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Summary

We report a case where dissection of the aorta occurred in pregnancy; only medical management was undertaken. Delivery was by Caesarean section during extradural anaesthesia and was accomplished safely several weeks after the dissection. The aetiology, association with pregnancy, diagnosis and management of acute dissection of the aorta are discussed (Br. J. Anaesth. 1995; 79: 358–360)

Key words


Aortic dissection in pregnancy is a rare but potentially catastrophic occurrence. It has been estimated that 50% of all dissections in women less than 40 yr of age occur during pregnancy, more commonly in the later months [1, 2]. Whereas definitive treatment of a DeBakey type I or II dissection (involving the ascending aorta) is surgical, for type III dissections (involving the descending aorta) medical management can be considered [3, 4]. Poor prognosis of aortic dissection occurring in pregnancy has led some to suggest immediate surgical repair with delivery by Caesarean section if pregnancy is in the third trimester [5]. We report a case where dissection of the aorta occurred in pregnancy, medical management was undertaken, and delivery by Caesarean section under extradural anaesthesia was performed several weeks after the dissection.

Case report

A 33-yr-old previously healthy white female (gravida 6, para 0, 26 weeks pregnant), presented at an emergency room with acute abdominal pain. She was given pethidine and sent home. However, the pain kept her awake all night and the next morning she was admitted to the community hospital. She had an ectopic pregnancy 8 yr previously and her right fallopian tube and ovary, and appendix, had then been removed. Physical examination revealed an obese female (weight 120 kg, height 184 cm) with an arterial pressure of 150/82 mm Hg. Abdominal palpation confirmed a gravid uterus and there was tenderness in both upper abdominal quadrants. Ultrasound examination of the abdomen revealed the presence of aortic dissection extending into the left iliac artery. She was then transferred to the University Hospital, 36 h after her initial emergency room visit.

On admission arterial pressure was 140/70 mm Hg, heart rate 80 beat min⁻¹, haemoglobin 12.9 g dl⁻¹, platelet count 254 x 10⁹/ml and her ECG was normal. An angiogram showed that the dissection started distal to the left subclavian artery (DeBakey type III). The false lumen was patent and re-entered the true lumen at the level of the mid-abdominal aorta. All major branches originated from the true lumen with no occlusions. Initial control of arterial pressure was obtained with an infusion of nitroglycerin and a decision was taken to postpone surgical repair and allow pregnancy to proceed until fetal maturity was assured. Subsequently, hydralazine, nifedipine and labetalol were commenced and nitroglycerin was discontinued. The objective was to gain control of arterial pressure and reduce myocardial contractility with agents that have been used in pregnant women without deleterious effects on the fetus (systolic pressure between 110 and 120 mm Hg, heart rate between 50 and 70 beat min⁻¹). At this time it was noted that the patient exhibited features suggestive of Marfan syndrome but a firm diagnosis could not be made. Regular ultrasound examinations confirmed fetal well being and also kept track of the dissection. Amniocentesis in the 36th week of gestation confirmed fetal maturity and the patient was scheduled for Caesarean section and tubal ligation.

On arrival in the operating room arterial pressure was 120/70 mm Hg and heart rate 70 beat min⁻¹. Two 14-gauge peripheral i.v. cannulae were inserted and, with the patient on her side, a catheter was placed in the extradural space. The right internal jugular vein was cannulated to monitor central venous pressure (CVP) and the right radial artery was cannulated for intra-arterial pressure monitoring. Both were monitored continuously throughout the procedure. Additional monitoring consisted of a five-lead ECG and a pulse oximeter. Oxygen was administered via a face mask. Lignocaine 2% was introduced (4-ml increments at 5-min intervals;
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total 30 ml) into the extradural space. An i.v. fluid preload was not administered; lactated Ringer’s solution was infused at a rate sufficient to maintain CVP at its initial level (5 mm Hg). Surgery was commenced when a T6 level of sensory block was obtained.

Five minutes later a male baby was delivered (Apgar scores of 7 and 8 at 1 and 5 min, respectively, birth weight 2600 g). After delivery, arterial pressure decreased to 70/40 mm Hg with a corresponding decrease in CVP but no change in heart rate. This decrease in arterial pressure was most likely a reflection of poor compensation, because of β and sympathetic block in the presence of acute blood loss. Rapid crystalloid infusion and three 50-μg injections of phenylephrine were needed to restore both arterial pressure and CVP. At the end of the procedure, ultrasound imaging confirmed that there had been no change in the extent of the dissection. Extradural fentanyl was infused for postoperative pain relief. The patient and her baby were discharged home 6 days after operation.

Following investigation of other members of her family in the ensuing months, a diagnosis of Marfan syndrome was made. However, the patient’s son was not affected. Ultrasound examination 6 months after Caesarean section revealed further dissection of the aorta despite medical treatment. This was then repaired uneventfully under partial bypass.

Discussion

Aortic dissection is rare in young women but, when it does occur, is often associated with pregnancy [1, 2]. During pregnancy, the highest incidence is in the later months, thus leading to speculation that the haemodynamic alterations of pregnancy may play a role [2]. Investigators have found that the aortic media in rabbits [6] and humans [7] changes morphologically and biochemically during pregnancy. In particular, the reticulum becomes fragmented, the elastica attenuates with lots of corrugation and there is a decrease in the amount of acid mucopolysaccharides. These changes probably make the aorta more vulnerable to dissection in pregnancy. In support of this, similar changes were found in the aortas of pregnant women who had had aortic dissections [8]. However, other investigators have not found differences between the aortas of pregnant and non-pregnant women [9].

A diagnosis of aortic dissection is suggested by clinical findings and confirmed by specialized investigations. A high index of suspicion with regard to pain in the abdomen or back is necessary, especially in a pregnant patient. This was exemplified by our case and in another in which unrelenting back pain following an extradural for labour was the first indication of aortic dissection [10]. Our patient survived the initial dissection and, although not diagnosed initially on clinical grounds, further management emphasizes the importance of specialized investigative procedures. Aortography has been the gold standard in the diagnosis of aortic dissection [11]. However, it is invasive and involves the risk of i.v. contrast material administration. Non-invasive techniques such as magnetic resonance imaging and transoesophageal colour-flow Doppler echocardiography may displace aortography in the future [12]. This case illustrates the usefulness of abdominal ultrasound imaging. Both the initial diagnosis and follow-up were made possible because this technique is widely available, non-invasive and does not involve exposure to radiation or contrast material.

Initial treatment of an acute dissection consists of adequate haemodynamic control. Depending on the site of the initial dissection, the natural history and subsequent treatment differ. DeBakey type I and II start in the ascending aorta (proximal) and entail surgical repair, whereas medical treatment may be considered for dissections involving the descending aorta (DeBakey type III or distal dissections) [3, 4]. In both medically and surgically treated groups, the highest mortality occurs in the first few days after distal dissections, and after the first year the slope of the survival curve for both groups approximates to that of the general population [4]. Having survived the initial dissection, it was felt that medical management would be best for both mother and fetus—cardiopulmonary bypass is associated with approximately 5% maternal mortality and 30–50% fetal mortality [13]. Correct medical management consists of arterial pressure control and reduction in velocity of ventricular contractions [3]. The appropriateness of this approach has been confirmed in experimental models of aortic dissection [14].

Antihypertensive agents and β blockers should be considered. In pregnant women, possible fetal effects of drug therapy must be considered also and recommended drugs for initiating therapy are i.v. nitroprusside and propranolol [3]. Although it has been used in pregnant women, nitroprusside poses the danger of fetal cyanide toxicity [15] and propranolol may be associated with intrauterine growth retardation [16] and, in common with other β blockers, placental transfer may lead to neonatal hypoglycaemia and bradycardia [16]. i.v. nitroglycerin, although associated with loss of fetal heart beat-to-beat variability, had no adverse effects on fetal acid–base status [17]. Hydralazine and labetalol have been used extensively in pregnant women without deleterious effects on the fetus [18, 19]. Nifedipine although relatively new, has been used safely in pregnant hypertensive women with no undesirable fetal effects [20].

Marfan syndrome is an important predisposing factor for aortic dissection [3]. In pregnant women with the syndrome, pregnancy is worse if significant cardiovascular involvement (aortic dilatation > 40 mm, aortic regurgitation or haemodynamically significant mitral valve dysfunction) existed before pregnancy [21]. There is a 50% chance that the fetus is affected also, and this, in combination with the expectation that pregnancy may aggravate cardiovascular problems, has led some to recommend therapeutic termination of pregnancy [22].

There is little in the literature on the anaesthetic management of patients with unrepaired dissecting aneurysm of the aorta. From first principles, we felt that regional anaesthesia offered advantages over general anaesthesia. In particular, the haemo-
dynamic responses to laryngoscopy and intubation that a general anaesthetic would entail would be avoided. We felt that the increase in arterial pressure, heart rate and myocardial contractility that would result from a rapid sequence induction could have had disastrous consequences. A slowly titrated extradural block avoided the potential problems of a general anaesthetic and also those of an abrupt sympathetic stimulation that spinal anaesthesia may produce in a patient with limited powers of compensation caused by β blockers. In addition, the catheter was used to maintain analgesia in the postoperative period. Adequate preoperative β blockers, a slow opioid-based induction followed by maintenance with an agent such as halothane which reduces myocardial contractility, may make general anaesthesia safe in terms of aortic rupture or extension of the dissection. However, for the mother there is the real risk of aspiration during induction and halothane may cause postpartum haemorrhage. Also, respiratory depression from the anaesthetic–opioid agents may have serious consequences in terms of morbidity or mortality in a preterm infant.

The mode of delivery in patients with cardiovascular disease is controversial. Vaginal delivery results in less blood loss, quicker recovery and lowers the risk of postoperative complications. However, the timing of delivery is unpredictable and there is always the risk of an emergency Caesarean section for fetal distress. In addition, our patient’s cervix was long and closed (and therefore unfavourable for induction of labour) and she had expressed a desire for permanent sterilization. Therefore, the decision was made to perform a Caesarean section after consultation with cardiology, anaesthesia and perinatology staff.

References