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VALVULAR HEART DISEASE

CASE REPORT: CLINICAL CASE

Ventricular Fibrillation Cardiac Arrest With Endocarditis During Pregnancy

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ABSTRACT

A 34-year-old pregnant woman collapsed at home without preceding symptoms. Following bystander cardiopulmonary resuscitation, ventricular fibrillation was documented and successfully reverted with an external shock. The electrocardiogram showed sinus rhythm, right bundle branch block, ST-segment depression, and T-wave inversion in anterior leads. She was then brought to the hospital for emergency management. (JACC Case Rep. 2024;29:102465)

PRESENTATION AND PAST MEDICAL HISTORY

A 34-year-old, previously well woman, who was in her 23+5 week of pregnancy, collapsed at home with no preceding symptoms. She received immediate bystander cardiopulmonary resuscitation. She was found in ventricular fibrillation (VF) and was successfully defibrillated with one shock. She was intubated and remained stable during the transfer to the hospital.

LEARNING OBJECTIVES

- To understand that in young patients with no cardiovascular risk factors, less common causes of VF need to be considered.
- To note that embolization through coronary circulation leading to myocardial infarction is a rare, but plausible, cause of cardiac arrest in the context of aortic valve endocarditis.
- To realize that in complex cases, prompt involvement of the relevant teams, that is, a multidisciplinary approach is required.
- To understand that serial echocardiography can be a useful monitoring tool.

DIFFERENTIAL DIAGNOSIS

In the context of the patient's pregnancy, younger age, and no comorbidities, differential diagnoses in the order of likelihood should include:

- Pulmonary embolism (given the increased risk associated with pregnancy)
- Arrhythmia secondary to undiagnosed cardiomyopathy/channelopathy
- Myocardial infarction due to spontaneous dissection, embolism, or early atherosclerosis
- Seizure related to undiagnosed eclampsia/HELLP (hemolysis, elevated liver enzymes and low platelets) syndromes

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ABBREVIATIONS AND ACRONYMS

AV = aortic valve

LV = left ventricle

VF = ventricular fibrillation

- Intracranial cause of collapse (ie, space-occupying lesion/cerebrovascular accident)
- Substance overdose/poisoning

INVESTIGATIONS

On examination, a diastolic murmur was heard at the lower left sternal edge. The patient's electrocardiogram showed sinus rhythm, a right bundle branch block pattern with unspecific T-wave inversion, and ST-segment depression anteriorly (leads V_2-V_3). The PR, QRS, and corrected QT intervals had normal duration. After stabilizing the patient, a set of blood tests was sent for urgent analysis. A blood gas analysis excluded significant metabolic, electrolyte, or hemoglobin abnormalities at the point of care (Table 1). Urine toxicology screen excluded substance misuse.

A portable chest x-ray did not reveal any obvious abnormality. A bedside transthoracic echocardiogram showed mild left ventricular (LV) dilation and mildly reduced systolic function alongside a diseased aortic valve (AV) with thickened leaflets, a small mobile structure on the AV, and a central jet of severe regurgitation (Figure 1). The ascending aorta appeared normal.

An extensive computed tomography scan ruled out large pulmonary embolism, acute aortic pathology, and intracranial abnormalities. Given her electrocardiogram findings and abnormal troponin result, the patient underwent coronary angiography that showed unobstructed coronary arteries (Figure 2).

Transesophageal echocardiogram confirmed severe aortic regurgitation of a thickened tricuspid AV with a small mobile structure (<1 cm) on the noncoronary cusp, but no evidence of aortic root abscess. No septal defects or shunts were detected (Figure 3). The patient was transferred to the intensive care unit.

TABLE 1 Blood Gas and Admission Blood Test Results

pH	7.35 (7.35-7.45)	Sodium	137 (135-146 mmol/L)
pCO_2	4.44 (4.27-6.40 kPa)	Potassium	4.5 (3.4-4.5 mmol/L)
pO_2	35.7 (11.1-14.4 kPa)	Ionized calcium	1.21 (1.15-1.29 mmol/L)
FiO_2	0.6 (60%)	Lactate	0.8 (0.5-1.6 mmol/L)
Hemoglobin	126 (120-175 g/L)	Glucose	5.1 mmol
White blood cell count	23.7 (4-11 × 10^9/L)	Neutrophils	21.5 (2.2-6.0 × 10^9/L)
Platelets	299 (150-450 × 10^9/L)	Creatinine	47 (45-120 μmol/L)
C-reactive protein	12.7 (<5 mg/L)	Urea	4.3 (3.3-5.0 mmol/L)
Magnesium	0.71 (0.7-1.0 mmol/L)	Calcium, corrected	2.15 (2.15-2.60 mmol/L)
Troponin I	1,115 (<16 ng/L)		

The normal range of each value is shown in parentheses.
FiO_2 = fraction of inspired oxygen; pCO_2 = partial pressure of carbon dioxide; pO_2 = partial pressure of oxygen.

MANAGEMENT

The patient remained hemodynamically stable. Fetal cardiotocography and ultrasound were reassuring. A single set of blood cultures collected before antibiotics and subsequent sets did not show any growth. There was no serological evidence of infection with *Brucella* spp. or *Coxiella burnetii*.

The transesophageal echocardiogram confirmed a vegetation, one of the modified Duke's major criteria for endocarditis.[1] Further evidence was required to refute or support this diagnosis as it would have had major implications for patient management.

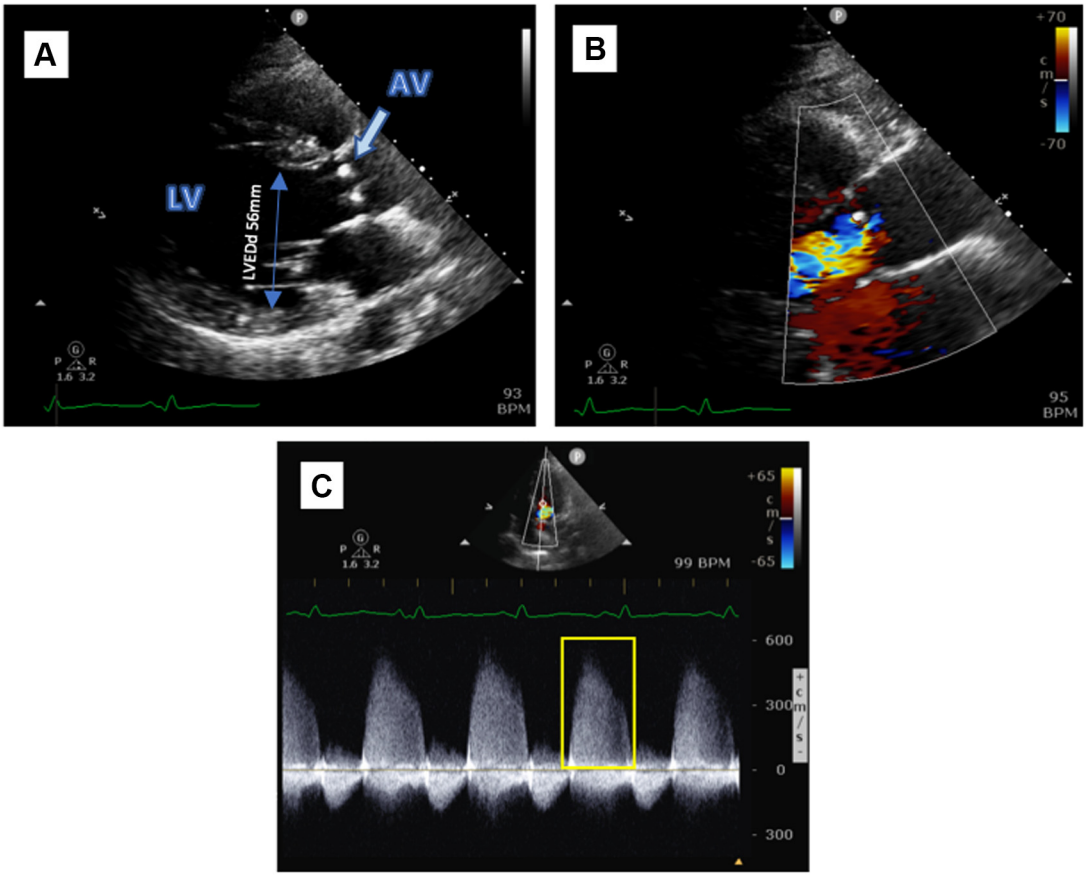
The patient had magnetic resonance imaging of the head that showed several small established embolic infarcts in the cerebellar hemispheres (Figure 4). The initial computed tomography aortogram did not suggest any infarcts or abscesses in the abdomen.

In light of embolic phenomena and an abnormal AV, a tentative diagnosis of embolic myocardial infarction due to endocarditis was assumed as the cause of VF arrest. She had empirical treatment for culture-negative endocarditis with antibiotics for 6 weeks.

Serial echocardiography toward the end of 32 weeks of gestation indicated a slow deterioration in the patient's LV function. Given this development, after consultation with the multidisciplinary team, a healthy baby boy was delivered by caesarean section, after completing 32 weeks of gestation without any complications.

Following delivery, decisions in the patient's management included the timing of AV surgery and the need for a defibrillator device for secondary prevention. The risks and benefits of the type of valve replacement, in particular the repercussions of anticoagulation on future pregnancies, were discussed with the patient. The patient proceeded to have cardiac magnetic resonance imaging with gadolinium contrast that showed normal biventricular function and 2 small areas of late enhancement in the high mid-inferolateral LV wall in keeping with small embolic infarcts (Figure 5).

FIGURE 1 **Presentation Bedside Transthoracic Echocardiogram**



Bedside transthoracic-echocardiogram shows the aortic valve (AV) as echogenic and abnormally thickened (A, arrow) with severe regurgitation on color Doppler (B). On 5-chamber view, the spectral Doppler shows an intense holodiastolic regurgitant flow (C, rectangle). BPM = beats/min; LV = left ventricle; LVEDd = left ventricular end-diastolic diameter.

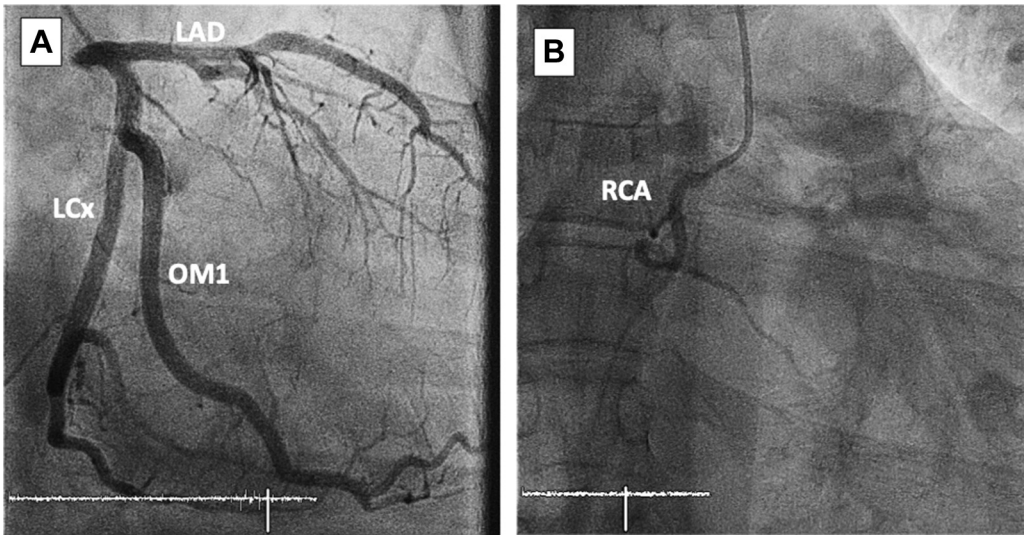
OUTCOME AND FOLLOW-UP

The patient recovered well in hospital post-caesarian section. The patient's blood pressure was meticulously monitored throughout. Two weeks after delivery, she underwent AV replacement surgery. On inspection, the explanted AV was thickened, but it did not show any obvious vegetation. A 25-mm bioprosthetic Inspirius replacement valve (Edwards Lifesciences) was used with excellent final result. The patient had an echocardiogram a week after her AV surgery that showed a well-seated and functioning prosthesis with normal ejection fraction and normalization of LV dimensions.

The AV histology showed intimal thickening, myxoid degeneration, and nodular calcific fibrosis with no acute inflammation or vegetation (**Figure 6**). Polymerase chain reaction analysis did not detect bacterial (16s) rDNA. Given the timing of surgery and the cycles of antibiotics, the yield of the pathology study was anticipated to be low.

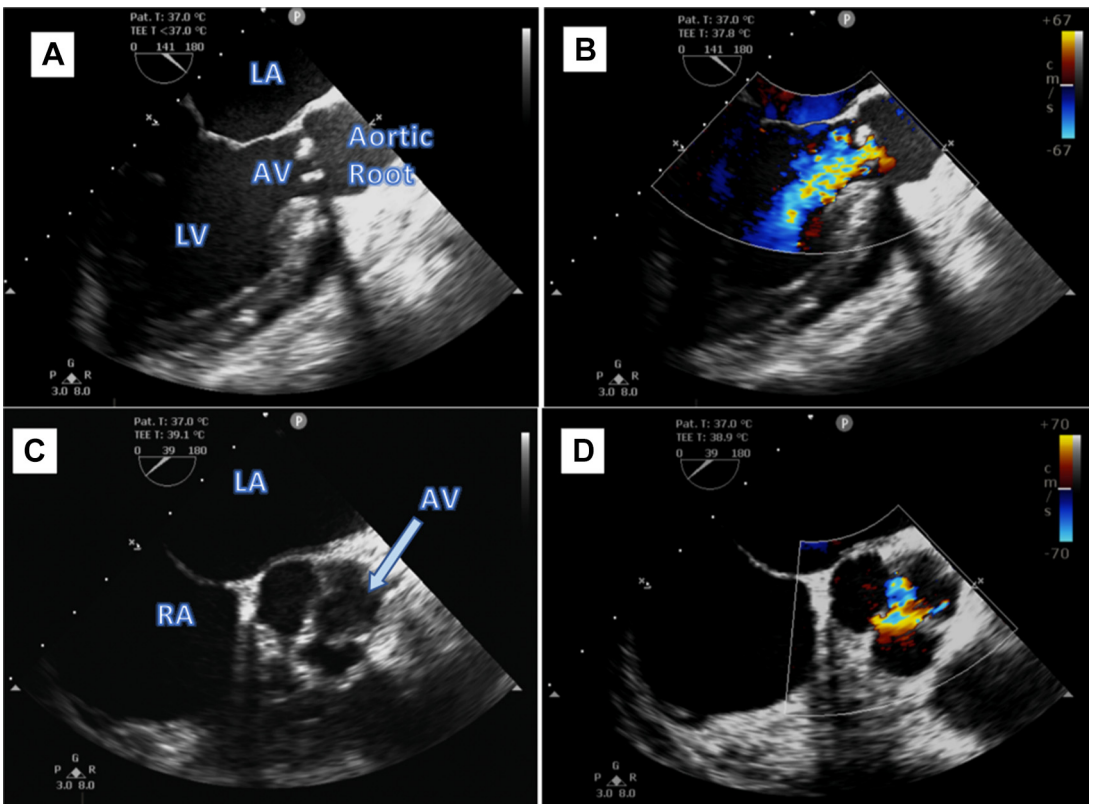
A number of multidisciplinary meetings concluded the cause of her VF arrest to most likely be attributable to an embolic phenomenon originating in the AV that travelled down the coronary circulation, leading to a minor myocardial infarction. In light of the recovery of the LV function, the small scar burden, and removal of the source of the

FIGURE 2 Coronary Angiogram Showing Unobstructed Vessels



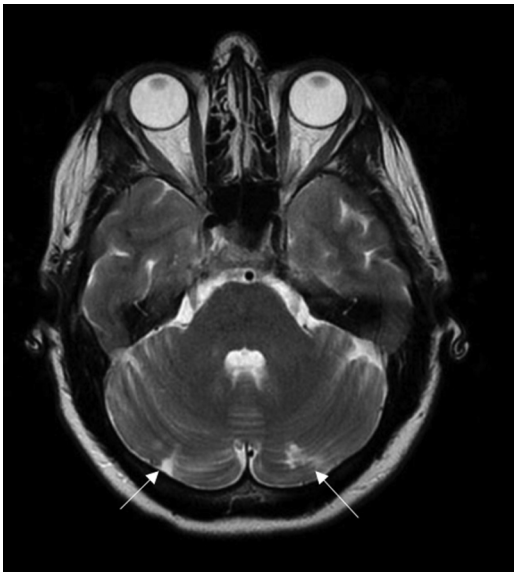
Dominant left coronary system (A) and a nondominant right coronary artery (B). LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; OM1 = first obtuse marginal; RCA = right coronary artery.

FIGURE 3 Transesophageal Echocardiogram



(A) The long axis of the heart, shows the aortic valve (AV) as the bright echogenic structure. (B) Color Doppler flow across the valve corresponding to a regurgitant jet. (C) The short axis (D shows the corresponding view with Doppler) confirming thickened aortic valve. LA = left atrium; LV = left ventricle; RA = right atrium.

FIGURE 4 Magnetic Resonance Imaging of the Brain



High-intensity T2 signal, without diffusion restriction (arrows) in keeping with established infarcts in the cerebellum.

emboli, she was deemed safe for discharge without the need for a defibrillator.

The patient was discharged after a 12-week in-hospital stay with a healthy baby and a recovered heart. The patient was followed up in the clinic with a repeat echocardiogram showing excellent valve function and an uneventful recovery.

DISCUSSION

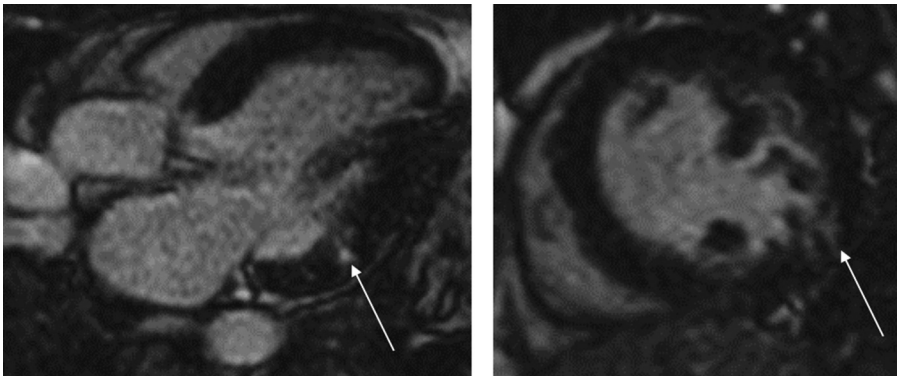
Endocarditis during pregnancy is associated with significant maternal and fetal morbidity and mortality.[2] Truly culture-negative infective endocarditis is uncommon (~5% of all cases) and caused by intracellular bacteria that cannot easily be cultured via conventional methods. Histological analysis of the infected tissue, polymerase chain reaction, and serological testing can prove helpful.[3] Noninfective causes are also rare, mostly limited to marantic endocarditis and related to systemic disease such as systemic lupus erythematosus. Whereas cerebral embolization is common (seen in up to 30% of cases),[4] coronary embolization leading to an acute coronary syndrome is rare.[5]

This case was managed by a multidisciplinary team composed of obstetricians, cardiothoracic surgeons, microbiologists, anesthetists, and cardiologists. Although the priority was to avoid risks to the mother's health due to hemodynamic repercussions and further embolic consequences of the diseased AV, careful consideration was also given to balance the risks of premature delivery to the fetus.

CONCLUSIONS

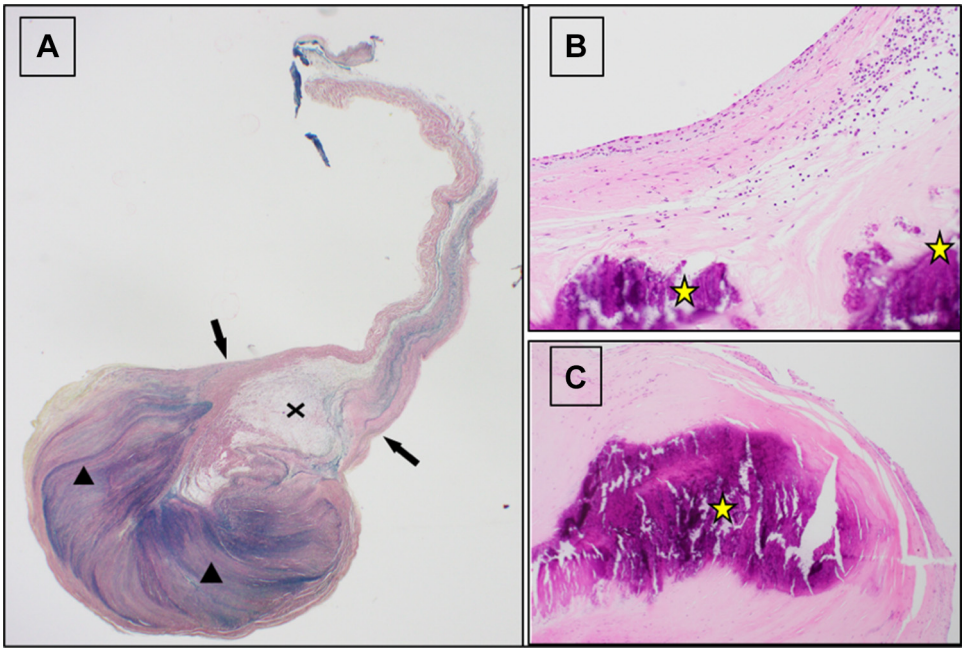
This case highlights the issues and subsequent management strategies in a complex case of culture-negative endocarditis with severe AV pathology in a pregnant patient. Monitoring with serial echocardiography, the timing of cardiac surgery, and the considerations around defibrillator therapy are key points in the decision-making process of this case.

FIGURE 5 Cardiac Magnetic Resonance Imaging: Borderline Left Ventricular Size With Normal Function



Two small areas of late enhancement in the high mid-inferolateral wall (arrows) in keeping with small infarcts.

FIGURE 6 Aortic Valve Histology



Intimal thickening (A, arrows), myxoid degeneration (A, X) and nodular calcific fibrosis (B and C, stars) with no evidence of acute inflammation or vegetation.

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