

Infective endocarditis in pregnancy requiring simultaneous emergent caesarean section and mitral valve replacement: a case report

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Background

Although infective endocarditis (IE) in pregnancy is rare, maternal and foetal mortality rates are very high. We herein report the successful treatment of a case of IE with simultaneous emergent caesarean section and mitral valve replacement performed at 27 weeks of gestation.

Case summary

A 29-year-old woman at 27 weeks of gestation was referred for congestive heart failure (HF) due to infective endocarditis (IE) with large mobile vegetations and overt disruption of the mitral valve. We held a multi-disciplinary conference and decided to perform mitral valve replacement immediately after caesarean section because of the high risk of embolism and sepsis, worsening and unstable haemodynamics, and sufficient foetal maturity for delivery. Although coronary artery embolization and asymptomatic multiple cerebral infarctions were observed, her post-operative course was uneventful. Ultimately, the patient was discharged 29 days after surgery. The neonate was treated in the NICU until the expected delivery date and was discharged home on Day 95 of life.

Discussion

Difficulties are associated with the selection of an operative plan and its timing for IE during pregnancy. Heart failure due to IE requires urgent surgery when medical treatment cannot stabilize the patient. However, cardiopulmonary bypass and medicine for pregnant women adversely affect the foetus. Therefore, the timing of surgery and delivery needs to be selected by a multi-disciplinary team and in consideration of the maternal condition and foetal maturity.

Keywords

Infective endocarditis • Pregnancy • Cardiac surgery • Case report

ESC Curriculum

4.3 Mitral regurgitation • 4.11 Endocarditis • 6.4 Acute heart failure • 7.5 Cardiac surgery • 9.8 Pregnancy with cardiac symptoms or disease

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Learning points

- Infective endocarditis (IE) is a rare complication of pregnancy that causes acutely decompensated heart failure.
- The timing of delivery and cardiopulmonary bypass depends on the maternal condition and foetal maturity.
- Since IE in pregnancy has a high risk of maternal and foetal morbidity and mortality, it needs to be managed by a multi-disciplinary team in consideration of the maternal condition and foetal maturity.

Introduction

Although infective endocarditis (IE) in pregnancy is rare, maternal and foetal morbidity and mortality rates are very high.^{1–3} We herein reported a case of IE in pregnancy that was successfully treated with simultaneous emergent caesarean section (CS) and mitral valve replacement (MVR).

Timeline

Time	Description
Day 1	Fever (>38°C), nasal discharge, and coughing
Day 5	Prescribed antibiotics
Day 9	Admission due to orthopnoea and the exacerbation of respiratory distress (Nohria–Stevenson classification: wet-warm) simultaneous emergent caesarean section and mitral valve replacement for infective endocarditis 4 weeks of intravenous ceftriaxone and sulbactam/ampicillin
Day 10	Extubation 4 h after returning to the ICU cardiac rehabilitation: 100 m walking the patient left the ICU for the general ward.
Day 14	Occlusion of the mid-left anterior descending artery and peripheral right coronary artery on coronary computed tomographic angiography.
Day 15	Multiple asymptomatic cerebral infarctions detected on head magnetic resonance imaging.
Day 37 (on Day 29 after admission)	Discharged
Day 103 (on Day 95 after admission)	The neonate was discharged.

Case presentation

A 29-year-old Asian woman (gravida 1, para 0) at 27 weeks of gestation presented with the acute onset of exacerbation of respiratory distress. Prior to the onset of respiratory symptoms, she received antibiotics for a 9-day fever (>38°C), nasal discharge, and coughing. On presentation, body temperature was 36.8°C, blood pressure 108/69 mmHg, pulse rate 138 beats/min, respiratory rate 35 breaths/min, and O₂ saturation of 94% in room air. The ‘wet-warm’ clinical

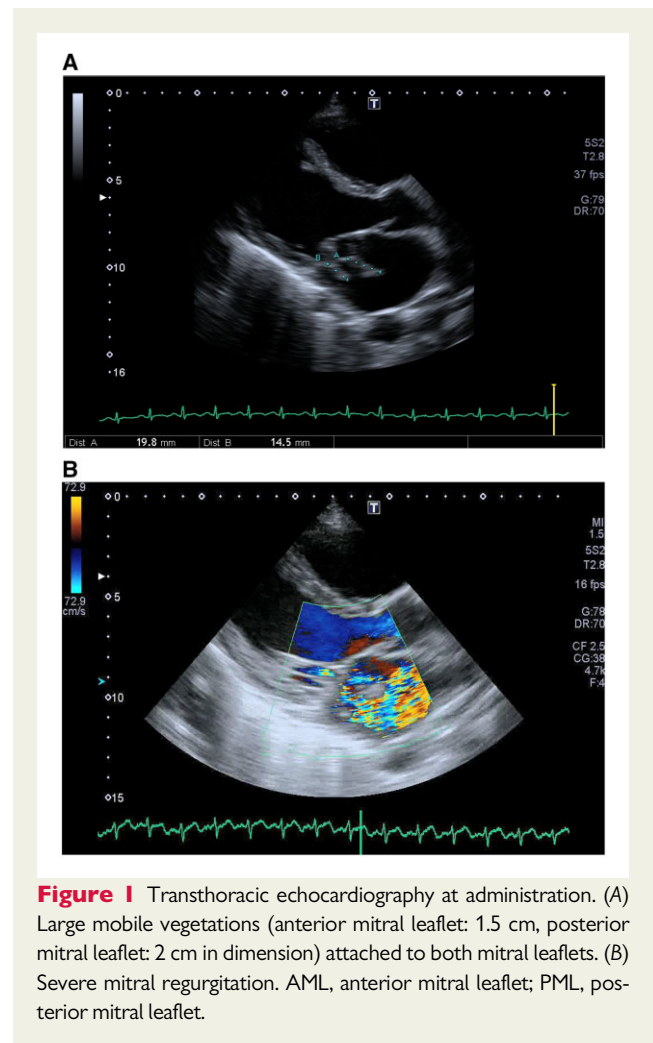


Figure 1 Transthoracic echocardiography at administration. (A) Large mobile vegetations (anterior mitral leaflet: 1.5 cm, posterior mitral leaflet: 2 cm in dimension) attached to both mitral leaflets. (B) Severe mitral regurgitation. AML, anterior mitral leaflet; PML, posterior mitral leaflet.

phenotype of heart failure (HF) was noted with a systolic murmur (3/6 grade) at the apex, bilateral coarse crackles, and bilateral leg oedema. There were no Janeway lesions or abnormal neurological findings. She had a history of hypothyroidism that was treated with levothyroxine and well controlled. She had no history of intravenous drug use or cardiovascular diseases, including congenital heart disease, rheumatic fever, and valvular heart disease. There were no signs or symptoms of malignancy, autoimmune disease, or coagulation abnormalities. She also did not have gestational hypertension or diabetes. Electrocardiography showed sinus tachycardia and a complete right branch bundle block. Massive pulmonary congestion was detected on chest X-ray. Laboratory findings included elevated brain natriuretic peptide 564.5 pg/mL (normal range: ≤18.4 pg/mL), C-reactive protein 4.86 mg/dL (normal range: ≤0.14 mg/dL), and white

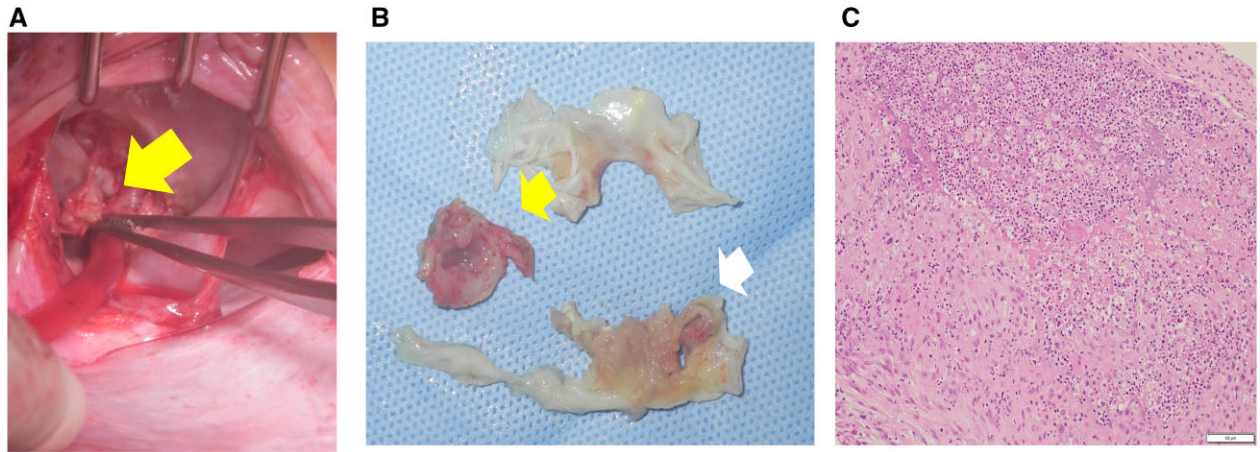


Figure 2 Removal of vegetations and mitral valve replacement—intraoperative image (A, B) and histopathological examination (C). (A) Floating vegetations (yellow arrow) attached to most of the mitral valve, but not P1. (B) Floating vegetations (yellow arrow) and anterior mitral leaflet perforation (white arrow). (C) Massive infiltration of neutrophil (haematoxylin and eosin stain).

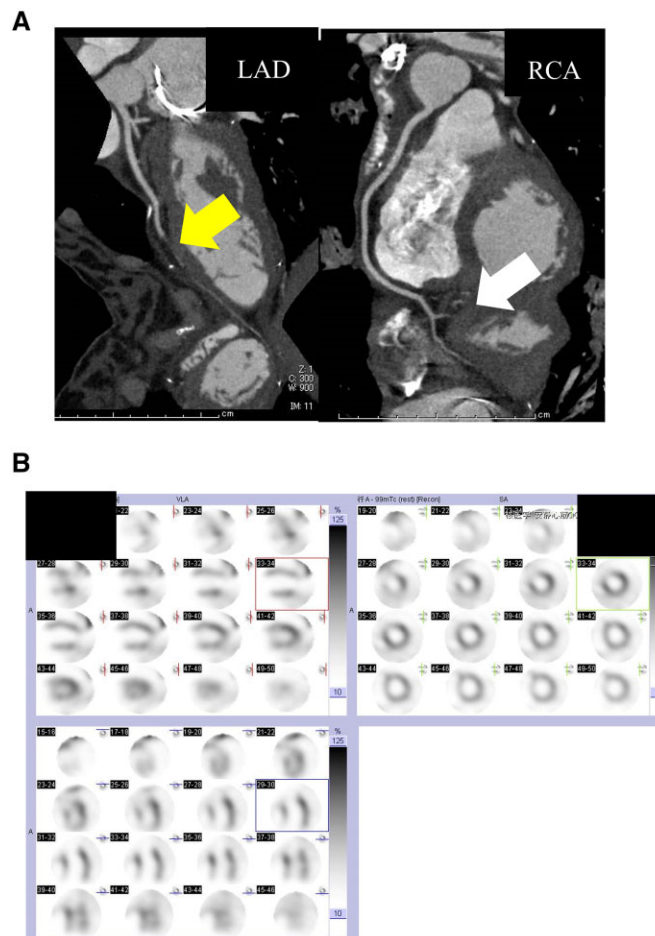


Figure 3 Coronary computed tomographic angiography and resting thallium-201 myocardial scintigraphy. (A) Occlusion of mid-left anterior descending artery (yellow arrow) and peripheral right coronary artery (white arrow). (B) Myocardial scintigraphy in resting images showing an irreversible ischaemic defect in the infarcted area.

blood cell count 13 890/ μ L (normal range: 3300–8600/ μ L). Transthoracic echocardiography showed severe mitral valve regurgitation (MR) due to prolapse of the anterior mitral leaflet, mobile vegetations of 1.5 and 2 cm in diameter on the anterior and posterior mitral leaflets, respectively (Figure 1), and severe pulmonary hypertension (estimated pulmonary artery pressure 112 mmHg). The left ventricular ejection fraction (LVEF) was 71%. Ultrasound and a non-stress test for the foetus were normal.

Based on these findings, IE was suspected.⁴ Large mobile vegetations, overt disruption of the mitral valve, and decompensated HF due to severe MR indicated the need for emergent interventions. Following a multi-disciplinary conference by cardiologists, cardiovascular surgeons, obstetricians and gynaecologists, paediatricians, and anaesthesiologists, simultaneous emergent MVR and CS were selected. After an intramuscular injection of 12 mg betamethasone to prevent infantile respiratory distress syndrome, CS was performed within 5 h of presentation, and a male infant weighing 1,154 g was delivered with APGAR scores of 1, 5, and 6 at 1, 5, and 6 min, respectively. A uterine compression suture was used to achieve complete haemostasis. Since the patient wanted to have more children in the future, a biological valve (MAGNA MITRAL EASE 29 mm) was selected. Most of the mitral valve leaflets were occupied by vegetations, apart from P1, and the anterior mitral leaflet was perforated (Figure 2A,B). After CS and MVR with a total operation time of four hours, the patient was returned to the intensive care unit (ICU) and left the ICU for the general ward the next day. A histopathological examination of the mitral valve showed acute neutrophil-dominant inflammation, which was consistent with vegetations from IE (Figure 2C). Although blood and vegetation cultures, and histopathological examinations did not identify the causative bacteria, this may have been due to the preceding use of antibiotics. Therefore, empiric intravenous antibiotic therapy with 2 g/day ceftriaxone and 9 g/day sulbactam/ampicillin was maintained over 21 days. On Day 6 after surgery, follow-up echocardiography revealed slightly reduced LVEF (41%) with abnormal anterior wall motion. Coronary computed tomographic angiography showed occlusion of the mid-left anterior descending artery and peripheral right coronary artery (Figure 3A), and head magnetic resonance imaging revealed multiple cerebral infarctions. These results indicated that septic emboli from vegetations had caused silent myocardial and cerebral infarctions. Since the patient was free of chest pain before and during hospitalization and myocardial scintigraphy in resting images showed an irreversible ischaemic defect in the infarcted area (Figure 3B), coronary interventions were not selected and medication including beta-blocker and angiotensin-converting enzyme inhibitor was initiated. Because of the lack of neurological deficits, additional treatments or further examinations for cerebral infarction were not performed. On Day 29 after surgery, the patient was discharged in a good condition. The neonate recovered uneventfully and was discharged on Day 95 of life. The patient and infant are doing well without any cardiac and neurological symptoms 1 year after surgery. One-year follow-up echocardiography showed normal mitral valve function with a slightly decreased LVEF of 43%.

Discussion

Infective endocarditis is a rare, life-threatening infection during pregnancy.¹ Recent studies reported a maternal mortality rate for IE in

pregnancy of 11%,² and in-hospital mortality rate during delivery of 17.2%.³ The timing of and indications for surgical interventions for IE in pregnancy remain controversial. Foetuses with a gestational age (GA) <26 weeks are at a markedly higher risk of mortality and severe morbidities.⁵ However, at GA of 26 weeks, the survival rate of foetuses increases from 28 to 84.8%.⁵

Three scenarios are proposed for the treatment of IE in pregnancy:

- (1) cardiac surgery while continuing pregnancy;
- (2) CS or vaginal delivery followed by elective cardiac surgery;
- (3) simultaneous CS and cardiac surgery

Scenario 1 may extend the foetal period. However, valvular surgery for IE often requires cardiopulmonary bypass (CPB), which has a high foetal mortality rate.¹ The peri- and post-operative use of vasoactive agents and warfarin may also adversely affect the foetus. Furthermore, CS following valvular surgery may be associated with the risk for subsequent prosthetic valve endocarditis. Preceding CS or vaginal delivery followed by elective cardiac surgery (Scenario 2) is another therapeutic option. Wang *et al.*⁶ reported a successful case of IE at 26 weeks GA, with CS being performed at 31 weeks followed by cardiac surgery on Day 10 after CS. However, CS or vaginal delivery may worsen HF in patients with an unstable condition and haemodynamics. Therefore, simultaneous CS and cardiac surgery (Scenario 3) may be the only option. In this scenario, there are fewer concerns about unstable haemodynamics, CPB, and medication adversely affecting the foetus. The condition of our patient was unstable with a very high risk of embolism from large vegetations. On the other hand, GA was >26 weeks. Therefore, we selected simultaneous CS and cardiac surgery. However, in this scenario, insufficient haemostasis after CS may lead to an overt bleeding event triggered by subsequent heparinization at CPB. For complete haemostasis, we used a compression suture. Infective endocarditis at GA <26 weeks, which requires emergent surgery due to critically unstable haemodynamics, appears to be the worst scenario. Botta *et al.*⁷ reported the successful treatment of a case of IE at 14 GA. Their patient underwent MVR with pregnancy, and CS was performed at 38 GA.

Conclusion

In the cases of IE in pregnancy, the timing of valvular surgery and delivery of the foetus needs to be decided by a multi-disciplinary team, considering the maternal condition and Foetal maturity.

Lead author biography



Shiori Maruichi-Kawakami studied Medicine and Surgery at the University of Kyoto, where she graduated in 2017. She spent two years at Osaka Red Cross Hospital as resident physician. She is a third-year resident in cardiology. Her clinical and academic interests are cardiac imaging modalities and heart failure.

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Consent: The authors confirm that consent for the publication of this case report, including images and the associated text, was obtained from the patient in line with COPE guidance.

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