation, aortic root dilatation, or coarctation, remained unknown at this point.

**Investigations**

Transthoracic echocardiography (**Figure 1**, **Video 1**) showed very severe/critical AS (peak velocity, Vmax: 5.7 m/s, mean pressure gradient: 75 mm Hg, aortic valve area: 0.7 $cm^2$), with moderate aortic regurgitation, but a normal left ventricular size and mild concentric hypertrophy. Left ventricular systolic function was preserved with an ejection fraction of 65%. The proximal ascending aorta was found to be mildly dilated at 3.3 $cm/m^2$ or 2.05 $cm/m^2$ indexed to body surface area. No other valve disease was present, and there was no pulmonary hypertension.

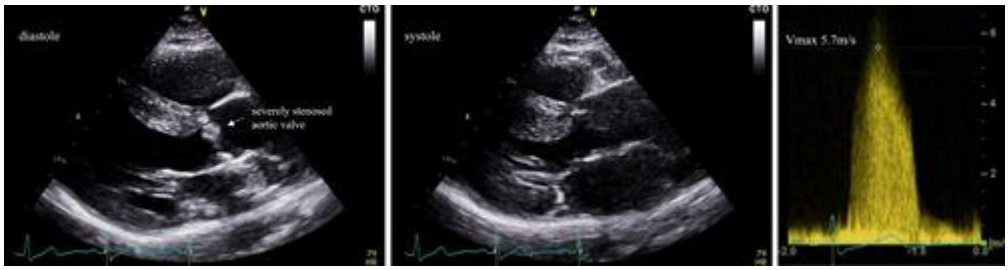


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**Figure 1**

**Antepartum Echo**

Parasternal long axis in diastole (left) and systole (middle) showing a normal size left ventricle with mild left ventricular hypertrophy and a severely stenotic aortic valve. The Doppler trace (right) indicates peak velocity (Vmax) of 5.7 m/s.

A fetal echocardiogram was normal. Fetal growth was normal on ultrasound.

**Management**

Her presentation was to a secondary care unit, where initial investigations and cardiology review were carried out, each time with interpreting services to ensure the patient had a good understanding of her clinical status. She was started on bisoprolol 1.25 mg once daily with a view to adding furosemide as necessary. Given the complexity of her cardiovascular disease, it was necessary to transfer her care to a tertiary center. The patient was alerted to the symptoms of deterioration, such as increasing breathlessness, chest pain, dizziness, presyncope, or syncope, and she was advised to attend emergency services at the tertiary center.

A peripartum care plan was devised by the pregnancy heart team comprising maternal cardiology, specialist obstetrics, and cardiac and obstetric anesthetics. This included timing of delivery at 36 to 37 weeks gestation by elective cesarean section (**Table 1**), unless in rapidly progressing labor. In this case, a trial of vaginal delivery would be attempted (**Table 2**). Emergency telephone numbers for obstetrics, anesthetics, and cardiology were documented in the handheld notes.

**Table 1Intrapartum Care Plan for Elective Cesarean Section**

| | |
|---|---|
| Analgesia | Regional or general |
| Epidural | Slow top-up, adrenaline containing solutions can be used |
| Spinal | Low-dose sequential CSE |
| General | Senior input required |
| Uterotonics | |
| Oxytocin | Cardiac regime (2 U over 15 min, then 5 U/h for 4 h) |
| Carboprost | As necessary |
| Misoprostol | As necessary |

| | |
|---|---|
| Ergometrine | Do not use |
| Monitoring | ECG, $SpO_2$, NIBP, arterial line, central venous catheter |
| Post delivery | HDU for 24 h with strict fluid balance, blood pressure, and heart rate monitoring |
| Follow-up | Review in cardiology clinic 1 week after discharge |

CSE = combined spinal-epidural anesthesia; ECG = electrocardiography; HDU = high dependency unit; NIBP = noninvasive blood pressure; $SpO_2$ = peripheral capillary oxygen saturation.

**Table 2Intrapartum Care Plan in the Event of Presentation in Rapidly Progressing Labor**

| | |
|---|---|
| First stage | |
| Analgesia | Epidural |
| Fluid balance | Hourly, aiming for even balance |
| Monitoring | ECG, $SpO_2$, NIBP, arterial line when possible |
| Second stage | |
| Delivery | Assisted |
| Transfer to | Obstetric HDU |
| Diagnostics | CXR or lung US if suspected pulmonary edema |
| Third stage | |
| Uterotonics | Cardiac oxytocin regime, no ergometrine |

CXR = chest x-ray; US = ultrasound; other abbreviations as in **Table 1**.

Postpartum contraception was discussed, and the patient opted for tubal ligation during cesarean section.

Before discharge, the woman was reviewed by a member of the cardiology team. A face-to-face follow-up appointment was arranged for 1 week postpartum.

The patient had an uneventful elective cesarean birth with bilateral tubal ligation at 36 + 5 weeks gestation. She was admitted to intensive care postpartum for observation. After 24 hours, she was well enough to step down to the postnatal ward and discharged home on day 2.

**Discussion**

Pregnancy is associated with physiological adaptations to the cardiovascular system, which occur from the first trimester. By 8 weeks' gestation, systemic vascular resistance falls by 30% to 70%, with a 30% to 50% increase in cardiac output and a 30%-50% increase in circulating volume.[3] These hemodynamic changes in pregnancy can lead to worsening symptoms in women with AS.[3]

ROPAC (Registry of Pregnancy and Cardiac Disease) data found that no woman with moderate or severe AS died during pregnancy or within 1 week postpartum.[4] Furthermore, a recent systematic review and meta-analysis found that pregnant women with severe AS had a maternal mortality rate of 2%,[5] in contrast to earlier studies reporting maternal mortality of 17.4%.[6] The leading cardiac complication during pregnancy in women with AS is heart failure,[4] mainly in patients with severe AS who were already symptomatic prepregnancy.[4] It has also been reported that pregnancy can have an impact the clinical course of AS, with 1 study previously reporting a 31% rate of cardiac surgery reported within a 3-year follow-up period.[7]

In this case a complicating factor is that this patient was displaced as a result of the Ukranian-Russian War and, as far as we are aware, did not receive prepregnancy counseling or comprehensive antenatal care prior to arriving in

the United Kingdom. Additional language and accessibility barriers contributing, this woman received only 4 weeks of antenatal care before she gave birth. Current European Society of Cardiology recommendations are that asymptomatic women with severe AS are risk stratified prepregnancy by using exercise testing and that all women with known cardiac or aortic disease who wish to embark on pregnancy require timely prepregnancy counseling,[8] which did not occur for this patient. Ukraine's health care system is facing multiple challenges with access to health care being severely affected.[9] We know that maternal mortality rates are increased for pregnant women in war zones, which is due to both the direct and indirect effects of war on health services.[10] Given these circumstances, it was fortunate that this patient did not develop worsening symptoms of her cardiac disease or new onset heart failure in pregnancy.

**Follow-up**

The patient was reviewed in the maternal cardiac clinic 2 weeks postnatally, where she remained well. A plan was made for further follow-up in the adult congenital heart disease clinic after 3 months with a cardiac magnetic resonance scan (**Figure 2**, **Video 2**) and a view to planning an aortic valve replacement.

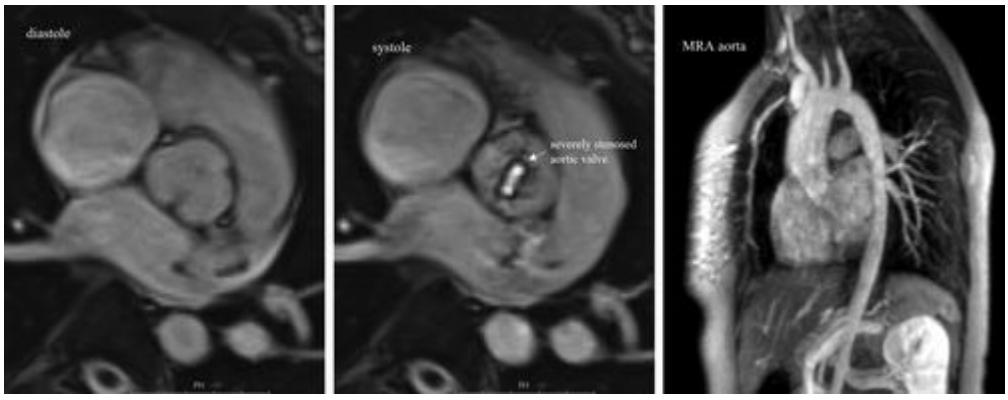


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**Figure 2**

**Postpartum CMR**

Cardiac magnetic resonance (CMR) showing a cross section of the severely stenosed aortic valve in diastole (left) and systole (middle). The angiogram shows the thoracic aorta. MRA = magnetic resonance angiography.

**Conclusions**

This case highlights the importance of appropriate prepregnancy counseling and multidisciplinary antenatal care for women with severe AS. It also shows how factors outside the patient's or the medical team's control can interfere with optimal care, necessitating timely and flexible arrangements with senior input. Over recent years, the maternal mortality risk in pregnant women with severe AS has fallen significantly, but these pregnancies can be associated with high maternal morbidity, especially in the context of complicating unforeseen circumstances as described here.

## 15. TAVR Beyond Fetal Viability: An Alternative to Preterm Delivery in Symptomatic Severe Aortic Stenosis

A 33-year-old G2P1001 with a history of bicuspid aortic valve, aortic dilation, and aortic insufficiency (AI) presented at 10 weeks gestation. She had undergone placement of a 27-mm Edwards Perimount Magna bovine pericardial valve and a 26-mm Dacron graft from the sinotubular ridge to just proximal to the innominate artery 10 years prior. An echocardiogram performed at this visit showed a mean aortic valve gradient of 44 mm Hg with a peak gradient of 74 mm Hg, consistent with severe aortic stenosis (AS). She was started on metoprolol XL 25 mg daily. She underwent serial echocardiograms and had progression of her aortic valve gradients, peaking at 25 weeks gestation with a mean gradient of 92 mm Hg (**Figure 1**). A plan was made for exercise restriction and ongoing expectant management unless she became symptomatic or there was worsening cardiac function.

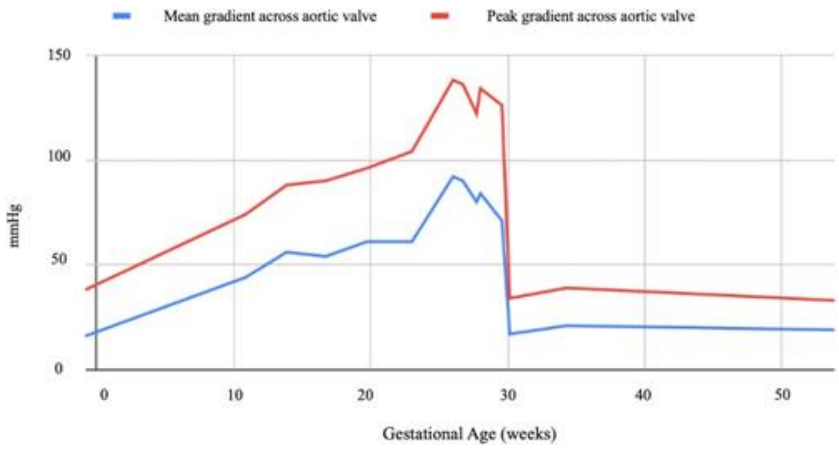


**Figure 1**

**Mean Gradient and Peak Gradient Across Aortic Valve**

The blue and red lines represent the mean and peak gradients in mm Hg across the aortic valve, respectively, progressing through pregnancy with gestational age in weeks on the y-axis.

**Learning Objectives**

- To demonstrate the management options for a pregnant patient with symptomatic severe AS before and after fetal viability.
- To understand that for pregnant patients with symptomatic severe AS, TAVR is a feasible treatment option with a multidisciplinary team to avoid preterm delivery.

16. **Advances in Diagnosis and Therapeutics in Recurrent Autoimmune Pericarditis in Pregnancy**

A 28-year-old woman at 24 weeks gestation presented with sudden-onset vision loss,; bilateral leg swelling; and new, nonspecific joint pain. She was found to have bilateral optic neuritis of unclear etiology and was subsequently treated with a course of high-dose steroids and plasmapheresis. While hospitalized, she complained of sharp pleuritic and positional chest pain that was not relieved by conservative measures, including acetaminophen and

topical analgesia. The chest pain worsened with inspiration and lying flat and improved when leaning forward. Two days later, she continued to endorse persistent, pleuritic, nonradiating chest pain. Her vital signs were significant for hypotension (blood pressure: 97/62 mm Hg) with distant heart sounds, which was a marked change from her normal vital signs on presentation.

## Learning Objectives

- To understand the management of autoimmune pericarditis.
- To understand key differences in management in patients who are also pregnant.
- To emphasize the importance of a multidisciplinary team with a cardiologist, rheumatologist, and obstetrician when delivering care.

## Past Medical History

Past medical history includes obesity (body mass index: 31.2 kg/m$^2$) and iron-deficiency anemia. There was no known history of autoimmune disease or malignancy. She also denied recent travel, new medications and supplements, or new exposures to radiation or toxins.

## Differential Diagnosis

Differential diagnoses include myocardial infarction, spontaneous coronary artery dissection, viral pericarditis, cardiac tamponade, costochondritis, malignancy, and autoimmune disease.

## Investigations

She was initially followed with serial high-sensitivity troponin tests, the results of which were persistently negative and suggested lack of myocardial involvement. Electrocardiograms (ECGs) showed diffuse ST-segment elevation with PR depression (**Figure 1**). Inflammatory markers were significantly elevated; erythrocyte sedimentation rate was 36 mm/h, and C-reactive

protein trended up from 1.1 to 3.3 mg/dL. Urine analysis was significant for few red blood cells, but no casts. Transthoracic echocardiogram (TTE) showed a large circumferential pericardial effusion with evidence of early diastolic right ventricle collapse, suggestive of increased intrapericardial pressures (**Figure 2A**, **Video 1**). There was respiratory variation across the mitral and tricuspid valves: 26% across the mitral valve and 58% across the tricuspid valve (**Figures 2B** and **2C**). The IVC was mildly dilated (2.2 cm) and collapsed <50% with inspiration (**Video 2**). The patient was in cardiac tamponade, and she underwent an emergent pericardiocentesis, which removed approximately 300 mL of serosanguinous fluid. Pericardial fluid cytology was sent, and the result was negative for any evidence of malignancy or bacterial infections such as tuberculosis.

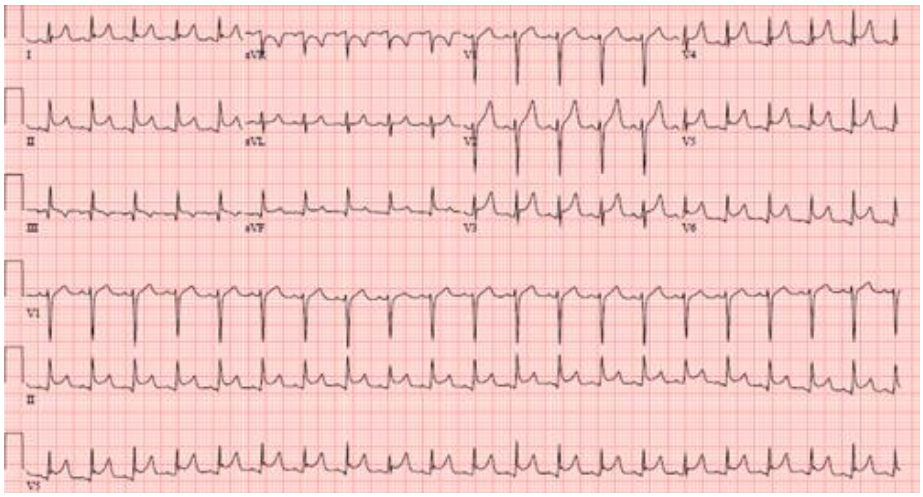

**Figure 1**

**Diffuse ST-Segment Elevation and PR-Segment Depression Highlighted in Multiple Leads**

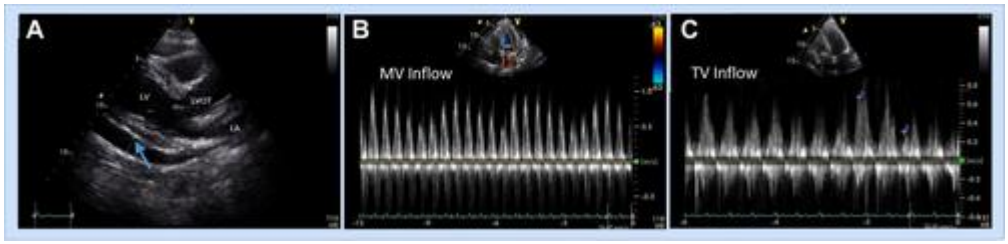


**Figure 2**

**Echocardiographic Findings**

(A) Echocardiogram on initial presentation. A large pericardial effusion is seen on the long-axis view (blue arrow), left ventricle (LV), left atrium (LA) and left ventricle outflow tract (LVOT) shown. (B, C) Respiratory variation across the mitral valve (MV) and tricuspid valve (TV). The mitral valve peak percentage difference is 48.4%, and the tricuspid valve peak percentage difference is –119.4%.

In addition, an autoimmune panel was ordered to assess for an underlying autoimmune disease. Anti-nuclear antibody (ANA), anti–double-stranded DNA antibody (via enzyme-linked immunosorbent assay), anti-neutrophilic cytoplasmic antibodies, anti-Ro (Anti-Sjogren's Syndrome A), anti-La (Anti-Sjogren's Syndrome B), anti-smith, and extractable nuclear antigen test results were negative. Complement levels, C3 and C4, were also assessed, and they were within normal limits.

The patient had elevated cardiolipin immunoglobulin M antibodies (14 MPL) during her initial presentation. Additionally, she underwent a kidney biopsy, which showed mild immune-complex–mediated focal mesangial proliferative glomerulonephritis. Per the pathologist review, the findings were likely related to an underlying autoimmune process. The constellation of symptoms, including polyarthritis, optic neuritis, pericarditis, and glomerulonephritis in the setting of elevated cardiolipin immunoglobulin M antibody levels led to a diagnosis of systemic lupus erythematosus (SLE) based on the Systemic Lupus Collaborating Clinics classification. The Systemic Lupus Collaborating Clinics classification requires satisfying 4 total criteria, including at least 1 clinical and 1 immunologic criterion.[1] Although SLE typically has a high positive rate of ANA, ANA-negative SLE has been reported in the literature with rates as high as 6.2% in newly diagnosed SLE.[2] We strongly suspect that the patient described in this case had an ANA-negative SLE.

**Management**

Because of the patient’s hemodynamic instability from tamponade physiology seen on TTE, she was admitted to the cardiac intensive care unit and

underwent pericardiocentesis and pericardial window. To treat her pericarditis and concomitant SLE, she was initially started on prednisone 60 mg. Because of the lack of clarity in treatment guidelines, her pregnancy posed a therapeutic challenge.

A multidisciplinary team consisting of her cardiologist, rheumatologist, and obstetrician held an extensive risk-benefit discussion with the patient regarding potential treatment options. The patient was advised that azathioprine carried a risk of low fetal birth weight and prematurity and low risk of teratogenicity and fetal malformations. Although colchicine was not strictly contraindicated in pregnancy, the data were scarce in terms of the risk of teratogenicity and fetal malformations. Because of her diagnosis of Focal segmental glomerulosclerosis and the potential for renal damage, the patient opted for low-dose colchicine 0.3 mg twice a day and azathioprine 50 mg twice a day.

Following pregnancy, prednisone was gradually tapered off, hydroxychloroquine 200 mg daily was added, and her dose of colchicine was increased to 0.6 mg twice a day. This treatment regimen was carefully selected after weighing the risks and benefits and determining the best course of therapy to treat and prevent autoimmune pericarditis recurrence in the setting of pregnancy.

Serial follow-up included inflammatory markers, ECGs, echocardiogram, and cardiac magnetic resonance (**Figure 3**). Although pericarditis in the setting of pregnancy challenged current therapeutic guidelines, her treatment plan resulted in a healthy delivery and controlled, chronic pericarditis on yearly follow-up.

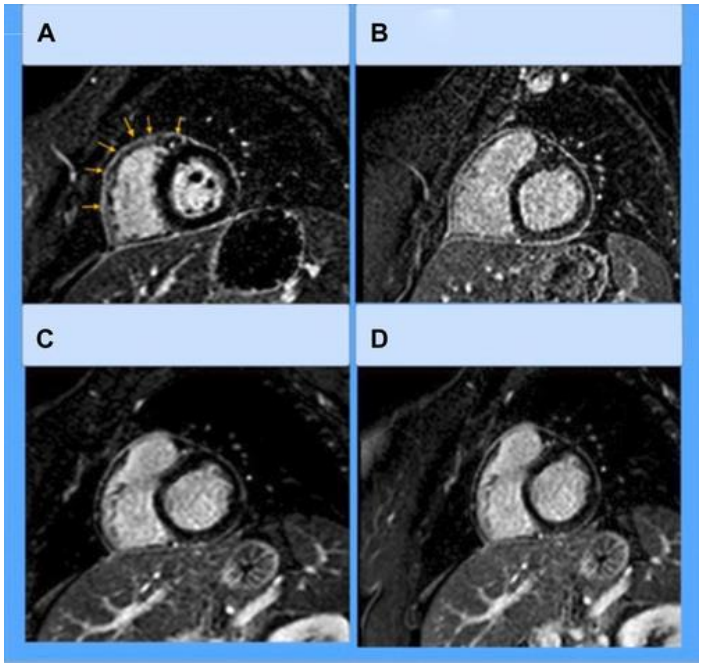


**Figure 3**

**Timeline of Cardiac Magnetic Resonance Showing Progression of Pericardial Disease**

(A) Moderate circumferential pericardial enhancement 1 month after presentation (yellow). (B) The interval decrease 5 months after presentation. (C) Only subtle delayed enhancement 19 months after presentation. (D) Stable mild delayed enhancement 43 months after presentation.

## Discussion

The etiology of pericarditis is variable and may often be attributable to idiopathic causes have bacterial, viral, or autoimmune origin. Echocardiogram is the mainstay of diagnosis and is supported by clinical presentation, ECG changes, and physical examination findings (pericardial friction rub). Most cases of pericarditis are managed medically without the use of invasive therapies, such as a pericardial window.[3] Chronic, recurrent pericarditis creates an additional therapeutic challenge that can be further complicated by pregnancy and undiagnosed autoimmune disease. Pregnancy complicates the treatment of pericarditis because many of the conventional therapies such as (nonsteroidal anti-inflammatory drugs [NSAIDs] and

colchicine) are either contraindicated or do not have evidence supporting their use.[3]

There are no formal guidelines that specify pericarditis management in pregnancy. However, there have been several case series and case studies that have discussed management and have contributed to the creation of guiding principles when approaching these patients. The European Society of Cardiology has commented on the use of certain therapies during pregnancy to manage recurrent pericarditis. Per their findings, NSAIDs, colchicine, corticosteroids, Imuran (Azathioprine), and intravenous immunoglobulin are relatively safe in the first, second, and third trimesters (**Table 1**). We used a multimodality image-based approach with repeat cardiac magnetic resonance to follow the course of her recurrent pericarditis in conjunction with following the existing guiding principles.

**Table 1Summary of Current Therapies for Pericarditis in Pregnancy**

| | **First Trimester** | **Second Trimester** | **Third Trimester** |
|---|---|---|---|
| NSAIDs | Allowed | Allowed | Avoid |
| Colchicine | Allowed | Allowed | Allowed |
| Corticosteroids (preferred dose: prednisone <20 mg/d) | Allowed | Allowed | Allowed |
| Biologics | Avoid | Avoid | Avoid |
| DMARDs (methotrexate, mycophenolate mofetil) | Avoid | Avoid | Avoid |

The table is organized by therapies approved by trimester and throughout lactation.

DMARD = disease-modifying antirheumatic drug; NSAID = nonsteroidal anti-inflammatory drug.

The treatment of autoimmune pericarditis incorporates many components of standard pericarditis treatment (NSAIDs, colchicine, steroids); however, it also favors the use of biologics such as interleukin 1 receptor antagonists and disease-modifying antirheumatic drugs, such as methotrexate and mycophenolate mofetil. Biologics such as interleukin-1 receptor antagonists, methotrexate, and mycophenolate mofetil are not recommended for use during any point while a patient is pregnant because they may cause adverse reactions to the fetus.[4]

Although NSAIDs continue to remain the standard of treatment during the first and second trimesters, they should be avoided in third trimester to prevent premature closure of patent ductus arteriosus.[5] There is no definitive consensus on whether NSAIDs are safe before conception. Some data point toward them affecting ovulation and increasing chances of miscarriage. Ibuprofen is preferred because of its better safety profile in terms of cross-placental transfer.[5,6]

Colchicine and low-dose steroids (0.2-0.5 mg/kg prednisone) play a central role in the treatment of recurrent pericarditis. Colchicine blocks mitotic division in metaphase but has been deemed safe during conception and pregnancy. Most of the safety data for colchicine are derived from a meta-analysis of 554 pregnancies where colchicine was continued for familial Mediterranean fever with no congenital fetal defects noted.[7] Steroids are also safe during conception and pregnancy, with ideal agents being prednisone or prednisolone. Rapid metabolism of steroids and dosing of <20 mg/day limits fetal exposure.[8]

There is a lack of data on the safety of azathioprine on pericarditis. It is a purine metabolism antagonist. The placenta metabolizes azathioprine to an inactive metabolite, which has been detected in fetal blood. It has not shown to have any teratogenic effects, but higher rates of other pregnancy complications, including low birth weight, prematurity, and jaundice, have been reported.[8] The mentioned pharmacologic agents are all safe during breastfeeding. Methotrexate and mycophenolate mofetil are the 2 agents

contraindicated during conception, pregnancy, and breastfeeding.[8] Outcomes of pregnancy in patients with pericarditis are good when the patients are managed by a multidisciplinary team, which has been evident in cohort analysis with pregnant patients who have a history of recurrent pericarditis.[9]

A multidisciplinary approach to treating pericarditis was paramount in ensuring a positive maternal-fetal outcome. By having an open lines of communication among multiple specialties, all aspects of this case were weighed carefully before making a decision on the best course of therapy. This case highlights the importance of multidisciplinary care when treating a patient with a complex condition with several variables that may affect her treatment outcomes.

**Follow-Up**

The patient described in our case had a total of 4 children with 3 pregnancies while being on colchicine 0.6 mg twice daily and azathioprine 150 mg at different timepoints. Following initial therapy, she did not have any documented evidence of hospital admissions or emergency department visits for symptomatic pericarditis with subsequent pregnancies, and her condition remained well controlled on the current regimen. All pregnancies were full-term vaginal deliveries. Newborn health assessment via Apgar scoring ranged between 7 and 9 for all 4 children, indicating normal births. There was no documented history of prematurity or evidence of fetal malformations or organ injury in any of her children. Her most recent regimen includes colchicine 0.6 mg twice daily and hydroxychloroquine 200 mg. Her subsequent imaging results have all been negative for pericardial effusion.

**Conclusions**

This case supports the use of colchicine for presumed autoimmune pericarditis and highlights the importance of shared decision making between physicians and patients when choosing azathioprine for treatment. These patients pose the most challenges with the initial diagnosis and management

choice. However, a multidisciplinary team approach with the trial of the discussed agents gave this patient successful pregnancy outcomes.

## 17. Preconception Counseling in a Patient With a Mechanical Aortic Valve and Recent Ischemic Stroke

This is a 31-year-old woman with anomalous left coronary artery from the pulmonary artery and congenital aortic stenosis. At age 1 year, she underwent surgical transposition of her left main coronary to the aorta and aortic valve commissurotomy. She enjoyed good health until age 26 years, when she developed severe aortic regurgitation from endocarditis with Cardiobacterium, presumed to be related to a foodborne illness. She underwent aortic valve replacement (AVR) with a 23-mm On-X valve (Artivion). She subsequently presented to the cardiology-obstetrics clinic for preconception counseling. A transthoracic echocardiogram (TTE) demonstrated normal AVR function.

### Learning Objectives

- To explain pregnancy risks associated with mechanical heart valves.
- To discuss maternal and fetal risks in pregnancies achieved after maternal stroke.
- To explain diagnosis and management of mechanical prosthetic valve thrombosis.
- To consider management of high-risk pregnancies after the Dobbs decision.

## 18. Acute Heart Failure During the Peripartum Period Due to Aggravated Tricuspid Regurgitation

A 38-year-old female patient (gravida II, para I) at 38 weeks of gestation (in vitro fertilization) was admitted to the gynecology department because of contractions. The pregnancy was uneventful. Spontaneous vaginal delivery occurred without complications. The day after discharge (fifth day postpartum), she presented to the emergency department because of shortness of breath and leg edema. Her blood pressure was 135/85 mm Hg, heart rate was 60 beats/min, oxygen saturation was normal (96%) with a respiratory rate of 20/min, and body temperature was 37 °C.

### Learning Objectives

- To highlight that acute HF can be consecutive to pre-existent or aggravated VHD during the peripartum period.
- To apply a multimodality imaging approach for VHD assessment VHD in the peripartum period.
- To emphasize the need for regular follow-up of women with VHD detected during pregnancy to determine the most appropriate timing for an intervention if indicated.

### Past Medical History

The patient was born and raised in Haiti. She moved to central Europe at the age of 25 years. She had 1 previous uneventful pregnancy and delivery 11 years earlier, resulting in a spontaneous vaginal delivery. She has been experiencing hypothyroidism for 3 years and was euthyroid under substitution on admission. The family history revealed a history of arterial hypertension, and there were no congenital heart defects.

## Differential Diagnosis

Tricuspid regurgitation (TR) in women of childbearing age may be related to congenital heart disease (eg, Ebstein anomaly, atrioventricular canal defects, arrhythmogenic right ventricular cardiomyopathy, tricuspid valve dysplasia), secondary to annular dilatation in the setting of right ventricular (RV) volume overload, or from mechanical valve damage (eg, prior infectious endocarditis). Moreover, in the presence of arterial hypertension and edema, pre-eclampsia must be ruled out.

## Investigations

The clinical examination revealed lower limb edema. Pulmonary auscultation was clear, and cardiac auscultation found a holosystolic murmur with punctum maximum at the lower left sternal edge. Laboratory analyses revealed mildly elevated C-reactive protein, mild anemia (hemoglobin, 102 g/L) without signs of hemolysis, normal platelet count, and elevated N-terminal pro–B-type natriuretic peptide (501 pg/mL) and D-dimer (3,431 µg/L). Renal function was normal, but liver parameters were mildly elevated (aspartate amino transferase: 42 U/L; alanine amino transferase: 45 U/L; alkaline phosphatase: 139 U/L). Urinary examinations revealed no abnormalities, in particular no proteinuria. The electrocardiogram showed sinus rhythm with no other abnormalities. Deep vein thrombosis and pulmonary embolism were ruled out using leg ultrasound and thoracic multislice computed tomography, which also excluded pleural effusions, pulmonary infiltrate, and aortic dissection. A transthoracic echocardiography (TTE) revealed visually slightly impaired systolic function of the RV with possible ballooning of the right apex but normal tricuspid annular plane systolic excursion and normal function of the left ventricle (LV). This resulted in severe TR caused by leaflet tethering with tricuspid annular dilatation (45 mm), leading to a coaptation gap of 7.2 mm with signs of RV volume overload and diastolic D-shaping of the LV (**Figures 1A to 1C**, **2A**, and **2B**, **Videos 1**, **2** and **3**).

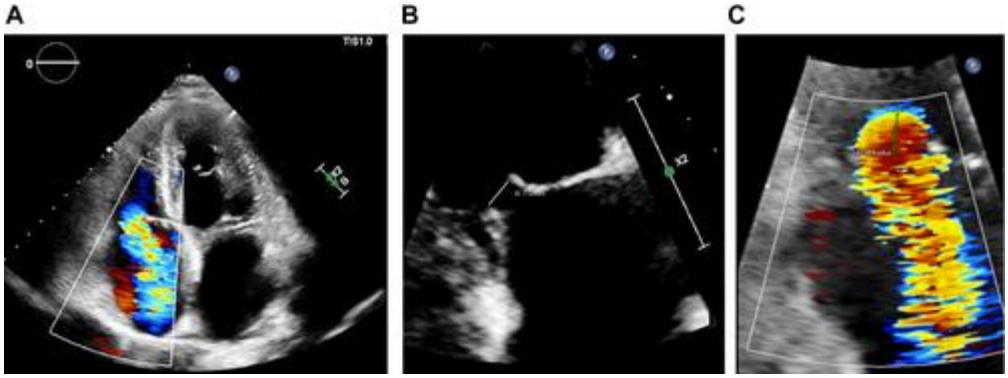


**Figure 1**

**TR Assessment at Presentation in Decompensated State**

(A) Four-chamber view with color Doppler showing severe TR. (B) RV inflow view showing a coaptation gap of 7.2 mm. (C) RV-focused 4-chamber view with color Doppler proximal isovelocity surface area radius: 0.9 cm. RV = right ventricle; TR = tricuspid regurgitation.

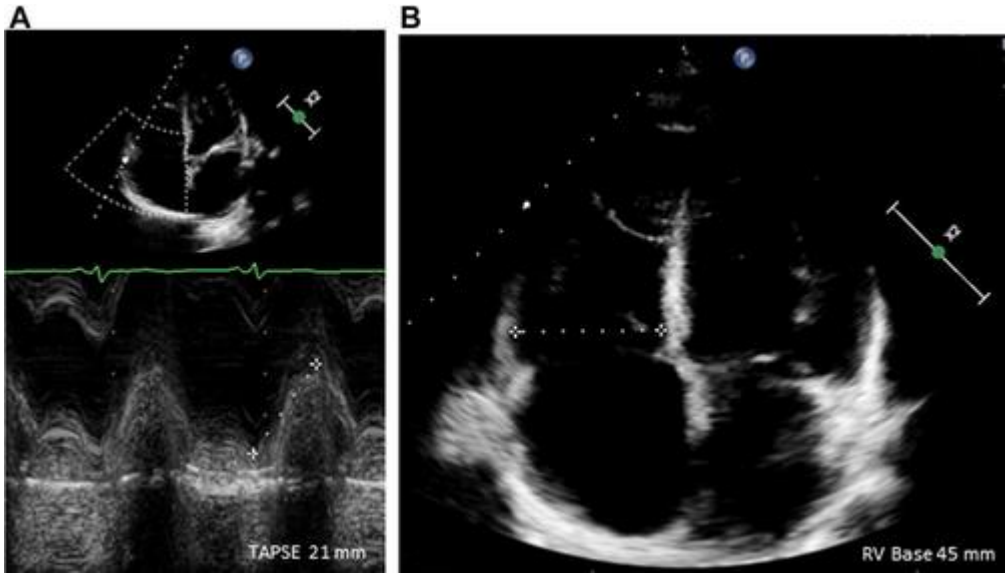


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**Figure 2**

**Right Ventricular Dimension and Longitudinal Function at Presentation in Decompensated State**

(A) Tricuspid annular plane systolic excursion within the normal range, 21 mm. (B) Four-chamber view with slightly enlarged right ventricle basal diameter of 45 mm.

**Management**

The patient was admitted to the mother–child unit with the preliminary diagnosis of acute cardiac decompensation caused by valvular heart disease (VHD), and forced diuresis was initiated with oral torsemide. However, because of hypertensive blood pressure readings (up to 190/130 mm Hg), she was transferred to the intensive care unit. Blood pressure was controlled with intravenous (IV) urapidil and oral amlodipine, and volume overload was treated with IV furosemide. Recompensation was rapidly achieved, and blood pressure was controlled, allowing the patient to be transferred to the cardiology ward. The antihypertensive medication was changed to oral hydrochlorothiazide and amlodipine, while IV furosemide was replaced by oral torsemide. No rhythm disturbances occurred. The laboratory and urine analysis results remained within the normal ranges. The patient did not develop symptoms suggestive of pre-eclampsia such as respiratory distress, abdominal pain, or new-onset and persistent headache or visual symptoms, and remained afebrile during the hospital stay, without signs of infection.

A second TTE showed normal RV dimensions with normal RV systolic function; however, there was persistence of severe secondary TR caused by leaflet tethering, resulting in a central jet. RV/right atrial gradient was at the upper limit (30 mm Hg) in the context of peripartum hemodynamic changes. There were no echocardiographic signs suggestive of congenital heart disease. In addition, secondary causes of systemic hypertension were excluded, including pre-eclampsia or hyperthyroidism. The patient was discharged home 2 days later with oral hydrochlorothiazide and amlodipine.

**Discussion**

TR is of secondary etiology in >90% of the patients,[1] and TR of any severity is often observed during pregnancy[2] as a result of increased circulating volume,

heart rate, and cardiac output, with or without consecutive dilatation of the tricuspid annulus. TR is usually well tolerated during the peripartum period,[3] and an acute presentation like the one described in this report is rare in an otherwise healthy woman and should prompt further investigations. In contrast, in women with pre-existing congenital heart disease, moderate/severe TR may be associated with maternal cardiac complications because of chronic volume overload of the RV that may not be able to accommodate the new hemodynamic conditions.[4]

The presence of VHD in general—in particular, moderate or severe chronic mitral regurgitation, TR, multivalve disease—LV systolic dysfunction, or pulmonary hypertension (PHT) diagnosed before pregnancy predicts cardiac adverse events during pregnancy.[5] Furthermore, adverse fetal and neonatal events are increased in the presence of all types of maternal regurgitant valve lesions.[5] The risk of maternal cardiac events during pregnancy can be further stratified using the CARPREG II (Cardiac Disease in Pregnancy Study II) risk score, a 10-predictor score comprising the aforementioned high-risk features.[6]

In our case, the exact etiology of TR remains unclear, and pregnancy accompanied by physiologic volume overload may have acted as the demasking factor of a so far subclinical condition. The presence of tethering with minimal annular dilatation confirms the acute presentation of VHD. Dyspnea linked to insufficient exercise cardiac output represents an early symptom of TR that should lead to further investigations and the use of multimodality imaging, in particular cardiac magnetic resonance, to exclude a congenital etiology, confirm TR grading, and assess RV function. The acute presentation of acute heart failure (HF) with visually impaired RV function and slight apical ballooning might also suggest isolated RV stress cardiomyopathy, possibly caused by stress during labor, which resolved in the follow-up visits with remaining moderate TR.

VHD is increasingly recognized as a cause of acute HF or a contributing factor that may precipitate the onset of HF symptoms. Recent recommendations

encourage the interdisciplinary management of patients with HF and VHD.[7] Symptoms or progression of chamber dilatation should trigger early referral to a center with competence in valve treatment, including emerging minimally invasive treatment options.

**Follow-Up**

The patient presented for a clinical visit at the adult congenital heart disease department 4 weeks, 3 months, and 5 months postpartum. Follow up TTE at 1 month (**Figure 3**, **Video 4**) and 5 months (**Figures 4A to 4C**, **5A**, and **5B**, **Videos 5** and **6**) showed reduction to moderate TR under medical treatment as prescribed at discharge. RV/right atrial gradient (23 mm Hg) was not suggestive of pulmonary hypertension. The right basal diameter decreased to 34 mm. In addition, cardiac magnetic resonance revealed normal LV and RV function with normal dimensions (indexed right ventricular end-diastolic volume: 90 mL/m$^2$; indexed right ventricular end-systolic volume 38 mL/m$^2$) (**Table 1**), and no structural abnormalities, in particular no sign of tricuspid valve and RV dysplasia, were observed (**Figures 6A to 6C**, **Videos 7** and **8**). Moderate TR was confirmed (regurgitant fraction: 16%). No intracardial shunts were seen ($Q_p/Q_s$: 1.0). Systemic blood pressures normalized under hydrochlorothiazide monotherapy. In the absence of clinical factors suggesting secondary causes (hyperthyroidism, hyperparathyroidism, coarctaction of the aorta, and sleep apnea were excluded), hypertension was considered of gestational etiology. Yearly echocardiographic controls are planned to detect symptoms, TR progression, and signs of RV dilatation early.

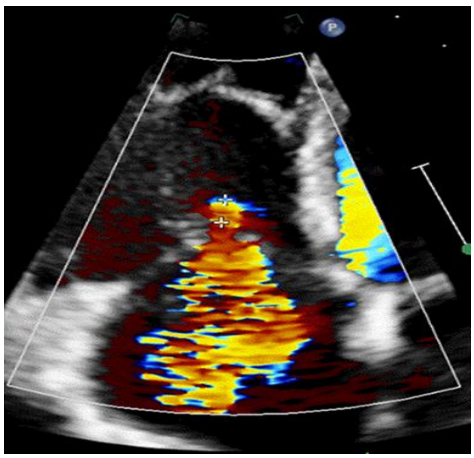

**Figure 3**

**Echocardiographic Follow-Up at 1 Month**

Right ventricle–focused 4-chamber with color Doppler; proximal isovelocity surface area radius: 0.5 cm.

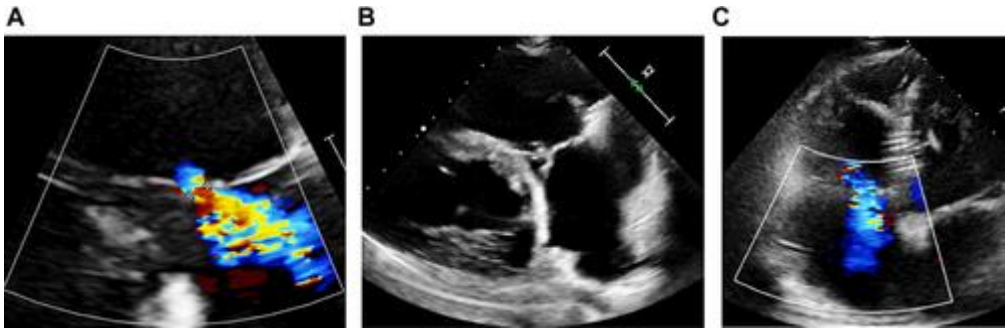


**Figure 4**

**Echocardiographic Follow-Up at 5 Months**

(A) Right ventricle inflow view with color Doppler; proximal isovelocity surface area radius: 0.4 cm. (B) Right ventricle inflow view: no relevant coaptation defect visible after diuretic therapy. (C) Four-chamber view with color Doppler showing moderate tricuspid regurgitation.

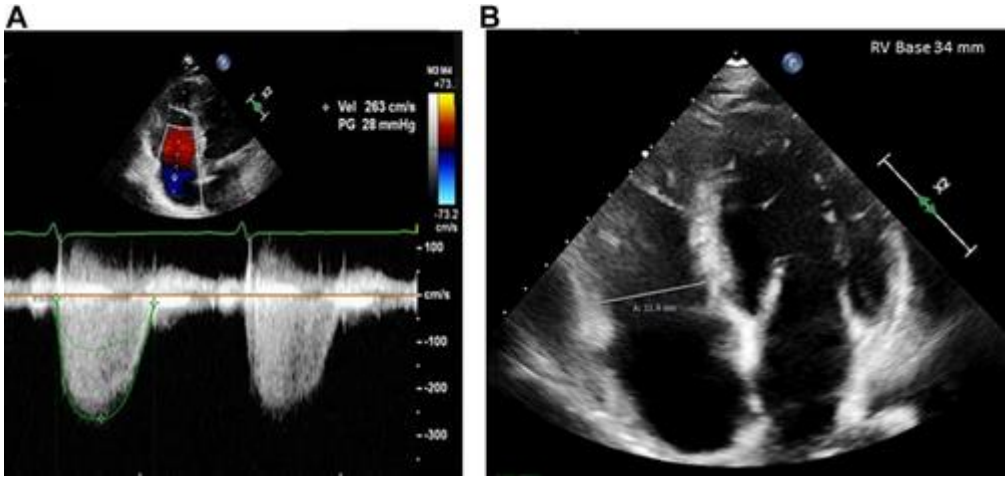


**Figure 5**

**Echocardiographic Follow-Up at 5 Months**

(A) Four-chamber view with continuous-wave Doppler. (B) Four-chamber view with right ventricle basal diameter within the normal range, 34 mm.

**Table 1Summary of RV and TR Measurements at Presentation, Baseline, and Follow-Up**

| | **TTE at Presentation** | **TTE at Discharge** | **TTE at the 5-Month Follow-Up** | **CMR at the 5-Month Follow-Up** |
|---|---|---|---|---|
| RVEDD base, mm | 45 | 39 | 38 | |
| RA ESV index, mL/m$^2$ | 40.8 | 29.13 | 22.10 | |
| TAPSE, mm | 21 | 21 | 28 | |
| RV S′, cm/s | 15.1 | N/A | 14.6 | |
| FAC, % | 33 | 35 | 43 | |
| RV/RA gradient, mm Hg | 25 | 25 | 24 | |
| IVC diameter during expirium, mm | 25 | 13 | 11 | |
| IVC diameter during inspirium, mm | 19 | 11 | 8 | |
| Estimated central venous pressure, mm Hg | 15 | 10 | 5 | |
| Estimated sPAP, mm Hg | 40 | 35 | 29-30 | |
| TR severity | Severe | Moderate | Moderate | |

**Table 1Summary of RV and TR Measurements at Presentation, Baseline, and Follow-Up**

| | TTE at Presentation | TTE at Discharge | TTE at the 5-Month Follow-Up | CMR at the 5-Month Follow-Up |
|---|---|---|---|---|
| TR Vmax, m/s | 2.5 | 2.5 | 2.45 | |
| Coaptation gap, mm | 7.2 | 0 | 0 | |
| TR EROA, $cm^2$ | 0.78 | 0.20 | n.a. | |
| Regurgitation volume, mL | 58.93 | 20.33 | n.a. | |
| Vena contracta, mm | 7.5 | 4.2 | 4 | |
| RV EDV index, $mL/m^2$ | | | | 90 (normal range: 48-104) |
| RV ESV, mL | | | | 38 (normal range: 13-48) |
| RV SV index, $mL/m^2$ | | | | 53 (normal range: 29-66) |
| RV CO, $L/min/m^2$ | | | | 3.7 |

**Table 1Summary of RV and TR Measurements at Presentation, Baseline, and Follow-Up**

| | TTE at Presentation | TTE at Discharge | TTE at the 5-Month Follow-Up | CMR at the 5-Month Follow-Up |
|---|---|---|---|---|
| RVEF, % | | | | 58 |
| TR regurgitant fraction, % | | | | 16 |
| $Q_p/Q_s$ PA/Ao | | | | 1 |

CMR = cardiac magnetic resonance; CO = cardiac output; EDV = end-diastolic volume; EROA = effective regurgitant orifice area; ESV = end-systolic volume; FAC = fractional area change; IVC = inferior vena cava; N/A = not applicable; PA/Ao = main pulmonary artery/aorta; RA = right atrium; RV = right ventricle; RVEDD = right ventricular end-diastolic diameter; RVEF = right ventricular ejection fraction; sPAP = systolic pulmonary artery pressure; SV = stroke volume; TAPSE = tricuspid annular plane systolic excursion; TR = tricuspid regurgitation; TTE = transthoracic echocardiography; Vmax = maximal velocity.

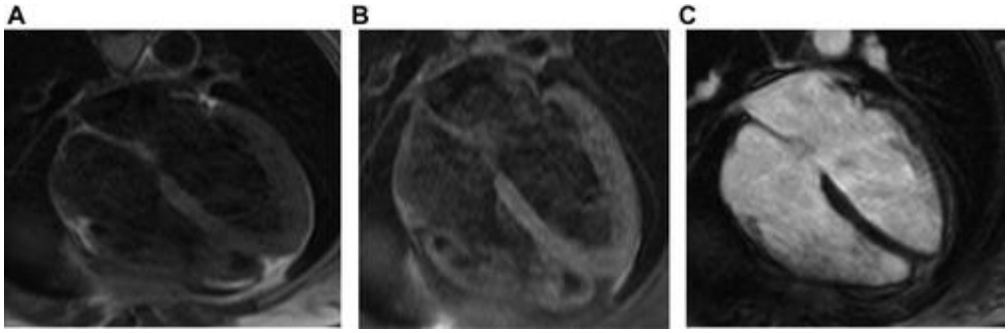


**Figure 6**

**Cardiac Magnetic Resonance at the 5-Month Follow-Up Showing Normal Dimensions and Function of the Heart Chambers**

(A) Cardiac magnetic resonance 4-chamber view without fat saturation. (B) Cardiac magnetic resonance 4-chamber view with fat saturation. No myocardial fatty deposits are visible, and there are no signs of arrhythmogenic right ventricular cardiomyopathy. (C) Cardiac magnetic resonance 4-chamber view with application of gadolinium magnetic resonance contrast. No late gadolinium enhancement could be observed, suggesting a structurally normal heart with no fibrosis or signs of inflammatory or systemic heart disease.

**Conclusions**

Our case report first raises awareness around the possible occurrence of TR during the peripartum period caused by increased circulating volume. Heart conditions like VHD that are unmasked during pregnancy may pose a cardiovascular risk factor later in life. An increasing body of evidence also suggests that pre-existing heart conditions in pregnant women increase the risk of adverse events not only in women but also in the fetus/newborn. According to current American College of Cardiology/American Heart Association[8] and European Society of Cardiology[9] guidelines, women with suspected VHD who are considering pregnancy should undergo a clinical evaluation and TTE before pregnancy (Class I indication). If VHD is first detected during pregnancy, early referral and interdisciplinary follow-up at a specialized Heart Valve Center are indicated.

## 19. Multidisciplinary Approach to Management of Hypertrophic Cardiomyopathy With Severe Left Ventricular Outflow Obstruction in Pregnancy

A 37-year-old woman (G7P6006) at 28.3 weeks gestational age was referred to Maternal-Fetal Medicine because she had worsening exertional dyspnea, occasional lightheadedness, and 2 syncopal episodes within the prior 2 weeks. Her past medical history was significant for recurrent syncope attributed to

hypertrophic obstructive cardiomyopathy (HOCM) diagnosed 3 months prior to pregnancy, at which time metoprolol tartrate 50 mg twice daily was initiated. Syncopal episodes had not recurred since then until the current presentation. There was no family history of HOCM or sudden cardiac death. She had not received preconception counseling as pregnancy was unplanned. She smoked 1 pack daily and used recreational drugs until 2 months prior to conception. Physical exam revealed heart rate of 93 beats/min, blood pressure 120/86 mm Hg, oxygen saturation 100% on room air, and a grade 3 systolic murmur at the left parasternal area.

**Learning Objectives**

- To discuss management options for a pregnant patient with HOCM in the antepartum, intrapartum, and postpartum periods.
- To identify complications that can occur in pregnancy in patients with HOCM.

20. **Sex-Specific Lifetime Risk of Cardiovascular Events**

AIMS

Better understanding of sex differences in cardiovascular disease (CVD) is essential in tailoring appropriate preventative strategies. Using a large population-based study with follow-up >25 years, we aimed to determine sex-specific lifetime risks of incident CVD and cardiovascular (CV) mortality amongst populations with and without prevalent CVD.

METHODS AND RESULTS

Participants were drawn from the European Prospective Investigation into Cancer-Norfolk and followed up for a median of 26.2 years. Sex-specific lifetime risks were ascertained accounting for the competing risk of death. Models were adjusted for ethnicity and time-updated covariates: material deprivation, CV risk factors, lifestyle factors, comorbidities, and medication.

A total of 23 859 participants [54.5% women; mean age (standard deviation) 59.2 (9.3) years at baseline] were included. Adjusted lifetime risks of incident CVD were higher in men than in women (69.1 vs. 57.7% at age 75): cause-specific hazard ratio (cHR) (99% confidence interval)-1.49 (1.41-1.57), while the risks of CV mortality at age 75 were 4.4% (men) and 3.1% (women): cHR-1.42 (1.31-1.54). Myocardial infarction was the predominant first presentation in men until the eighth decade. In women, the first CVD manifestations after their sixth decade were predominantly atrial fibrillation and stroke. The male-associated excess relative risks of incident CVD and CV mortality were halved in people with prevalent CVD.

CONCLUSION

We characterized the sex-specific lifetime CV risks in a large cohort. Men had substantially higher risk of incident CVD and CV mortality than women, which was attenuated amongst people with prevalent CVD. Our findings provide an evidence base for sex-specific CV prevention.

LAY SUMMARY

In this population-based study, we aimed to understand the sex-specific lifetime trajectories of different heart and circulatory disorders and their relationship with death from heart disease. We included ~24 000 participants in the analyses, who were followed up for >25 years. Men had a higher lifetime risk of heart and circulatory disorders compared with women. Heart attacks were the predominant first presentation in men until the eighth decade, while in women this was manifested as heart rhythm disorders and stroke after their sixth decade. The excess risk of death from heart disease observed in men with pre-existing heart disease was attenuated compared with those free of heart disease at baseline. In conclusion, men and women require tailored heart disease prevention efforts given the marked sex disparities in heart disease and death over the very long-term highlighted by our study.

## 21. Long-Term BP Control After a Hypertensive Pregnancy Following Physician-Optimized Self-Management

While we know that self-monitoring of blood pressure (BP) — typically using home BP measurement devices, either with or without concomitant telemonitoring — can help to improve BP control in the general population, few data exist on the use of self-monitoring in women with BP abnormalities during pregnancy.

With the recently reported CHAP trial,[1] proactive management of hypertension during pregnancy is timelier and more important than ever before. Accordingly, the results of the POP-HT trial, now presented in JAMA, are of importance. The authors show in a single-center, randomized, open-label trial that BP self-monitoring by new mothers with hypertension improved BP control at 9 months, with an improvement of 6.5/5.8 mm Hg in the 24-hour ambulatory BP control. Importantly, the self-monitored arm of this trial used not just home BP measurement but also a smartphone app that provided research clinical advice on medication titration; it is this latter aspect of the trial intervention that could be difficult to translate to routine care and challenging to scale to all women in need.

As such, further research including the cost-effectiveness and feasibility of the entire intervention (including the app) in real-world settings will be needed before more widespread implementation. I hope such studies are done and, assuming success in those studies, that this intervention then penetrates usual care. Too often we see the findings of this type of study being "put on the shelf" and not translated into routine care.

## 22. Cardiac Abnormalities in Hispanic/Latina Women With Prior De Novo Hypertensive Disorders of Pregnancy


BACKGROUND

Hypertensive disorders of pregnancy (HDP) are associated with long-term maternal risks for cardiovascular disease for reasons that remain incompletely understood.

METHODS

The HCHS/SOL (Hispanic Community Health Study/Study of Latinos), a multi-center community-based cohort of Hispanic/Latino adults recruited 2008 to 2011, was used to evaluate the associations of history of de novo HDP (gestational hypertension, preeclampsia, eclampsia) with echocardiographic measures of cardiac structure and function in Hispanic/Latina women with ≥1 prior pregnancy and the proportion of association mediated by current hypertension (>140/90 mm Hg or antihypertensive therapy).

RESULTS.

The study cohort included 5168 Hispanic/Latina women with an average age (SD) of 58.7 (9.7) years at time of echocardiogram. Prior de novo HDP was reported by 724 (14%) of the women studied and was associated with lower left ventricle (LV) ejection fraction -0.66 (95% confidence interval [CI], -1.21 to -0.11), higher LV relative wall thickness 0.09 (95% CI, 0-0.18), and 1.39 (95% CI, 1.02-1.89) higher risk of abnormal LV geometry after adjusting for blood pressure and other confounders. The proportion of the association mediated by current hypertension between HDP and LV ejection fraction was 0.09 (95% CI, 0.03-0.45), LV relative wall thickness was 0.28 (95% CI, 0.16-0.51), abnormal LV geometry was 0.14 (95% CI, 0.12-0.48), concentric left ventricular hypertrophy was 0.31 (95% CI, 0.19-0.86), and abnormal LV diastolic dysfunction was 0.58 (95% CI, 0.26-0.79).

CONCLUSIONS.

In a large cohort of Hispanic/Latina women those with history of de novo HDP had detectable and measurable subclinical alterations in cardiac structure and both systolic and diastolic dysfunction that were only partially mediated by current hypertension.

## 23. Severe Maternal Morbidity According to Mode of Delivery Among Pregnant Patients With Cardiomyopathies

BACKGROUND

Women with cardiomyopathies are at risk for pregnancy complications. The optimal mode of delivery in these patients is guided by expert opinion and limited small studies.

OBJECTIVES

The objective of this study is to examine the association of delivery mode with severe maternal morbidity events during delivery hospitalization and readmissions among patients with cardiomyopathies.

METHODS

The Premier inpatient administrative database was used to conduct a retrospective cohort study of pregnant patients with a diagnosis of a cardiomyopathy. Utilizing a target trial emulation strategy, the primary analysis compared outcomes among patients exposed to intended vaginal delivery vs intended cesarean delivery (intention to treat). A secondary analysis compared outcomes among patients who delivered vaginally vs by cesarean (as-treated). Outcomes examined were nontransfusion severe maternal morbidity during the delivery hospitalization, blood transfusion, and readmission.

RESULTS

The cohort consisted of 2,921 deliveries. In the primary analysis (intention to treat), there was no difference in nontransfusion morbidity (adjusted OR [aOR]: 1.17; 95% CI: 0.91-1.51), blood transfusion (aOR: 1.27; 95% CI: 0.81-1.98), or readmission (aOR: 1.03; 95% CI: 0.73-1.44) between intended

vaginal delivery and intended cesarean delivery. In the as-treated analysis, cesarean delivery was associated with a 2-fold higher risk of nontransfusion morbidity (aOR: 2.44; 95% CI: 1.85-3.22) and blood transfusion (aOR: 2.26; 95% CI: 1.34-3.81) when compared with vaginal delivery.

CONCLUSIONS

In patients with cardiomyopathies, a trial of labor does not confer a higher risk of maternal morbidity, blood transfusion, or readmission compared with planned cesarean delivery.