



CASE REPORT

Open Access

# Cardiovascular Urgencies and Emergencies in Pregnancy: A Case Based Review of Some Common Problems

Timothy E Paterick<sup>1\*</sup> and Venay Mehta<sup>2</sup>

<sup>1</sup>Aurora Health Care, USA

<sup>2</sup>Cardiac Electrophysiology, Aurora BayCare Medical Center, Green Bay, Wisconsin, USA

## ABSTRACT

This manuscript explores clinical presentation and physical examination findings in pregnant patients with potential heart disease. These patients were seen in cardiac consultation by the authors. This manuscript allows the cardiology readership to be introduced to the complexity of pregnancy and cardiovascular disease.

We review the clinical presentation, physical examination, and multimodality imaging that established a diagnosis in pregnant patients with coronary artery disease, cardiomyopathies, arrhythmias, valvular heart disease, and pulmonary hypertension. The cases represent a subset of cardiovascular conditions affecting pregnant patients. For a comprehensive review, the readership is directed to the 2018 European Society of Cardiology (ESC) Guidelines for the management of cardiovascular disease during pregnancy.

## ARTICLE HISTORY

Received January 25, 2022  
Accepted January 31, 2022  
Published February 28, 2022

**Keywords:** Pregnancy, Cardiovascular Disease and Common Cardiovascular Presentations.

## Introduction

The cardiovascular diseases possible in pregnancy are many, as detailed in the 2018 ESC Guidelines for the management of cardiovascular disease in pregnancy. The recognition and management of cardiovascular disease impacting the health of pregnant females and their baby is daunting, but it is essential that cardiologists understand these diseases and treatment approaches to prevent fetal and maternal morbidity and mortality. The cases presented just brush the surface of cardiovascular conditions that physicians may encounter in daily clinical practice

## Methods

Each author reviewed cases retrospectively after participating in the case at presentation. Individual patients were contacted by telephone to obtain consent for publication on condition of anonymity. All living patients and the deceased patient's mother agreed. There was an expedited IRB review.

The case reviews highlight the clinical presentations, physical examination, imaging features, and treatment approaches associated with pregnant patients with cardiovascular disease.

## Results/Case Presentations

A summary of the cases presented can be found in Table 1.

**Table 1: Diagnosis, Treatment, and Prognosis of cardiovascular Diseases in Pregnancy**

Case #	Disease	Incidence	Diagnosis	Treatment	Prognosis
1	SCAD	Rare	Cath	Medical vs. PCI	Good
2	TC	Rare	Echo/Cath	GDMT for HF	Good
3	PE/AFE	Rare	Echo/CT	A/C: SC	Good/Guarded
4	PPH	Low	Echo	Vasodilators	Poor
5	PPC	Low	Echo	GDMT	Good/Guarded

**Contact** Timothy E Paterick ✉ tpaterick@gmail.com 📍 Aurora Health Care, Milwaukee/Green Bay, USA.

6	VT	Rare	RM	A/Ab/D	Guarded
7	DCM	Rare	FH/G/Echo	GDMT/A	Poor
8	BAV/A	Common	Echo/FH	PE/Echo	Good

A/Ab/D, antiarrhythmic/ablation/device; A/C, anticoagulation; BAV/A, bicuspid aortic valve with aortopathy; DCM, dilated cardiomyopathy; FH, family history; G, genetics; GDMT, guideline directed medical therapy; HF, heart failure; PE/AFE, pulmonary embolism/amniotic fluid embolism; PPC, postpartum cardiomyopathy; PPH, primary pulmonary hypertension; RM, rhythm monitoring; SC, supportive care; SCAD, spontaneous coronary artery dissection; TC, takotsubo cardiomyopathy; VT, ventricular tachycardia;

**Case 1**

**32-Year-Old Woman with Shortness of Breath (Sob) and Chest Pressure**

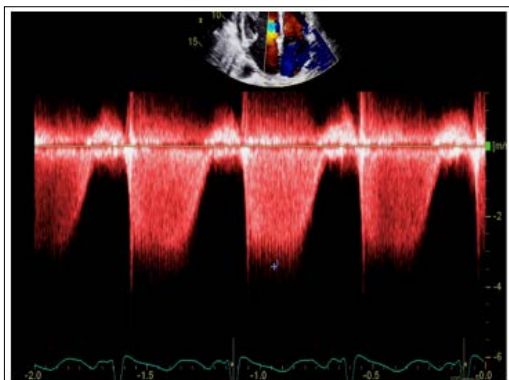
The patient had two prior normal deliveries. The patient was G3P3 and had experienced an uneventful pregnancy. Post-delivery, she became acutely SOB required intubation and had a blood pressure (BP) of 190/100 mmHg and heart rate (HR) of 130 bpm. The electrocardiogram (ECG) revealed sinus tachycardia, low voltage QRS, and poor R wave progression with diffuse, non-specific ST changes (Figure 1A).



**Figure 1A:** Sinus tachycardia with loss of R waves across the precordium and non-specific ST changes

Treatment with a diuretic, beta-blocker, and intravenous sodium nitroprusside resulted in a BP of 150/90 mmHg and HR of 95 bpm. Oxygenation was adequate with assisted ventilation.

Echocardiography demonstrated a dilated left ventricular ejection fraction (LVEF) of 20%, dilated right ventricle (RV) with severe reduction of RV systolic function, severe tricuspid regurgitation (TR), and elevated right heart pressure (Figure 1B). An atrial septal defect (ASD) was identified.



**Figure 1B:** The RV-RA gradient is approximately 50 mm Hg

Cardiac computed tomography (CT) was negative for pulmonary embolism (PE) and aortic dissection. Magnetic resonance imaging (MRI) revealed a dilated LV cavity with global systolic dysfunction and a calculated LVEF of 22% and an enlarged RV with depressed RV systolic function. The differential diagnosis included biventricular stress-induced cardiomyopathy, paradoxical embolism to the coronary tree, and spontaneous coronary artery dissection of the

proximal left anterior descending coronary artery. The patient became hemodynamically stable with medical therapy including a diuretic, beta-blocker and angiotensin converting enzyme inhibitor and was extubated with normal oxygen saturations.

Follow-up evaluation of the ASD two days post stabilization with transesophageal echocardiography revealed a thin, dyskinetic distal anterior septum and apex, a sinus venous ASD, and normal LV and RV systolic function. Right heart pressure was normal. The patient was feeling well without symptoms, and the baby was doing well. The normalization of RV size/function and right heart pressure suggested the ASD was not hemodynamically significant.

Case review raised a question of an apical myocardial infarction due to spontaneous coronary artery dissection (SCAD) versus embolism to the coronary tree. ECG analysis supported the notion of an anterior infarct, as the initial ECG was normal (Figure 1C).



**Figure 1C:** Normal ECG prior to delivery

The post-delivery ECG revealed loss of R waves across the precordium (Figure 1A).

This raised a suspicion that the initial insult was an unrecognized coronary artery dissection versus a paradoxical embolism across the ASD to the coronary circulation. The interventional team felt the angiogram represented a dissection.

Because of the apical infarct, cardiovascular surgery requested cardiac catheterization, which revealed an occluded left anterior descending coronary artery (Figure 1D). This occluded vessel retrospectively identified the etiology of the post-delivery hemodynamic instability.



**Figure 1D:** Coronary angiography reveals an occluded LAD. (Blue arrow)

### Pregnancy and SCAD (P-SCAD)

SCAD is a rare condition with an incidence of 0.1% for all patients referred for coronary angiography [1]. Mean age at presentation is 35-40 years, and >70% of SCAD cases are female. 2 Patients with spontaneous coronary artery dissection are divided into four etiologic groups: peripartum, atherosclerotic, idiopathic, and vasculitic in patients with connective tissue disease. 2 This classification does not cover all etiologies of SCAD.

33% of P-SCAD cases occur in the peripartum period, with one-third late in pregnancy and two-thirds in the early puerperal period [2]. The peak incidence is two weeks after delivery. The role of the peripartum period in the pathogenesis is an enigma. Theories include hormonal changes, such as high estrogen levels, resulting in subtle changes in arterial wall architecture with ensuing susceptibility to spontaneous dissections. These changes include hypertrophy of the smooth muscle cells, loosening of the intracellular matrix owing to increase in acid mucopolysaccharides, and decreased collagen production in the media [3, 4]. Additionally, increased total blood volume, high cardiac output, and shearing forces during labor may result in increased wall stress. Thus, hemodynamic and hormonal increase the risk of intimal tears. Patients in the pregnancy subset with underlying connective tissue disorders, such as Marfan syndrome, Ehler-Danlos type 4, and systemic lupus erythematosus with vasculitis, may be at even higher risk for P-SCAD [5, 6].

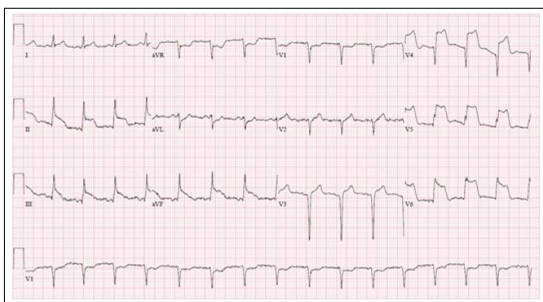
### Pearls of SCAD

P-SCAD is the consequence of an intramural hematoma of the coronary artery, resulting in a false lumen compressing the true lumen and myocardial ischemia [7]. Clinical presentation ranges from unstable angina to sudden cardiac death. A young woman with angina and ECG changes should arouse high suspicion for SCAD, and urgent evaluation should be considered. In young women, the left anterior descending coronary artery is the most common location. 2 When angiography is performed, the approach ranges from mostly conservative to rarely revascularization with stenting depending on the angiographic and clinical circumstances.

### Case 2

29-Year-Old Female in Sixth Month of Pregnancy and No History of Cardiovascular Disease Presenting with Chest Pain

The patient had a prior miscarriage two years ago, which resulted in depression. Resting at home, she developed chest pain. Emergency medical responders performed an ECG that revealed inferolateral ST elevation (Figure 2). In the emergency room, she developed ventricular fibrillation requiring cardiac defibrillation.



**Figure 2:** ECG revealing ST elevation inferiorly and laterally

Echocardiography revealed an akinetic apex with hypercontractile basal segments. LVEF was 35%. Angiography revealed normal coronary arteries. Laboratory results revealed a mildly elevated troponin and normal electrolytes.

Treatment included a beta-blocker and diuretic. She recovered without symptoms. Repeat echocardiography on Day 7 revealed normalization LVEF.

The patient was discharged home and at nine months gave birth via cesarean section to a healthy baby.

### Takotsubo Cardiomyopathy (TC) in Pregnancy

This case represents TC in pregnancy with a malignant, life-threatening rhythm disturbance. There is no evidence that pregnancy predisposes to a stress-induced cardiomyopathy. The majority of stress-induced cardiomyopathies recover to normal LV function. If LV dysfunction persists, guideline-directed medical treatment is the standard of care, and the criteria for an implantable cardioverter-defibrillator are determined on a case-by-case basis [7, 8].

### Takotsubo Cardiomyopathy (TC) in Pregnancy Pearls

TC is a unique form of transient non-ischemic cardiomyopathy that occurs in a setting provoked by a stressor—physical, emotional, or both. The syndrome most commonly occurs in post-menopausal females. The characteristic clinical syndrome of TC involves acute LV dysfunction with distinctive echocardiographic features of apical to mid ventricular hypo- to akinesis and sparing of the basal myocardium in the absence of significant obstructive coronary artery disease. Alternatively, patients may exhibit reverse TC with basal hypokinesis and hyperkinesia of the apical and mid segments of the LV. Occurrence in premenopausal women is rare, and a literature search revealed few cases in pregnancy [9, 10].

Patients present with chest pain (70-90%) and dyspnea (20%); other, less common, presentations include syncope, pulmonary edema, and cardiac arrest [8]. Dynamic ECG changes and elevated cardiac biomarkers are usually present [9]. Coronary angiography typically reveals no evidence of coronary obstruction. However, patients with coronary artery disease can experience stress-induced cardiomyopathy. Symptoms can be severe and lead to death in 2% of patients [10].

The most common ECG changes reported in TC are ST-segment elevations in precordial leads. Subsequent deep, symmetrical T-wave inversion in multiple leads and Q-wave formation are frequently found [8,11]. Also, QT interval prolongation can be present [12, 13]. The clinical symptoms and ECG changes at presentation makes differentiation of TC from acute coronary syndrome challenging. Most TC patients present with elevated cardiac biomarkers within 24 hours, but levels are lower than anticipated given the extent of wall motion abnormalities and ECG findings [14, 15].

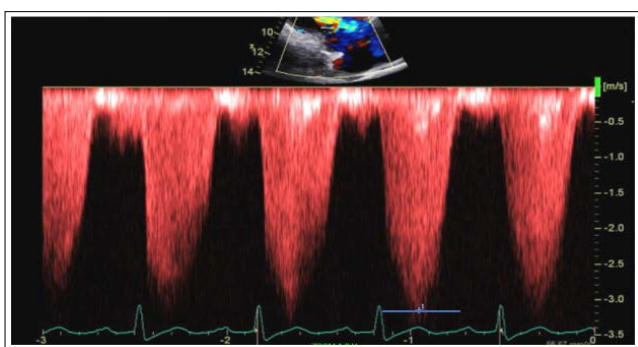
Management focuses on supportive care in the acute phase, while avoiding vasopressor medications because a hyperdynamic basal LV can result in outflow tract obstruction. Mortality is low if patients survive the initial critical period and, typically recover in full. Recurrence has been reported but is rare. TC follow-up revealed the rate of major adverse cardiac and cerebrovascular events was 9.9% per patient year, and the rate of death was 5.6% per patient year [15].

**Case 3**

**25-Year-Old Female Presenting to Emergency Room with SOB and Hypoxia**

The patient had delivered a full-term baby girl via cesarean section and was discharged five days previously. She had SOB post-delivery and upon discharge, and it progressively worsened.

Chest X-ray revealed diffuse alveolar infiltrates suggestive of pulmonary edema. She was hemodynamically stable, BP 160/90 mmHg and HR 95 bpm. Respiratory rate was elevated at 20 breaths per minute. Her jugular venous pressure was elevated. She had a 2/4-holosystolic-murmur at the right 4th intercostal space. There was no leg edema. Echocardiography revealed an enlarged RV with decreased systolic function and severe TR. Estimated pulmonary artery systolic pressure (PASP) was 55 mmHg (Figure 3). LVEF was normal.



**Figure 3:** The RV - RA gradient + RAP ~ 50- 55 mm Hg

Treatment included a beta-blocker, diuretic, and low-dose angiotensinogen converting enzyme inhibitor. CT pulmonary angiogram was negative for PE. The working exclusionary diagnosis became amniotic fluid embolism (AFE).

She stabilized clinically with improved hemodynamics and respiratory status. She was discharged on medical therapy. In six weeks, repeat echocardiography revealed a normal LVEF, decreased RV size, mild TR, and an estimated PASP of 25 mmHg. The patient and baby were doing well.

**PE Versus AFE in Pregnancy – A Challenging Differential.**

Pregnancy and the puerperium are risk factors for venous thromboembolism and anaphylactic syndrome of pregnancy-related AFE. Both PE and AFE should be considered in a pregnant or postpartum patient with hypotension, hypoxia, and right heart failure [16].

There are no clinical symptoms or signs specific for (PE). There is an overlap between symptoms of PE and normal physiologic changes of pregnancy [16]. Thus, identifying a clinically important PE during pregnancy, or postpartum, is challenging.

Diagnostic considerations include D-dimer levels, leg ultrasound imaging, and CT pulmonary angiography (CTPA). CTPA has high sensitivity (100%) and specificity (89%) for detecting PE [17, 18]. Rapid diagnosis leads to appropriate anticoagulation therapy and improved survival of infant and mother. Undiagnosed PE has a mortality rate approaching 30%, which is reduced to ≈2.8% when diagnosed and treated appropriately [19, 20].

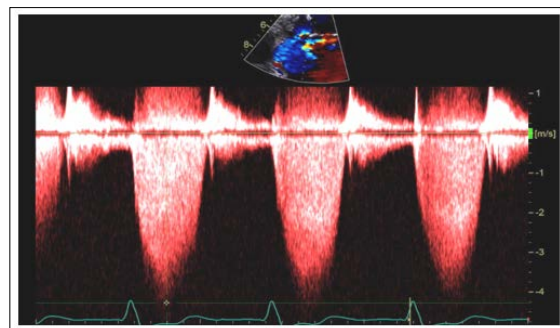
**PE Versus AFE in Pregnancy Pearls**

When the CTPA is negative for a PE there should be consideration of AFE. AFE is a rare and potentially fatal obstetric emergency that may occur during pregnancy, but is commonly identified during labor and the early postpartum period. Risk factors include fetal distress, maternal age, placental abnormalities, eclampsia, polyhydramnios, cervical lacerations, cesarean section, and rapid delivery. AFE is an allergic-like reaction to amniotic fluid entering the mother’s circulation. Management is monitoring and treating the respiratory, cardiovascular, and hematological perturbations that arise. AFE is not preventable, but it is essential to recognize it and treat it expectantly [21].

**Case 4**

**21-Year-Old Primigravida Female Presenting to Emergency Room at 32 Weeks of Gestation**

The patient had a seven-week history of SOB and tachycardia. Physical examination revealed tachycardia (110 bpm) and tachypnea. BP was 100/60 mmHg. Jugular venous pressure was elevated to the angle of the jaw. She had a right parasternal heave and lower-extremity pitting edema. Her second heart sound was accentuated. She had a pan-systolic murmur at the right sternal border. ECG revealed P pulmonale, right axis deviation (RAD), and RV strain pattern. Chest X-ray revealed prominent RV silhouette and dilated pulmonary arteries. Echocardiogram revealed severe RV dilation and hypertrophy, moderate RV systolic dysfunction, a large right atrium (RA), severe pulmonary regurgitation, severe TR, plethoric inferior vena cava, RV systolic pressure of 80 mmHg (Figure 4), and systolic flow reversal in the hepatic vein. Mitral inflow Doppler and tissue Doppler were normal.



**Figure 4:** RV-RA gradient + RAP ~ 75-80 mm Hg

Duplex ultrasound of the lower extremities and a negative D-dimer suggested low probability for pulmonary embolic disease. Blood screening revealed no evidence of vasculitis or connective tissue disease.

The clinical diagnosis was primary pulmonary hypertension (PPH). The patient was started on a diuretic, sildenafil, and an intravenous infusion of heparin for two days prior to delivery.

An elective cesarean section under general anesthesia was performed with a pulmonary artery catheter and arterial line. PASP was 100 mmHg. She was intubated and ventilated. Nitric oxide was started after induction of anesthesia. A healthy baby was delivered. The PASP was 90 mmHg. The patient was extubated seven hours post-delivery. Nebulized iloprost was initiated and allowed weaning of the nitric oxide. Heparin was initiated. Repeat Doppler echocardiography revealed an RV systolic pressure of ≈90 mmHg. She was discharged from the hospital 11 days after arrival.

Follow-up in the pulmonary hypertension clinic was arranged.

### PPH and Pregnancy Pearls

Pulmonary hypertension in pregnancy is high-risk. Women with PPH should be strongly advised against pregnancy. In early pregnancy, there should be a careful discussion regarding termination because of the high risk to fetus and mother [12]. Hemodynamic deterioration occurs in the second trimester with symptoms of dyspnea, syncope, and chest pain. This symptomatic period corresponds to the physiologic increase in cardiac output and blood volume of 40%. When PPH is not diagnosed until late in pregnancy an elective delivery with cesarean section is preferred. Delivery should be planned for 32-34 weeks of gestation. An opiate-based general anesthetic is appropriate for a failing RV. The use of pulmonary vasodilators such as oxygen, nitric oxide, and iloprost are recommended. A multidisciplinary approach between obstetrics, cardiology, and anesthesia is of utmost importance for a successful maternal-fetal outcome. PPH complicating pregnancy remains a fatal condition with deaths occurring between two and nine days post-delivery, usually from RV failure. Iloprost and nitric oxide therapies may have a role in controlling PPH, but there is no evidence of increased survival [22].

### Case 5

#### 27-Year-Old Caucasian Female Presenting to Emergency Department Four Days After Giving Birth with SOB for Two Days

SOB manifested in this patient as a respiratory rate of 20 breaths per minute, use of accessory muscles of respiration, and oxygen saturation of 81% on room air. She had previously had good health except for developing gestational hypertension during her last month of pregnancy and dependent peripheral edema. She was treated with labetalol 100 mg orally, twice daily, for BP management.

In the emergency department, BP was 160/90 mmHg, pulse 95 bpm, respiratory rate 22 breaths per minute, and oxygen saturation 95% on 2 liters of oxygen with nasal cannula. Auscultation revealed an S3. Lungs were clear. There was mild peripheral edema, but no calf tenderness. D-Dimer was normal, and brain natriuretic peptide was elevated to 2,000 pg/ml. ECG revealed sinus rhythm. Chest X-ray revealed cardiomegaly with increased vascular congestion. CT revealed no PE. Echocardiography revealed an ejection fraction of 35%. A diuretic, labetalol, and lisinopril were initiated. Six months later, repeat echocardiogram revealed normal LV systolic function.

### Pregnancy and Peripartum Cardiomyopathy (PPCM) Pearls

PPCM occurs one in 4,000 live births in the United States [23]. The development of heart failure in the last month of pregnancy or within 5 months of delivery, absence of an alternative identifiable cause of heart failure, absence of recognizable heart disease before the last month of pregnancy, and LVEF <45% characterize PPCM in pregnancy [24]. Mortality is as high as 25% [25].

Clinical features of PPCM include symptoms and signs of heart failure. BNP may be elevated. ECG may be normal. Chest X-ray typically has an enlarged cardiac silhouette. Echocardiography is crucial to the diagnosis [26]. Treatment is salt restriction, diuretics, beta-blockers, and digoxin. Hydralazine can be used during pregnancy to reduce afterload.

In ≈50% of patients, LVEF normalizes. Despite recovery of ventricular function, a second pregnancy is not recommended because >30% of PPCM patients recur. During the period of depressed LV systolic function, the mother is at risk for rhythm disturbances. Ventricular arrhythmias should trigger an electrophysiology consult for advice on therapeutic approach based on heart structure and function [12].

### Case 6

#### 29-Year-Old Female, With Body Mass Index of 20, With History of Paroxysmal Supraventricular Tachycardia from Age 14 Years

The patient became more symptomatic with tachycardia during her pregnancy. ECG, 2D echocardiogram, and Holter monitor were normal. A longer episode of tachycardia was documented on ECG at 34 weeks of gestation, revealing wide complex tachycardia at a rate of 160 bpm (Figure 5). During this episode, the patient was hemodynamically stable. She was started on metoprolol 50 mg/day. Because of concerns that induction of labor may exacerbate her cardiac problems, a cesarean section was performed, which took place two weeks later. A healthy male was delivered. Spinal anesthesia-induced hypotension was prevented with an infusion of ephedrine 30 mg and phenylephrine 400 mg in 500 ml of normal saline, which was commenced immediately after the spinal injection. Syntocinon 5 units were administered slowly after delivery without problem. She was discharged home three days later.

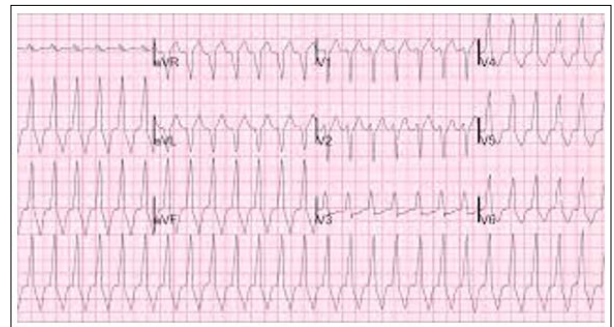


Figure 5: Wide complex tachycardia, LBBB and inferior axis

The patient followed up with the electrophysiology service to discuss ablation therapy versus continued medical therapy for her RV outflow tract ventricular tachycardia.

### Cardiac Ventricular Arrhythmias and Pregnancy Pearls

Rhythm disturbances occur during pregnancy in women with and without structural heart disease. Arrhythmias may manifest for the first time in pregnancy, or pregnancy may prompt an escalation of rhythm disturbances in women with a known pre-existing rhythm disorder [27].

Women with established arrhythmias or underlying structural heart disease are at the highest risk. Women with congenital heart disease are at particularly high risk for arrhythmias. Since arrhythmias are frequently associated with acquired or congenital structural heart disease, any pregnant patient with arrhythmias should be evaluated with an ECG and transthoracic echocardiogram [28, 29].

Arrhythmogenesis in pregnancy has been ascribed to the hemodynamic, hormonal, and autonomic changes of pregnancy. Palpitations occur during pregnancy, and the sensation of

palpitations in the absence of arrhythmias may be related to the enhanced cardiac output, increased heart rate, decreased peripheral resistance, and increased stroke volume [30].

Ventricular premature beats (VPBs) are frequently detected in pregnant women, though these produce few or no symptoms in most. No therapy is required for VPBs in asymptomatic patients. Patients should be told that the VPBs are benign [31].

Ventricular arrhythmias are rare in pregnancy. Ventricular tachycardia can occur in patients with and without structural heart disease. The risk is highest with structural heart disease. Structural diseases including hypertrophic cardiomyopathy, PPCM, congenital heart disease, and valvular disease can be associated with ventricular tachycardia in pregnancy. Myocardial infarction may occur in pregnancy and may be complicated by ventricular tachycardia or fibrillation. Pregnant patients with ion channelopathies are also at risk of ventricular tachycardia [32].

In pregnant patients with ventricular tachycardia without structural heart disease, the arrhythmia is considered to be idiopathic. The most common form is idiopathic RV outflow tract tachycardia that manifests as left bundle branch block and inferior axis. LV outflow tract tachycardia is less common and manifests as left bundle branch block and inferior axis. Idiopathic ventricular tachycardia usually has a benign prognosis; RV outflow tract tachycardia responds well to beta-blockers and LV outflow tract tachycardia to verapamil [33, 34]. These idiopathic ventricular tachycardias are treatable with ablation post-delivery.

Long QT syndrome has high risk for ventricular tachycardia in the postpartum period. This may be related to the decrease in HR and associated increase in QT duration. These patients should be treated with beta-blocker therapy during pregnancy and in the postpartum period [35].

Acute treatment of sustained ventricular arrhythmias in pregnant patients is similar to that in non-pregnant women. Decisions regarding drug therapy, electrical therapy, ablation, and implantation of defibrillators require careful coordination with the electrophysiology team [12].

#### Case 7

##### **28-Year-Old Woman with History of Idiopathic Dilated Cardiomyopathy (DCM) and LVEF 20% Presents at 21 Weeks of Gestation with Heart Failure**

The patient's medical history included asthma, obesity, obstructive sleep apnea, diabetes, and active tobacco use. She had an implantable cardioverter-defibrillator that had been placed for primary prevention. She was considered at prohibitively high risk for adverse maternal and fetal outcomes. She adamantly refused termination of pregnancy.

She was hospitalized for congestive heart failure at 26 weeks, which was managed with in-hospital with medical therapy. She developed progressive heart failure and declining LV systolic function.

At 28 weeks of gestation the baby was delivered by urgent cesarean section with an epidural and conscious sedation. A pulmonary artery catheter and arterial line were placed for hemodynamic monitoring. Her postpartum course was complicated by a transient ischemic attack. Subsequently, she

developed hemodynamically unstable ventricular tachycardia that was unresponsive to resuscitation. The baby survived.

#### **Pregnancy in Patients with Known DCM Pearls**

Pregnant women with DCM have been counseled to avoid pregnancy based upon observations of adverse fetal and maternal outcomes [36]. Increase in intravascular volume and cardiac output during pregnancy leads to a greater risk of complications for women with DCM, particularly in the third trimester, when the hemodynamic burden is extreme. A prior history of cardiac events is highly predictive of pregnancy-related cardiac complications [37].

Patients with an LVEF <40% are at higher risk for adverse fetal and maternal outcomes. These patients should be counseled regarding the potential adverse outcomes. In patients with class 2-3 heart failure symptoms and an LVEF <40% in the first and second trimester should be counseled about the risks of carrying the pregnancy to term [38].

Management of labor and delivery in DCM patients requires a multidisciplinary approach including obstetrics, anesthesiology, and cardiology. The patient should be monitored continuously throughout labor, delivery, and the early postpartum period, including ECG monitoring and noninvasive BP monitoring. Invasive monitoring should be decided on an individual basis and be related to the complexity of the patient's hemodynamic status [39].

Management of labor and delivery requires a multidisciplinary approach. When heart failure becomes refractory to medical therapy and the patient is on the precipice of hemodynamic instability, delivery should occur. When possible, delivery should be through a vaginal approach because of the blood loss associated with cesarean section.

NYHA functional class and LV systolic function can be used to identify pregnant women with DCM at highest risk for cardiac complications during pregnancy. When compared with non-pregnant women, pregnancy seems to have a negative impact on the clinical course for women with DCM, at least over the short term. Fetal and/or neonatal complications are also increased in mothers with DCM, and the risk is magnified by the presence of both cardiac and obstetric issues.

#### Case 8

##### **29-Year-Old Female, Gestational Age 35 Weeks, Presents to Obstetrics Clinic with Acute SOB, Fever, and Cough with Yellowish Sputum**

Chest X-ray was consistent with pneumonia. History revealed cryptogenic cirrhosis and mild preeclampsia. She was being treated with methyldopa for her hypertension and antibiotics for her pneumonia. Cardiac examination revealed a hyperdynamic precordium with a systolic ejection murmur and a 3/6 diastolic murmur.

Echocardiography revealed a bicuspid aortic valve with moderate to severe aortic regurgitation, a dilated aorta (45 mm), and normal ventricular function. Treatment with BP medications, oxygen, antibiotics, a bronchodilator, antipyretic, and diuretic was initiated. Further evaluation revealed normal liver enzymes and a platelet count of 100,000/mm<sup>3</sup>, which improved to 100,000/

mm3 on platelet transfusion. On the second day of admission, the patient decompensated with progressive SOB and oxygen saturation of 89% with facemask at FiO<sub>2</sub> of 40%. The physical examination revealed a holodiastolic diastolic murmur and a wide pulse pressure of 150/50 suggesting severe aortic regurgitation. No features of fetal distress were evident. Emergent cesarean section delivery was planned. There was continuous ECG and oxygen saturation monitoring throughout the delivery. She had no hemodynamic instability or arrhythmias. The postoperative period was uneventful. She was discharged ambulatory on the sixth postoperative day.

### **Bicuspid Aortic Valve (BAV) With Associated Dilated Aorta and Pregnancy Pearls**

Women with BAV should be counseled about potential risks prior to and during pregnancy. Potential risks include heritable congenital heart disease, aortic root enlargement with potential for dissection, and aortic valve issues including aortic stenosis and/or aortic regurgitation.

Pregnant women with BAV and aortic root dilation are at risk for spontaneous aortic dissection, usually in the third trimester; this is especially pertinent if there is coarctation [40]. Pregnancy-associated increases in HR, BP, and cardiac output result in increased aortic root stress and may play a role in promoting aortic root dilation and dissection. For women planning pregnancy, prophylactic aortic root/ascending aorta surgery should be discussed if the aortic diameter is >45 mm, if the rate of increase in the diameter is >5 mm/year, and if there is moderate or severe aortic regurgitation [39]. All pregnant women with thoracic aortic dilation should maintain strict blood pressure control, with the goal being 130/80 mmHg. What is unknown is whether pregnancy changes the natural history of BAV with aortopathy, as demonstrated in Marfan syndrome [41].

BAV that is stenotic poses more complexity to pregnancy than BAV that is regurgitant. All women with symptomatic BAV stenosis and asymptomatic patients with high-risk features, such as severe aortic stenosis and LVEF <40%, should delay conception until the aortic stenosis has been managed surgically or percutaneously. Moderate to severe aortic regurgitation with preserved LVEF is usually well-tolerated in pregnancy.

### **Conclusion**

This manuscript attempts to introduce the readership to some interesting cardiovascular conditions that surface during pregnancy. Hopefully the cases will act as a stimulus to reading the comprehensive 2018 ESC guidelines for management of cardiovascular disease during pregnancy. The guidelines provide an exhaustive review of the fascinating cardiovascular conditions that physicians may encounter when caring for patients during pregnancy.

### **References**

- [1] Maeder M, Ammann P, Angehrn W, Rickli H. Idiopathic spontaneous coronary artery dissection: incidence, diagnosis and treatment. *Int J Cardiol.* 2005; 101: 363-369.
- [2] DeMaio SJ, Jr, Kinsella SH, Silverman ME. Clinical course and long-term prognosis of spontaneous coronary artery dissection. *Am J Cardiol.* 1989; 64: 471-474.
- [3] Heefner WA. Dissecting hematoma of the coronary artery. A possible complication of oral contraceptive therapy. *Jama.* 1973; 223: 550-551.
- [4] Bonnet J, Aumailley M, Thomas D, Grosogeat Y, Broustet JP et al. Spontaneous coronary artery dissection: case report and evidence for a defect in collagen metabolism. *Eur Heart J.* 1986; 7: 904-909.
- [5] Judge DP, Dietz HC. Marfan's syndrome. *Lancet.* 2005; 366: 1965-1976.
- [6] Adès LC, Waltham RD, Chiodo AA, Bateman JF. Myocardial infarction resulting from coronary artery dissection in an adolescent with Ehlers-Danlos syndrome type IV due to a type III collagen mutation. *Br Heart J.* 1995; 74: 112-116.
- [7] Kamineni R, Sadhu A, Alpert JS. Spontaneous coronary artery dissection: report of two cases and a 50-year review of the literature. *Cardiol Rev.* 2002; 10: 279-284.
- [8] Bybee KA, Kara T, Prasad A, Lerman A, Barsness GW, et al. Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. *Ann Intern Med.* 2004; 141: 858-865.
- [9] Lyon AR, Rees PS, Prasad S, Poole-Wilson PA, Harding SE. Stress (Takotsubo) Cardiomyopathy--a novel pathophysiological hypothesis to explain catecholamine-induced acute myocardial stunning. *Nat Clin Pract Cardiovasc Med.* 2008; 5: 22-29.
- [10] Sharkey SW, Lesser JR, Maron MS, Maron BJ. Why not just call it tako-tsubo cardiomyopathy: a discussion of nomenclature. *J Am Coll Cardiol.* 2011; 57: 1496-1497.
- [11] Akashi YJ, Goldstein DS, Barbaro G, Ueyama T. Takotsubo cardiomyopathy: a new form of acute, reversible heart failure. *Circulation.* 2008; 118: 2754-2762.
- [12] Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, Lundqvist CB, Cifkova R, et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. *Eur Heart J.* 2018; 39: 3165-3241.
- [13] Song BG, Park SJ, Noh HJ, Jo HC, Choi Jo, et al. Clinical characteristics, and laboratory and echocardiographic findings in takotsubo cardiomyopathy presenting as cardiogenic shock. *J Crit Care.* 2010; 25: 329-35.
- [14] Hurst RT, Prasad A, Askew JW 3rd, Sengupta PP, Tajik AJ. Takotsubo cardiomyopathy: a unique cardiomyopathy with variable ventricular morphology. *JACC Cardiovasc Imaging.* 2010; 3: 641-649.
- [15] Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu, et al. Clinical Features and Outcomes of Takotsubo (Stress) Cardiomyopathy. *N Engl J Med.* 2015; 373: 929-938.
- [16] Togli MR, Weg JG. Venous thromboembolism during pregnancy. *N Engl J Med.* 1996; 335: 108-14.
- [17] Epiney M, Boehlen F, Boulvain M, Reber G, Antonelli E, et al. D-dimer levels during delivery and the postpartum. *J Thromb Haemost.* 2005; 3: 268-71.
- [18] Perrier A, Roy PM, Sanchez O, Gal GL, Meyer G, et al. Multidetector-row computed tomography in suspected

- pulmonary embolism. *N Engl J Med.* 2005; 352: 1760-1768.
- [19] Rodger M, Wells PS. Diagnosis of pulmonary embolism. *Thromb Res.* 2001; 103: V225-V238.
- [20] Guidelines on diagnosis and management of acute pulmonary embolism. Task Force on Pulmonary Embolism, European Society of Cardiology. *Eur Heart J.* 2000; 21: 1301-36.
- [21] Amniotic Fluid Embolism Foundation. What is Amniotic Fluid Embolism? <http://afesupport.org/amniotic-fluid-embolism-faqs/>. Accessed on June 2, 2021.
- [22] Hemnes AR, Kiely DG, Cockrill BA, Safder Z, Wilson VJ, et al. Statement on pregnancy in pulmonary hypertension from the Pulmonary Vascular Research Institute. *Pulm Circ.* 2015; 5: 435-65.
- [23] Libby P, Bonow RO, Mann DL, Zipes DP. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed. 8th ed. Philadelphia, PA: Saunders. 2007; <https://www.amazon.in/Braunwalds-Heart-Disease-Textbook-Cardiovascular/dp/0323463428>.
- [24] Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. *Jama.* 2000; 283: 1183-8.
- [25] Abboud J, Murad Y, Chen-Scarabelli C, Saravolatz L, Scarabelli TM. Peripartum cardiomyopathy: a comprehensive review. *Int J Cardiol.* 2007; 118: 295-303.
- [26] Davidson NM, Parry EH. The etiology of peripartum cardiac failure. *Am Heart J.* 1979; 97: 535-6.
- [27] Li JM, Nguyen C, Joglar JA, Hamdan MH, Page RL. Frequency and outcome of arrhythmias complicating admission during pregnancy: experience from a high-volume and ethnically-diverse obstetric service. *Clin Cardiol.* 2008; 31: 538-41.
- [28] Siu SC, Sermer M, Colman JM, Alvarez AN, Mercier LA, et al. Prospective multicenter study of pregnancy outcomes in women with heart disease. *Circulation.* 2001; 104: 515-21.
- [29] Drenthen W, Pieper PG, Roos-Hesselink JW, Van Lottum WA, Voors AA, et al. Outcome of pregnancy in women with congenital heart disease: a literature review. *J Am Coll Cardiol.* 2007; 49: 2303-11.
- [30] Silversides CK, Colman JM. Physiological changes in pregnancy. In: Oakley C, Warnes CA (eds.). *Heart Disease in Pregnancy*, 2nd Ed., London: BMJ Books. 2008; 6.
- [31] Shotan A, Ostrzega E, Mehra A, Johnson JV, Elkayam U. Incidence of arrhythmias in normal pregnancy and relation to palpitations, dizziness, and syncope. *Am J Cardiol.* 1997; 79: 1061-4.
- [32] Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, et al. ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Circulation.* 2006; 114: e385-484.
- [33] Lin FC, Finley CD, Rahimtoola SH, Wu D. Idiopathic paroxysmal ventricular tachycardia with a QRS pattern of right bundle branch block and left axis deviation: a unique clinical entity with specific properties. *Am J Cardiol.* 1983; 52: 95-100.
- [34] Cleary-Goldman J, Salva CR, Infeld JI, Robinson JN. Verapamil-sensitive idiopathic left ventricular tachycardia in pregnancy. *J Matern Fetal Neonatal Med.* 2003; 14: 132-5.
- [35] Rashba EJ, Zareba W, Moss AJ, Hall J, Robin J, et al. Influence of pregnancy on the risk for cardiac events in patients with hereditary long QT syndrome. *LQTS Investigators. Circulation.* 1998; 97: 451-6.
- [36] Siu SC, Sermer M, Harrison DA, Grigoriadis E, Liu G, et al. Risk and predictors for pregnancy-related complications in women with heart disease. *Circulation.* 1997; 96: 2789-94.
- [37] Siu SC, Colman JM, Sorensen S, Smallhorn JF, Farine D, et al. Adverse neonatal and cardiac outcomes are more common in pregnant women with cardiac disease. *Circulation.* 2002; 105: 2179-84.
- [38] Grewal J, Siu SC, Ross HJ, Mason J, Balint OH, et al. Pregnancy outcomes in women with dilated cardiomyopathy. *J Am Coll Cardiol.* 2009; 55: 45-52.
- [39] Howlett JG, McKelvie RS, Costigan J, Ducharme A, Holder EE, et al. The 2010 Canadian Cardiovascular Society guidelines for the diagnosis and management of heart failure update: Heart failure in ethnic minority populations, heart failure and pregnancy, disease management, and quality improvement/assurance programs. *Can J Cardiol.* 2010; 26: 185-202.
- [40] Anderson RA, Fineron PW. Aortic dissection in pregnancy: importance of pregnancy-induced changes in the vessel wall and bicuspid aortic valve in pathogenesis. *Br J Obstet Gynaecol.* 1994; 101: 1085-8.
- [41] Donnelly RT, Pinto NM, Kocolas I, Yetman AT. The immediate and long-term impact of pregnancy on aortic growth rate and mortality in women with Marfan syndrome. *J Am Coll Cardiol.* 2012; 60: 224-9.
- [42] Expert consensus document on management of cardiovascular diseases during pregnancy. *Eur Heart J.* 2003; 24: 761-81.