Acute myocardial infarction in pregnancy: a case report and subject review

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Myocardial infarction in pregnancy is uncommon. Use of thrombolytic treatment is relatively contraindicated. Early recognition as well as a multidisciplinary approach to the management of these cases is important, as untreated there is a high maternal and fetal mortality. The case presented here highlights the importance of early transfer to a specialist centre for percutaneous coronary intervention.

A 33-year-old Caucasian pregnant woman presented to the emergency department with a 1 h history of sudden onset severe central chest pain while shopping. The pain radiated through to the back, left shoulder and arm. She also complained of dyspnoea, sweating, dizziness, nausea and vomiting.

She was gravida 7, para 6 and cardiovascular risk factors included a 15 cigarettes per day smoking habit.

On physical examination she was pale, clammy and distressed, and initially hypotensive with a blood pressure 90/50 mm Hg; however, repeat differential blood pressures were 122/102 mm Hg and 114/43 mm Hg in the left and right arm, respectively. The patient's temperature was 35.9°C with a pulse of 94 beats/min; pulse oximetry detected an oxygen saturation of 95% on 10 litres of oxygen. Chest auscultation was clear with normal heart sounds, and there was no radio-radial or radio-femoral delay. No calf tenderness or swelling was present. Examination of the abdomen was consistent with 32 weeks of pregnancy. Obstetric assessment including ultrasonography determined a viable intrauterine pregnancy with normal fetal heart sounds.

Arterial blood gases on 10 litres of oxygen were a pH of 7.4, P_{O_2} of 47.32 and P_{CO_2} of 3.07. Biochemical analysis showed: C reactive protein 10.7, white blood cell count 15.4 (raised neutrophils and lymphocytes), Hb 10.2, platelets 522, HCO_3 20.3, urea 2.7, creatinine 64, glucose 6.4, calcium 2.87, albumin 21, cholesterol 5.5 mmol/l, triglycerides 5.1 mmol/l, normal clotting screen and an initial troponin I of 0.07 ng/ml.

Initial ECG changes showed ST elevation myocardial infarction in lead I, AVL. The patient complained of continuing severe chest pain despite intravenous opiate analgesia and serial ECGs revealed evolving ST elevation in leads V2 and V3 with reciprocal

changes in lead II, III and AVF, consistent with an anterolateral myocardial infarction (figs 1 and 2). Chest x ray showed clear lung fields and a normal appearance of the mediastinum. Transthoracic echocardiogram in the emergency department showed a non-dilated right ventricle, mild impairment of left ventricular systolic function with anteroapical segmental hypokinesia, and no visible thrombus.

A diagnosis of anterolateral myocardial infarction was made; the patient was given aspirin, clopidogrel and intravenous heparin, and arrangements were made for interhospital transfer for urgent primary percutaneous coronary intervention (PCI). Coronary angiography revealed an atheroma occluding the left anterior descending artery (LAD) proximally with normal circumflex and right coronary arteries. Angioplasty and bare metal stenting of the LAD lesion was performed. The patient was subsequently transferred from the coronary care unit to the maternity ward where she made a full recovery and underwent successful elective caesarean section at 39 weeks into her pregnancy.

DISCUSSION

Ischaemic heart disease in pregnancy is uncommon, occurring in an estimated 1 in 10 000 deliveries.¹ The highest incidence of acute myocardial infarction (AMI) has been reported in multi-gravidas older than 33 years during the third trimester.² Physiological changes in pregnancy result in a procoagulant state and increasing stress on the cardiovascular system, particularly during delivery. Cigarette smoking increases the risk of thrombosis due to enhanced platelet aggregability. If untreated, AMI in pregnancy has a high mortality up to 37–50%.³

A recently published report *Confidential enquiry into maternal deaths (CEMD), why mothers die 2000–2002*, stated that heart disease has become a leading cause of maternal deaths with 44 cases in the UK during 2000–2002. The vast majority of these deaths (35) were due to acquired heart disease, cardiomyopathy or myocardial infarction.⁴

The differential diagnosis of chest pain in pregnancy includes acute pulmonary embolism, aortic dissection, myocarditis and sickle cell disease. Management of confirmed AMI in pregnancy should involve early coronary angiography. Spontaneous coronary artery dissection with lesions in the LAD may occur; however, in this case, and in 20% of women with AMI in pregnancy, atherosclerosis is the underlying pathology.⁵ The anterior wall of the left ventricle is the most common location for AMI,⁶ and reperfusion is the priority. Medical management principles are similar to those for non-pregnant patients; both PCI⁷ and coronary artery bypass graft surgery⁸ have been performed successfully during pregnancy.

Thrombolysis of intracoronary thrombus with tissue plasminogen

Figure 1 ST elevation in leads I and aVL and evolving changes in V2, V3.

Figure 2 Acute anterolateral myocardial infarction, marked elevation in leads I, aVL, V2, V3 and reciprocal ST segment depression in leads II, III, aVF.

activator (tPA) is theoretically possible. The large molecular weight of tPA means it should not cross the placenta; however, there is a risk of premature labour and catastrophic haemorrhage.

Experience of the use of thrombolytics for AMI in pregnancy is limited.⁹ It may be considered if PCI is not possible as fetomaternal mortality is considerable in the absence of treatment.

Primary PCI is probably the treatment of choice even if "door to balloon time exceeds 90 minutes"¹⁰ and a cardiac network should be in place to allow for expeditious transfer to tertiary centres for definitive treatment. Due to the rare nature of AMI in pregnancy, there are currently no randomised trials comparing PCI to thrombolysis.

For the emergency physician attending these cases prompt recognition and early cardiology involvement for urgent primary PCI is the key. Initial treatment with aspirin and heparin is probably safe.^{2 11} In addition, newer antithrombotic drugs such as clopidogrel are increasingly being used before coronary intervention. The safety profile of clopidogrel in pregnancy, especially in combination with aspirin, is unknown

as no human studies have been performed.¹² Coronary angioplasty and stenting with optimisation of cardiac function including prenatal assessment by echocardiography is required before planned elective delivery. Integrated management by obstetricians, cardiologists and anaesthetists is essential. Invasive maternal monitoring during labour and delivery and continuous fetal heart rate recording should be considered.¹³

The mode of delivery in a pregnant patient with gestational myocardial infarction is determined by obstetric reasons and clinical status.

CONCLUSIONS

As the average maternal age increases, in part due to use of assisted reproductive technology, so may the incidence of AMI in pregnancy. The emergency physician needs to be aware that successful management of these cases requires a multidisciplinary approach including expeditious transfer for primary PCI if possible.

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