Acute Cardiac Failure: A Complication Of Infective Endocarditis In Pregnancy

J Suen, J Chiu*, W Lee

Citation

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Abstract

We report a case of acute cardiac failure in pregnancy resulting from unsusp ected infective endocarditis of the prolapsed mitral valve. Cardiorespirator y collapse rapidly ensued but prompt cardiopulmonary resuscitation saved the mother. Emergency mitral valve replacement was carried out later.

INTRODUCTION

Although mitral valve prolapse (MVP) is the commonest valvulopathy in adults, its course during pregnancy is usually benign. However, with the development of infective endocarditis and cardiac decompensation, cardiopulmonary arrest (CPA) can result. The precise incidence of CPA in pregnancy is unknown, but it has been reported to occur about once in 30 000 pregnancies.[1,2] The rarity and unpredictability of the condition render management difficult and survival rates poor.

CASE HISTORY

A twenty-nine year old primigravida presented in her 27th week of pregnancy with a complaint of breathlessness in the previous six hours. She had been antenatally well. She had a history of mitral valve prolapse (MVP) that had been discovered during a routine physical examination in her teenage years. She was not an intravenous drug abuser and had not received any dental treatment or antibiotic prophylaxis recently.

Clinical examination revealed a tachypnoeic and diaphoretic patient. She had a respiratory rate of 35 breaths per minute and appeared cyanosed. Her pulse rate was 130 beats per minute and her blood pressure was 90/60mmHg. She would not lie down because it made her more dyspnoeic. Bilateral coarse crepitations were evident and a grade 4/6 pan-systolic murmur radiated from the mitral area to the axilla. The foetal heartbeat was detectable at this time. Chest X-ray revealed a ‘batwing’ appearance indicative of acute pulmonary oedema. Oxygen was administered via a Hudson mask and she was transferred immediately to the intensive therapy unit.

En route the patient suffered cardiorespiratory collapse and became asystolic. Cardiopulmonary resuscitation (CPR) was promptly started.

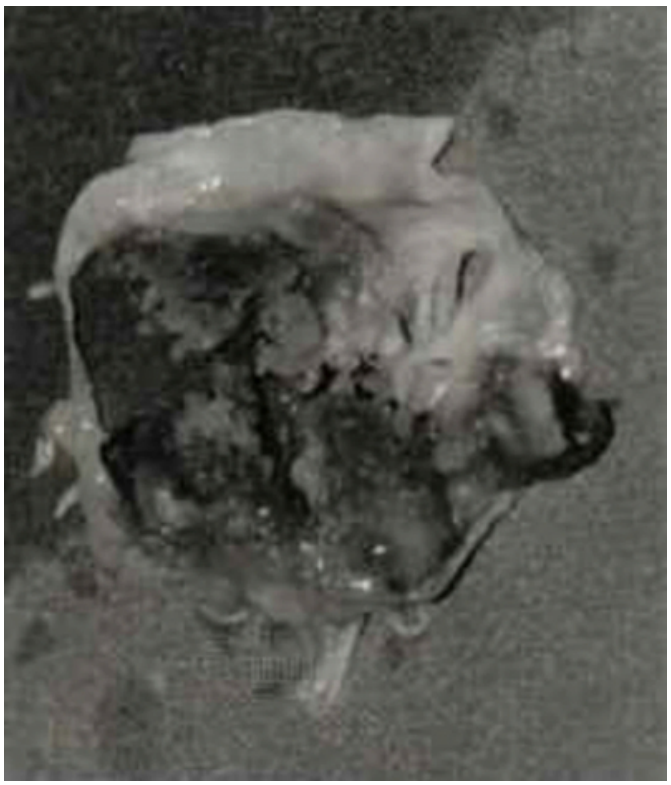
Endotracheal intubation followed by ventilation with 100% oxygen and an intravenous dose of adrenaline 1mg restored the rhythm to sinus tachycardia. There was a return of spontaneous circulation, producing a blood pressure of 150/90mmHg.

At this point the foetal heart could not be detected. Two-dimensional echocardiography of the heart revealed severe mitral valve regurgitation. The edges of the flail leaflets were noted to be thickened and irregular.

Six hours later mitral valve replacement was carried out under cardiopulmonary bypass. Florid vegetations on both leaflets of the mitral valve were present. (Fig.1) The papillary muscles and chordae tendinae were also studded with vegetations. Blood culture and tissue culture of the valves grew alpha-haemolytic Streptococci sensitive to penicillin.

Figure 1

Figure 1: The vegetation-encrusted and prolapsed mitral valve



The post-operative course was complicated by an embolic infarction of the left parietal lobe. The stillborn foetus was delivered spontaneously three weeks later.

The patient was discharged four weeks later in an ambulant state. She suffered from mild dysphasia and had no recollection of events post-collapse till a few days after her valve replacement.

DISCUSSION

This patient presented no diagnostic problem with her florid symptoms and signs of cardiac failure: dyspnoea aggravated by recumbency, bilateral coarse crepitations and a loud pan-systolic murmur in the mitral area. The chest X- ray was typical of acute pulmonary oedema.

Previous anecdotal reports have documented the importance of intravenous drug abuse, alcoholism and chronic haemodialysis in the pathogenesis of endocarditis. Other important contributing factors include valves that have been scarred by rheumatic disease or are congenitally malformed. This patient had mitral valve prolapse. It is possible that the reduced immunity associated with pregnancy was contributory to the colonisation of the valve leaflets.

MVP is the most common valvular heart lesion in adults and it is usually asymptomatic. Previous reports have detailed the obstetrical outcome of pregnancies in women diagnosed antenatally with this condition.[3,4,5] Generally, women tolerated pregnancy and delivery well. Serial echocardiographic imaging revealed that pregnancy causes either no change or an improvement in the valvular prolapse.[6] However, Strasberg reported that Group B streptoccccal endocarditis could develop even after an uncomplicated vaginal delivery7 and this raises the issue as to whether instituting antibiotic prophylaxis for this condition is cost-beneficial. Analogous to our experience, Souma et al. also reported on the success of mitral valve replacement for infective endocarditis during pregnancy.[8]

Resuscitation of a pregnant patient can be most demanding due to potential difficulties in securing the airway, ventilation of the lungs and external cardiac compression.2

The airway may be oedematous and airway anatomy may be easily traumatised by repeated laryngoscopy. Enlarged breasts may obstruct the correct placement of the laryngoscope handle; hence a short handled laryngoscope is preferable in obstetric patients. There is also the increased risk of regurgitation and aspiration associated with pregnancy. Oesophageal occlusion by pressure on the cricoid cartilage is advocated for all cases undergoing endotracheal intubation.

Ventilation of the lungs will usually need a higher airway driving pressure due to the decreased compliance of the chest wall from upward migration of the gravid uterus. Furthermore the minute volume in pregnancy usually places the normal PaCO2 at 30mmHg. Normal ventilation of the obstetric patient is therefore mild hyperventilation.

Aorto-caval compression from the gravid uterus causes significant aortic outflow obstruction with ensuing decreases in renal and uterine perfusion. Compression of the inferior vena cava and major pelvic veins could lead to sequestration of up to 30% of the circulating blood volume as pregnancy progresses. As a consequence, as many as 10% of patients in late pregnancy develop hypotension, bradycardia, and impaired cardiac output in the recumbent position (supine hypotension syndrome). Aortocaval compression is obviated by uterine displacement to the left by means of a pillow, slanted board or bimanual displacement of the uterus.2 Had the patient not responded to basic life support, some authorities have advocated immediate perimortem caesarean section to deliver the foetus within five minutes of maternal

collapse.[9,10,11] This improves foetal outcome where no possibility of survival would exist in a non-perfused uterus. Additionally the removal of the low-resistance placental bed and aorto-caval compression would assist in achieving higher perfusion pressures to the mother's vital organs.

CONCLUSION

We report this interesting case of undiagnosed infective endocarditis presenting as acute cardiac failure during pregnancy in a setting of mitral valve prolapse. The favourable outcome of maternal survival was achieved through expeditious institution of CPR, diagnosis of the precipitating cause of acute left ventricular failure, and emergent mitral valve replacement. Unfortunately the foetal wastage even with current advances in resuscitation remains an ominous challenge.

References

1. Hibbard BM, Anderson MM, Drife JO et al. Report on confidential enquiries into maternal deaths in the United Kingdom 1991-93. London: HMSO, 1996.
2. Rees GAD, Willis BA. Resuscitation in late pregnancy. Anaesthesia 1988; 43:347-9.
3. Chia YT, Yeoh SC, Lim M et al. Pregnancy outcome and mitral valve prolapse. Asia Oceanic J Obst Gynaecol 1994, 20:383-8.
4. Jana N, vasishta K, Khunnu B et al. Pregnancy in association with mitral valve prolapse. Asia Oceanic J Obst Gynaecol 1993; 19:61-5.
5. Rayburn WF, Fontana ME. Mitral valve prolapse and pregnancy. Am J Obstet Gynecol 1981; 141:9-11.
6. Rayburn WF, LeMire MS, Bird JL, Buda AJ. Mitral valve prolapse. Echocardiographic changes during pregnancy. J Reprod Med 1987; 32:185-7.
7. Strasberg GD. Postpartum group B streptococcal endocarditis associated with mitral valve prolapse. Obstet Gynecol 1987; 70:485-7.
8. Souma T, Yokosawa T, Iwamatsu T, Irisawa T. Sucessful mitral valve replacement for infective endocarditis in pregnancy. Nippon Kyobu Geka Gakkai Zasshi 1990; 38:1035-8.
9. Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. Obstet Gynecol 1986; 68:571
10. Strong TH, Lowe RA. Perimortem cesarean section. Am J emerg Med 1989; 7:489-94.
11. American Heart Association Subcommittee on Emergency Cardiac Care: Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care. JAMA 1992; 268:2172-249.

Author Information

Jeffrey C.H> Suen, MBBS, FRCA, FFARCSI, FANZCA
Registrar, Department of Anaesthesia, Changi General Hospital

Jen W. Chiu*, MBBS, MMed (Anaes), DEAA
Associate Consultant, Department of Anaesthesia, KK Women's and Children's Hospital

Wei-Hong Lee
Associate Consultant, Department of Reproductive Medicine, KK Women's & Children's Hospital