CASE REPORTS

Peripartum cardiomyopathy presenting as a cardiac arrest at induction of anaesthesia for emergency Caesarean section

A. K. McIndoe, E. J. Hammond and P. C. B. Babington

Summary
Peripartum cardiomyopathy is defined as the onset of acute heart failure without demonstrable cause in the last trimester of pregnancy or within the first 6 months after delivery. It occurs in about 1 in 4000 deliveries and is often unrecognized as symptoms of normal pregnancy commonly mimic those of mild heart failure. We describe a previously asymptomatic patient who presented with a cardiac arrest at induction of general anaesthesia for emergency Caesarean section and subsequently developed acute heart failure. This case is unique both in its mode of presentation and the total absence of antecedent symptoms or signs of cardiac disease. (Br. J. Anaesth. 1995; 75: 97–101)

Key words
Anaesthesia, obstetric. Complications, cardiomyopathy.

Case report
A healthy, 32-yr-old, 70-kg woman was admitted at term in early labour during her third pregnancy. Cardiotocograph (CTG) monitoring was normal and no problems were anticipated. Four hours after admission to the labour ward (04 : 45), vaginal examination revealed a 3–4 cm semi-effaced cervix and suxamethonium 100 mg, with cricoid pressure applied by a trained anaesthetic assistant. Tracheal intubation was achieved easily using an 8.0-mm cuffed oral tube which was clearly seen to pass between the cords. The patient’s lungs were initially ventilated manually using the circle system with a fresh gas flow of nitrous oxide 4 litre min⁻¹, oxygen 2 litre min⁻¹ and 2 % isoflurane. Correct placement of the tracheal tube was confirmed and the capnometer (Ohmeda Modulus CD) displayed a normal end-tidal carbon dioxide trace. After securing the tube, heart rate was noted to have decreased to 40 beat min⁻¹ (sinus bradycardia). Atropine 600 µg was administered i.v. and the rate of i.v. infusion was increased. Oxygen saturation remained at 100 % and although initially the carotid pulse was easily palpable, bradycardia failed to respond to atropine and within less than 30 s the patient became asystolic with no palpable pulse. External cardiac massage was commenced immediately and left lateral tilt was maintained. Manual ventilation was continued with 100 % oxygen. Adrenaline 1 mg and a further 2 mg of atropine were administered i.v.

The consultant obstetrician proceeded to deliver the baby as quickly as possible by classic Caesarean section while active resuscitation was continued. Delivery of a live, pink, crying, 3.92-kg boy was achieved within 2 min during which time the mother remained asystolic, despite cardiopulmonary resuscitation. Although cord blood-gas tensions were not measured, the baby attained Apgar scores of 9 at 1 and 5 min. After approximately 3 min of maternal cardiac massage there was evidence of spontaneous cardiac electrical activity and return of carotid and femoral pulses associated with an unstable sinus tachycardia of rate 120–150 beat min⁻¹ punctuated by multifocal narrow complex supraventricular and ventricular activity. Further external cardiac mass-
age was withheld but was required on three subsequent occasions during episodes of intermittent pulseless tachyarhythmias. These episodes were short-lived and self-terminating. Arterial pressure and cardiac rhythm remained labile (80–130 mm Hg systolic) despite pulse oximetry readings of 100% saturation, so surgical anaesthesia was maintained with morphine 20 mg i.v. and midazolam 10 mg i.v. The rhythm disturbances settled during the following 5 min leaving the patient with a sinus tachycardia with arterial pressure of 132/84 mm Hg. However, arterial pressure drifted to 70/45 mm Hg over the next 5 min. Administration of two 100-µg boluses of adrenaline restored it to 150/90 mm Hg and allowed the introduction of low inspired concentrations of isoflurane. However, adrenaline precipitated short-lived self-terminating supraventricular and ventricular arrhythmias similar to those in the immediate post-asystolic period. The patient remained relatively stable for the remaining 30 min of the operation allowing administration of Syntocinon 10 u. and neuromuscular block with atracurium 25 mg.

Despite the infusion of a total of 2000 ml of crystalloid and 500 ml of colloid, arterial pressure remained pressor dependent. Total peroperative blood loss was measured as 450 ml. Initially anaphylaxis was suspected and the patient received hydrocortisone 200 mg and chlorpheniramine 20 mg i.v. A right subclavian triple lumen central catheter and right radial arterial cannula were inserted. The initial central venous pressure (CVP) was 12 mm Hg but the patient remained hypotensive despite adequate filling pressures. She was given an adrenaline infusion before transfer to the intensive care unit with full invasive monitoring. The patient also received ampicillin 500 mg and metronidazole 500 mg before the end of surgery as routine antimicrobial prophylaxis.

Elective intermittent positive pressure ventilation was continued in the intensive care unit and the patient was sedated with morphine and midazolam. Her haemodynamic variables stabilized over the next 2 h and she reverted to normal sinus rhythm with an arterial pressure of 120/70 mm Hg and CVP of 10 mm Hg. Her 12-lead EGG was unremarkable except for slight left axis deviation. There was no evidence of ischaemia, right ventricular strain or underlying conduction disturbance. Chest x-ray was unremarkable except for slight rounding of the cardiac shadow on a portable supine AP radiograph (fig. 1).

Clinical examination revealed a prominent left apical impulse with no right ventricular heave. There were no cardiac murmurs or added cardiac sounds and no evidence clinically of left ventricular failure. The patient remained stable during the following 6 h after cardiac arrest. Adrenaline was reduced and she was weaned from the ventilator as planned following discussions with the cardiology unit in Oxford. The trachea was extubated uneventfully and she was fully awake by 15 : 15, 10 h after the initial cardiac arrest. Over the course of the next 90 min she became increasingly drowsy with \( \text{S}_\text{pO}_2 \) decreasing to 85%. Naloxone had no effect. She developed a sinus tachycardia of 140 beat min\(^{-1}\) and arterial blood-gas analysis revealed a \( P_{\text{aO}_2} \) of 7.8 kPa with an \( P_{\text{aCO}_2} \) of 0.4 in the presence of a normal pH. \( \text{S}_\text{pO}_2 \) improved with an \( P_{\text{aCO}_2} \) of 0.6. Clinical examination now showed a regular tachycardia with prominent left apical impulse and gallop rhythm. There were crepitations in both lung bases. Arterial pressure decreased to 100/65 mm Hg with a CVP of 12 mm Hg.

ECG was unchanged but a repeat chest x-ray showed evidence of pulmonary oedema with a globular cardiac silhouette (fig. 2). An underlying cardiomyopathy was felt to be the most likely diagnosis despite the absence of antenatal symptoms or signs. Echocardiography facilities were not available locally. In view of her rapid deterioration she was transferred to the coronary care unit in Oxford for further investigation. On arrival in Oxford she developed a tachyarrhythmia unresponsive to adenosine. Amiodarone was then commenced as...
there was evidence of independent atrial activity on further analysis of the ECG. Echocardiogram revealed a dilated and poorly contractile left ventricle with normal right heart and valve function. Over the next few hours her worsening gas exchange and increasing pulmonary oedema required CPAP therapy with 80% oxygen. She reverted spontaneously to sinus rhythm 8 h after admission to Oxford. Her condition stabilized over the next few days but the echocardiogram remained unchanged with global hypokinesia. She was anticoagulated and heart failure was treated with frusemide and captopril. A MUGA scan later confirmed an ejection fraction of 0.57 with evidence of left ventricular dilatation and global reduction in function. An exercise tolerance test was performed before discharge from Oxford and no arrhythmias were noted. She managed to attain stage 2 of the modified Bruce protocol for 8 min before the test was terminated because of exhaustion. She was discharged home 10 days after the cardiac arrest.

Investigations performed in Swindon and Oxford showed no evidence of underlying cardiac ischaemia. Viral studies, autoantibodies, thyroid function and urine metanephrines were all within the normal range. There were no increases in inflammatory mediators or histamine after the arrest suggesting that anaphylactic reaction to induction of anaesthesia was unlikely. Peripartum cardiomyopathy presenting atypically at Caesarean section was felt to be the most likely diagnosis in this patient.

**Discussion**

Peripartum cardiomyopathy is a relatively rare form of acute heart failure associated with pregnancy [1]. It was recognized first in the 19th century by Ritchie [2] and is defined as the onset of acute heart failure in the last trimester or early postpartum period in the absence of infectious, metabolic, toxic, ischaemic or valvular causes of myocardial dysfunction [3].

The reported incidence in the USA and Europe ranges from 1 in 1300 to 4000 deliveries, representing less than 1% of cardiovascular problems associated with pregnancy [4, 5]. Sixty percent present within the first 2 months postpartum but up to 7% may present in the last trimester of pregnancy [6, 7]. Geographical variations exist with a higher incidence reported in areas of Africa because of malnutrition and local customs in the puerperium [8, 9]. Pre-disposing factors include maternal age greater than 30 yr, multiparous or eclamptic patients, twinning, racial origin (black), hypertension and nutritional deficiencies [10]. In the majority of cases there is no family history. The only pre-disposing factors in this patient were age greater than 30 yr and two previous pregnancies.

Peripartum cardiomyopathy usually presents with symptoms of worsening cardiac failure. These include dyspnoea on exertion, fatigue, ankle oedema, embolic phenomena, atypical chest pains and hae-moptysis. Examination may reveal evidence of a raised CVP, tachycardia, cardiomegaly with a gallop rhythm (S₃), mitral regurgitation, pulmonary crackles and peripheral oedema. Chest radiographs may show cardiomegaly with pulmonary oedema and pulmonary venous congestion. The echocardiogram may show non-specific ST and T wave changes, atrial or ventricular arrhythmias and conduction defects. Echocardiographic changes include dilatation of the left atrium and ventricle with global hypokinesia [1, 7, 11].

The treatment of this condition follows that for other forms of congestive cardiac failure [1]. This includes bed rest, diuretics, vasodilators, ACE inhibitors and anticoagulation to counter the risk of endocardial clot formation. Digoxin is often added to the above regimen. The role of immunosuppressive therapy remains controversial. Cardiac transplantation may be an option for those patients with intractable heart failure unresponsive to medical therapy.

The clinical course and outcome of this disease appears to be variable. Prognosis depends on the degree of cardiomegaly at presentation and in the following 6 months [10]. There appears to be an initial high risk period with a mortality of 25-50% in the first 3 months post-partum [7]. Patients with persistent cardiomegaly at 6 months have a reported mortality of 85% at 5 years [5].

This case is unusual in that it presented at the time of emergency Caesarean section as a cardiac arrest. We have been unable to find any similar presentation of this condition reported in the literature. The preoperative anaesthetic and obstetric assessment revealed no evidence of cardiac dysfunction. The patient had been completely asymptomatic in the antenatal period. This was subsequently reconfirmed when the patient was interviewed at a later date. The initial presentation as a sinus bradycardia rapidly progressing to an asystolic cardiac arrest is particularly unusual as most cases present as symptomatic heart failure in the postoperative period. We have found only one case of peripartum cardiomyopathy presenting during Caesarean section as sinus bradycardia and hypotension. This rapidly progressed to acute cardiac failure [11].

The sequence of events that occurred in our patient illustrated the worst case scenario of a cardiac arrest of unknown aetiology occurring at induction of anaesthesia for emergency Caesarean section in the early hours of the morning. The initial treatment options were limited to resuscitation of the mother from asystole while expediting the delivery of a fetus already in distress. Effective cardiac massage and resuscitation were likely to be severely compromised until this could be achieved. The practical problems associated with cardiopulmonary resuscitation in a pregnant woman at term cannot be overstated. This subject has been reviewed extensively in both the USA and Europe, and guidelines for management published [12, 13]. There is general agreement that early delivery offers the best chance of a successful outcome for both mother and baby. Our patient underwent a fully monitored induction which allowed early recognition of a cardiac arrest with prompt institution of advanced cardiac life support according to UK Resuscitation Council guidelines [14]. Fortunately the patient’s lungs had also been fully preoxygenated.
The initial differential diagnosis was limited to anaphylaxis/drug reaction, primary cardiac arrhythmia or massive pulmonary embolus. However, irrespective of the diagnostic dilemma the initial management was the prompt institution of external cardiopulmonary massage and adrenaline i.v. The use of sodium bicarbonate i.v. should be considered if arterial pH is less than 7.3 after correction of hypoxaemia and possible underlying hypercapnia as acidosis increases the α adrenergic reactivity of the uteroplacental vasculature [13]. In this patient bicarbonate was not used as end-tidal carbon dioxide was maintained in the range 4.2–5 kPa and arterial blood-gas analysis revealed no base deficit. The timing of events suggested that this was an anaphylactic reaction to the induction agents and she was treated according to Wessex regional protocols [15] with adrenaline, hydrocortisone, chlorpheniramine and appropriate samples taken for subsequent analysis. However, the absence of chest signs was noted.

An idiosyncratic drug reaction was also considered. The use of i.v. ranitidine has been associated with bradycardia and cardiac arrest if given as a rapid i.v. bolus injection. The incidence has been reported at 3.6 cases per 10 million prescriptions. It is recommended that in the presence of known risk factors such as advanced age, renal failure or conduction abnormalities, i.v. doses should be administered slowly over at least 5 min [16]. Oxytocin has been linked with maternal death from severe hypertension and cardiac arrhythmias [17]. Midazolam may cause cardiorespiratory depression leading to cardiac arrest and there has been much concern about the use of the drug for conscious sedation [17]. However, it should be noted that both drugs were given after initial resuscitation from the asystolic cardiac arrest.

The presence of a tension pneumothorax could have explained a sudden loss in cardiac output but there were no clinical signs to suggest this.

On reflection, after the patient was stabilized in the ITU, further consideration was given to the acute presentation and the possibility of underlying myocardial disorder. However, there were no clinical or radiographic signs of cardiac failure. In the light of her apparent recovery, a pulmonary artery flotation catheter was not felt to be indicated before tracheal extubation, but in retrospect it may have provided information on her underlying myocardial dysfunction. The decision to extubate the trachea was made after consultation with cardiologists and in the absence of an echocardiogram. The second striking feature of this case was the rapid onset of cardiac failure after extubation. It was at this point that the diagnosis of peripartum cardiomyopathy presenting as a primary arrhythmia became the most likely explanation for this extraordinary series of events.

With hindsight, one may suggest that tracheal extubation should perhaps not have been attempted at such an early juncture. However, in the absence of clinical signs of cardiorespiratory failure extubation seemed possible. The consequences of this decision unmasked the primary diagnosis. However, we were then faced with the dilemma of what to do should the patient require ventilatory support for worsening cardiorespiratory failure.

Some valuable lessons may be learned from this case. Women in the latter stages of pregnancy frequently complain of symptoms indistinguishable from mild cardiac failure. Occasionally this may herald the onset of cardiomyopathy. However, our patient had a total absence of subjective and objective signs of underlying cardiac problems. It is difficult to see how we could have predicted such a catastrophic reaction. Previous reports of peripartum cardiomyopathy presenting at Caesarean section under regional anaesthesia have used standard induction doses of thiopentone and suxamethonium to facilitate tracheal intubation for IPPV and control of acute pulmonary oedema [11]. The use of monitoring before induction of anaesthesia was undoubtedly a significant factor in the success of the resuscitation. The presence of resuscitation drugs on the anaesthetic machine was also a great asset, as was familiarity with UK Resuscitation Council guidelines on the management of an asystolic cardiac arrest.

Although the sequence of events initially suggested anaphylaxis, it was the absence of bronchospasm, hypovolaemia and tachycardia in combination with minor ECG and radiographic abnormalities that alerted our suspicions to an alternative diagnosis.

Fortunately, we are pleased to report that 4 months after these events, mother and child are well. Although the mother is still receiving medical treatment for mild heart failure it is hoped that she will make a full recovery over the next 6 months. The matter of future pregnancies has been discussed with the patient. The outcome seems to be linked to heart size and continuing cardiomegaly. Published data indicate a 7–60 % risk of death with subsequent pregnancies, although the numbers of patients in these studies is small [5]. Therefore, at present she has been strongly advised against further pregnancy until she has been reviewed at 12 months and further echocardiographic assessment of her heart size performed.

References


