Clinical Reports

Pulmonary oedema in two parturients with hypertrophic obstructive cardiomyopathy (HOCM)

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Two patients with hypertrophic obstructive cardiomyopathy (HOCM) presented for delivery. The first had a repeat Caesarean section with general anaesthesia and the second gave birth vaginally with epidural analgesia. Both patients developed pulmonary oedema in the peripartum period. These cases highlight the delicate fluid requirements of the pregnant patient with HOCM. The fluid management of the parturient is discussed with particular emphasis on the pathophysiology of HOCM. The indications for invasive monitoring are presented.

Deux patientes atteintes de cardiomyopathie hypertrophique obstructive (HOCM) se sont présentées pour accouchement. La première se présentait pour une seconde césarienne sous anesthésie générale alors que la seconde accoucha par voie naturelle sous épidurale. Les deux patientes ont développé de l'oedème pulmonaire lors de l'accouchement. Ces cas démontrent la fragilité de l'équilibre hydrique chez les patientes enceintes atteintes de HOCM. La conduite à faire dans des cas pareils est discutée en mettant l'emphase sur la pathophysiologie de l'HOCM. Les indications pour une surveillance par des techniques invasives sont présentées.

Key words
ANAESTHESIA: obstetric;
COMPLICATIONS: pulmonary oedema;
HEART: cardiomyopathy, hypertrophic obstructive.

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Hypertrophic obstructive cardiomyopathy (HOCM) is a relatively recently described abnormality which appears to be increasing in frequency.[1] It is observed in all races, at all ages, in both sexes and can be lethal.[2–4]

Hypertrophic obstructive cardiomyopathy presents a major challenge to the anaesthetist managing parturients. Women in labour are stressed by the pain of labour and its associated increase in catecholamine secretion.[5] Cardiac output normally increases as a result of the catecholamine-induced increase in heart rate and stroke volume.[6] However, in patients with HOCM, elevated circulating catecholamines can lead to decreased forward flow. Increased contractility leads to greater obstruction of the left ventricular outflow tract and tachycardia limits diastolic filling which reduces left ventricular end–diastolic volume.[7] The Valsalva manoeuvre, used to help the fetus descend in the second stage of labour, can also impair cardiac output in these patients through reducing preload.[2]

Many authors have stated that regional anaesthesia (spinal or epidural) is relatively contraindicated in the patient with HOCM.[9–11] General anaesthesia with halothane to reduce myocardial contractility and heart rate is the recommended form of anaesthesia.[9] This cannot be applied absolutely to the parturient in labour and lumbar epidural analgesia is often administered.[11]

Two patients with HOCM who presented for delivery are reported. Each was managed with a different anaesthetic technique yet both developed pulmonary oedema. The discussion will focus on the pathophysiological changes in HOCM and their implications for management once pulmonary oedema has occurred.

Case 1

A 27-yr-old female, G_3P_2 was admitted to the antepartum ward at 37 weeks gestation and was scheduled for elective Caesarean section the following morning. She had had

two previous Caesarean sections; the indication for the first was a breech presentation in a primigravida.

She was first diagnosed as having HOCM seven years before presentation when a heart murmur was noted on routine examination. The diagnosis was confirmed by cardiac catheterization during her first pregnancy five years earlier. During the catheterization propranolol was shown to increase her cardiac output and decrease her pulmonary capillary wedge pressure.

The patient's original and repeat Caesarean sections were performed under general anaesthesia with intra-arterial and central venous pressure monitoring. Both procedures were uneventful. Her only medication during the current pregnancy was iron supplementation. Review of systems revealed minimal swelling of her fingers and feet, and weight gain of 10 kg. She did not complain of palpitations, syncope or chest discomfort. She did fatigue a little more easily by the end of the third trimester. The rest of review of systems was normal. On examination, she weighed 58 kg, her blood pressure was 130/50, and her heart rate was $74 \cdot min^{-1}$ and regular. She had a grade III/VI systolic murmur, maximal at the fifth intercostal space radiating to the axilla and base. Breath sounds were normal. The patient's upper respiratory tract was normal. Her haemoglobin concentration was $130\ g \cdot L^{-1}$ and the rest of routine preoperative laboratory assessment was normal. Her ECG revealed left ventricular hypertrophy with nonspecific ST-T wave changes. A chest *x*-ray showed cardiomegaly and clear lung fields. An echocardiogram performed six months earlier revealed an outflow gradient of 68 mmHg with a massively enlarged intraventricular septum and diffuse muscular hypertrophy of the left ventricle.

The plan was to perform a repeat elective Caesarean section under general anaesthesia. Prior to induction of anaesthesia, an 18-ga IV catheter, a 20-ga left radial artery catheter and a 22-ga brachial CVP catheter were inserted with local infiltration. The CVP did not fluctuate with respiration and was thought to be improperly positioned. This was confirmed on postoperative chest *x*-ray, when it was seen coiled outside the thoracic inlet. Once the intravascular catheters were secure and other routine monitoring devices were placed, general anaesthesia was induced via a rapid sequence technique with fentanyl 100 μg, lidocaine 100 mg and thiopentone 275 mg IV. Succinylcholine 160 mg was administered to facilitate tracheal intubation. Cricoid pressure was applied upon injection of the thiopentone. The trachea was intubated easily with a #7.0 tracheal tube. After intubation general anaesthesia was maintained with N_2O/O_2 (50:50) and intermittent halothane up to 0.75 per cent (inspired). Vecuronium 3 mg was administered for muscle relaxation after the effects of the succinylcholine had dissipated.

The baby was delivered and had an APGAR score of two (for heart rate only) at one minute. The baby's trachea was intubated and the lungs ventilated for approximately two minutes. (Naloxone 0.035 mg was administered IM.) The five-minute APGAR score was 9 (one off for colour). The uterine incision to delivery time was $3\frac{1}{2}$ minutes and probably contributed to the depression of the neonate at one minute. The newborn infant's subsequent hospital course was uneventful.

During the Caesarean section the patient was hemodynamically stable. She received approximately 3000 ml of 0.9 per cent saline IV and 750 ml of blood was lost. Her urine output was 600 ml over the $2\frac{1}{2}$ hr surgical period. An intraoperative blood gas analysis revealed pH 7.35, PCO_2 33, PO_2 100, and HCO_3^- 18 ($FIO_2 = 0.5$). Neuromuscular blockade was reversed with edrophonium 50 mg and atropine 0.6 mg. The trachea was extubated when the patient was awake. Over the next ten minutes the patient became tachycardic (pulse of $106 \cdot min^{-1}$) and tachypnoeic (respiratory rate of $29 \cdot min^{-1}$). Chest auscultation revealed bibasilar crackles. Arterial blood gas analysis showed pH 7.42, PCO_2 33, PO_2 54, and HCO_3^- 21 ($FIO_2 = 0.21$). Furosemide 5 mg IV resulted in a diuresis of 1600 ml over the next two hours and the patient's condition improved. Repeat arterial blood analysis at this time showed pH 7.43, PCO_2 30, PO_2 130 and HCO_3^- 20 (FIO_2 5 $L \cdot min^{-1}$ by nasal prongs).

The rest of her hospital stay was unremarkable. She was discharged six days later with her baby.

Case 2

A 29-yr-old female G_1P_0 was first seen on the obstetrical ward at 39 weeks in labour. She was diagnosed as having HOCM at age 13. An echocardiogram early in her pregnancy revealed systolic anterior motion of the mitral valvular apparatus in a small left ventricular cavity and an enlarged left atrium. During her pregnancy she began to have dyspnoea in bed and occasional palpitations mostly in the morning. She denied having orthopnoea, paroxysmal nocturnal dyspnoea, or syncope. Her cardiologist classified her as NYHA class III. Therapy with β-blockers was advised but the patient declined because of the possible fetal effects. The patient smoked one-half pack of cigarettes per day. Review of systems was otherwise normal.

On admission to the labour floor, the patient was flushed but in no acute distress. Her blood pressure was 110/70 mmHg and her heart rate was $90 \cdot min^{-1}$ and regular. She had a grade IV/VI harsh blowing holosystolic murmur, which was loudest at the apex and radiated to the axilla. Auscultation of the lungs revealed good air entry

with a few crepitations in the left base. Her upper airway was normal. There was no ankle oedema.

An infusion of lactated Ringer's solution was started at 150 ml·hr^{-1} via an indwelling 18-ga IV catheter at 1530 hr. In early labour, 1745 hr, she received IM meperidine 75 mg for analgesia. Her heart rate began increasing up to 120·min^{-1} with contractions. At 2030 hr an 18-ga epidural catheter was inserted using the loss or resistance technique through the L_{3-4} interspace, following a bolus of 800 ml Ringer's lactate.

A test dose of 2 ml lidocaine two per cent was followed by a maintenance dose of 9 ml bupivacaine 0.25 per cent. The onset of epidural anaesthesia was not associated with any adverse haemodynamic changes. At 2130 hr the patient complained of shortness of breath. Her respiratory rate was 30·min^{-1}, heart rate 130·min^{-1} and blood pressure varied between 100/60 mmHg and 130/70 mmHg. Arterial blood gas analysis revealed a pH 7.42, PCO_2 30, PO_2 57, HCO_3^{-1} 20 (FiO_2 = 0.21). Oxygen by non-rebreathing facemask (FiO_2 1.0) was started. Intermittent boluses of propranolol to a total dose of 5 mg were administered IV over two hours in an effort to slow the tachycardia and improve forward flow. At 0036 hr a male baby was delivered vaginally with outlet forcepts assistance. The one minute APGAR score was five and the five minute APGAR score was seven. Because of the administration of parental propranolol, neonatal hypoglycaemia was considered. However, the baby's hospital stay was unremarkable.

However, the parturient was in pulmonary oedema, and probably had been since 2130 hr. Because of her HOCM there was concern regarding administration of diuretics and 125 ml·hr^{-1} of lactated Ringer's solution was administered IV. A further 2 mg propranolol was injected IV at 0300 hr to decrease obstruction and slow the tachycardia (heart rate 100·min^{-1}). No diuretics were given. Five hours later, the patient was still in pulmonary oedema and was being treated on the labour floor. The IV fluid administration was decreased, propranolol withheld and furosemide 40 mg was administered IV. The patient diuresed 5,400 ml over the next eight hours. This diuresis cleared her pulmonary congestion and her condition improved. Follow-up arterial blood gas analysis showed pH 7.48, PCO_2 29, PO_2 77, HCO_3^- 21 (FiO_2 = 0.21).

Three days later the patient was discharged home with her baby.

Discussion

The characteristic pathological features of HOCM include pronounced asymmetric myocardial hypertrophy usually involving the interventricular septum.[2-4] The hypertrophy of the cardiac muscle is seen, histologically, to be composed of a bizarre arrangement of the muscle bundles.[4] This abnormal ultrastructure causes rhythm disturbances as well as aberrant ventricular function in diastole and systole.[12] Between 8 (on standard 12 lead ECG)[13] and 46 per cent (on 48 hr Holter monitoring)[14] of patients exhibit atrial fibrillation and/or supraventricular tachycardia.

Diastolic relaxation is abnormal with a marked reduction in left ventricular compliance.[12] The rapid filling phase is significantly decreased and isovolumetric relaxation time is prolonged. These changes produce an altered pressure–volume curve for the ventricle, resulting in high diastolic filling pressures at smaller end–diastolic volumes.[7] Systolic function is also affected with excessive left ventricular contractility.[2,12] Nearly 80 per cent of the stroke volume is ejected in the first half of systole[2,3] compared with the normal 65 per cent, and a pressure gradient arises within the body of the left ventricle, which is separated from a subaortic chamber by the thickened septum and the anterior leaflet of the mitral valve.[2] Therefore, increased contractility, decreased preload and decreased afterload worsen the clinical situation.[3]

An important physiological change in pregnancy for the patient with HOCM is a decrease in systemic vascular resistance.[6,15] This reduction in afterload begins as early as the 12th week of gestation[6] and remains reduced throughout pregnancy.[15] One would postulate that this would have an adverse effect on the patient with HOCM, but it may be counterbalanced by the increase in intravascular volume which is occurring at the same time. However, the patient may have major blood loss peripartum which would produce a decrease in preload[6] and increase the outflow obstruction.[2]

Many authors have examined the use of β-blockers in pregnancy.[16,20] In HOCM, they are used to slow tachycardia and depress contractility in the mother, despite the possibility of contributing to transient neonatal bradycardia and hypoglycaemia. Acute administration of β-blockers has received less attention.[16] It has been recognized, however, that the use of β-blocking medication can be beneficial during labour and delivery in the patient with HOCM because of the increased plasma catecholamine concentrations at this time.[13]

Should epidural analgesia be necessary but it results in hypotension despite adequate prehydration, IV boluses of phenylephrine (40–80 μg) are recommended.[2,8] Because ephedrine increases myocardial contractility and heart rate through β agonism, it is contraindicated. The use of oxytocin has also been condemned because of its vasodilating properties.[9] Ergot derivatives, on the other hand, may lead to severe hypertension especially if used in conjunction with other vasoconstrictors.[6] If a drug is

required to augment uterine contraction after delivery, a slow oxytocin IV infusion has been used safely[8] and is the optimal management. With all the possible physiological derangements it is surprising that pregnancies in patients with HOCM are well tolerated.[11,21]

However, in both cases presented, fluid management became critical during parturition. For case #2, the patient was overhydrated during the institution of epidural block because of the concern regarding relative hypovolaemia and decreased afterload.[22] The patient had a long labour with increasing discomfort. She had been tachycardic before the insertion of the epidural catheter. The tachycardia led to decreased left ventricular filling and, combined with the excessive intravascular volume, caused pulmonary venous congestion. In case #1 the sudden infusion of intravascular volume after delivery together with the IV fluids resulted in pulmonary venous congestion.

Once pulmonary oedema was diagnosed in the patient in case #2, the administration of graduated doses of propranolol to slow the tachycardia was appropriate. However, a point is reached where the depression of myocardial contractility by β blockers is excessive even for the hyperdynamic tachycardic patient with HOCM. Excessive intravenous fluid administration is never appropriate.

Use of diuretic agents has been discouraged in patients with HOCM.[2,23] Yet, diuresis can clear pulmonary venous congestion and decrease symptoms.[12,23] One should be careful not to diurese excessively but a reduction in left ventricular end-diastolic, mean left atrial, and pulmonary pressures is indicated once pulmonary oedema is established.[12,23]

Better monitoring of central venous or pulmonary arterial pressure would have helped in the management of these two patients and may have prevented the pulmonary oedema. Pulmonary arterial pressure monitoring is well established as being more sensitive in predicting and diagnosing incipient pulmonary oedema. The data provided by pulmonary artery catheterization were invaluable in a recent report of a patient with significant mitral stenosis.[24] Had a pulmonary artery catheter been in position in our patients, the opportunity to perform serial measurements of pulmonary capillary wedge pressures (PCWP), mixed venous oxygen saturation, and cardiac output could have changed the patients' courses. Information from a pulmonary artery catheter would have altered management in case #1 by indicating a rising PCWP and in case #2 by demonstrating the failure of β blockade alone to reduce PCWP. Since both patients survived without sequelae, outcome would not have been improved by the use of pulmonary arterial monitoring. Both patients had serious consequences which occurred as a result of inadequate monitoring.

In conclusion, two patients with HOCM are presented who developed pulmonary oedema peripartum. Our current management would be to treat mildly symptomatic obstetric patients clinically. Should they begin to demonstrate signs of pulmonary venous congestion or become increasingly symptomatic pulmonary arterial catheterization would be used to monitor the haemodynamic profile and β-blocker therapy instituted to control the hyperdynamic circulation. Once pulmonary edema is established IV diuretics are necessary to restore an appropriate preload.

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