

Acute myocardial infarction in pregnancy following unlicensed use of methylenedioxymethamphetamine ('ecstasy')

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Scottish Medical Journal
58(3) e4–e6
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DOI: 10.1177/0036933013496918
scm.rsmjournals.com



Abstract

Acute myocardial infarction is rare in pregnancy; however, the emerging trend towards advanced maternal age and the rising prevalence of obesity and diabetes suggest that more cases of myocardial infarction are likely to be encountered in pregnancy. However, there is scanty evidence on the risk of myocardial infarction with regard to socially misused substances in pregnancy. We describe the management of a case of acute myocardial infarction following unlicensed use of 3,4-methylenedioxymethamphetamine ('Ecstasy') in pregnancy. The case highlights a rare but serious risk associated with substance misuse in pregnancy.

Keyword

Pregnancy, myocardial, infarction, 'ecstasy'

Introduction

There is some evidence that at an advanced maternal age, the presence of chronic diseases such as diabetes and hypertension and lifestyle choices such as smoking are important factors towards an increased risk of acute myocardial infarction (AMI) in pregnancy.¹

The cardiac risk posed by a socially misused substance such as methylenedioxymethamphetamine ('Ecstasy') has yet to be quantified in pregnancy. Substance misuse remains a significant burden in some antenatal populations in both developed and developing world;^{2,3} it is imperative to raise the awareness of the risk of myocardial infarction which carries a potentially high morbidity and mortality.¹

Case report

A 32-year-old Gravida 5 Para 4 woman with an otherwise uneventful obstetric history was admitted to the Accident and Emergency Department at 33 weeks gestation with sudden onset of severe chest pain following the ingestion of 'Ecstasy'. It was her first attempt at taking 'Ecstasy' which was offered to her by a new acquaintance.

Her initial 12-lead ECG revealed sinus tachycardia with ST segment elevation suggestive of an anterior infarct. She was stabilised and immediately transferred to the cardiac catheterisation laboratory, and coronary angiography was performed via the right femoral artery. A large thrombus was found to be occluding the proximal part of the left anterior descending artery (LAD). There was also some evidence of distal LAD occlusion. No evidence of atheromatous plaque or coronary artery dissection was found. The thrombus was aspirated and primary transluminal coronary angioplasty was completed. The patient did not require a stent. Post procedure fetal monitoring with ultrasound and cardiotocograph was satisfactory.

In the immediate post operative period on the Coronary Care Unit (CCU), she received Enoxaparin,

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Aspirin, Clopidogrel and Eptifibatide. She also received two doses of betamethasone 12 mg, 24 h apart in anticipation of possible preterm delivery. Her Troponin T was 0.9 µg/L (normal <0.5 µg/L) and her cholesterol was raised at 7.2 mmol/L (normal <5 mmol/L). Transthoracic echocardiography carried out revealed hypokinesia of the anterior wall and septum, indicating mild left ventricular dysfunction. She made good progress on the CCU and was transferred to the antenatal ward after one week. A multidisciplinary team review involving the Cardiologist, Obstetric Anaesthetist, Obstetrician, Senior Midwife and a Social Worker took place prior to her discharge from the antenatal ward. The review finalised further follow-up care and advance planning for the timing and mode of delivery. She was discharged home on Aspirin, Clopidogrel and Bisoprolol.

Maternal and fetal well being was reassuring at subsequent follow ups, and she was admitted for a planned induction of labour at 37 weeks gestation. Clopidogrel was stopped five days before the planned induction and recommenced post partum. She had an uneventful labour and a relatively quick spontaneous vertex delivery of a live baby boy weighing 3050 g with apgars of 9, 10 at first and fifth minute, respectively. She was monitored for 24 h on the Obstetric High Dependency Unit and was subsequently transferred to the post natal ward from where she was discharged home on day 2 post partum. At an initial follow-up at two weeks post partum, her cardiac status remained stable and she remained on Aspirin, Clopidogrel and Bisoprolol.

Discussion

AMI during pregnancy and the puerperium remains a rare event but carries a significant risk of maternal mortality.^{1,4} The majority of reported deaths occurred at the time of the infarction, or within two weeks in association with labour.⁵

The diagnosis of AMI is one that must be made promptly in order to reduce the morbidity and significant mortality associated with the condition. In addition to the traditional risk factors such as hypertension and diabetes, our case demonstrates that a history of amphetamine use is a less known but an important risk factor for AMI in pregnancy. Identifying the risk factors for a disease naturally raises the index of suspicion for making a prompt diagnosis.

The pathophysiologic basis of most AMI in pregnancy is attributed to coronary artery thrombosis secondary to atherosclerotic plaque destabilisation.^{4,6} However, this does not appear to be the mechanism in our patient. The primary pathology appears to be caused by coronary artery thrombosis in the absence of atherosclerosis. The possible mechanism in

amphetamine-related AMI includes sudden release of catecholamine and coronary artery vasospasm. The released catecholamine then induces platelet aggregation and sets the stage for thrombus formation in a pro-coagulant pregnant state.⁷

The diagnostic criteria for AMI in pregnancy are the same as in non-pregnant patients; however, it must be recognised that some of the diagnostic features on ECG can overlap with features of normal pregnancy.^{5,6,8} A practical consideration in the initial assessment of patients with suspected AMI in pregnancy would be to triage such patients whenever possible directly to Accident and Emergency Departments rather than the traditional obstetric triage units to avoid delays in diagnosis and treatment.

The cornerstone of managing AMI consists of urgent reperfusion therapy and pharmacological management. Percutaneous coronary intervention (PCI) is becoming the treatment of choice for revascularisation following AMI in pregnancy with reassuring reports on both its safety and efficacy.^{9–11} Stent placement may be required depending on the findings during PCI. Pharmacologic management in the acute phase consists of anticoagulation with low-molecular-weight heparin and antiplatelet therapy with Aspirin and Thienopyridine derivatives such as Clopidogrel. The role or presence of a senior obstetrician in the cardiac catheter lab at the time of performing PCI in the rare event of a need to perform *peri mortem* caesarean section is debatable.

Following the acute phase management in a CCU, an individualised approach to the timing and mode of delivery should be agreed following a multidisciplinary team review.^{1,12} The input of a substance misuse liaison midwife and social services would be beneficial in cases of AMI linked to the use of unlicensed medications to address the risks associated with the high-risk behaviour.

The mode of delivery following AMI remains an area of controversy because of paucity of evidence on which to base such decisions. It is therefore important to individualise such decisions on the basis of maternal and fetal well being and the presence or absence of any additional obstetric complications.^{1,4,5} Elective caesarean section offers the opportunity to plan delivery and avoids the unpredictable haemodynamic effects of labour and vaginal delivery. However, a successful vaginal delivery reduces the risks associated with anaesthesia and surgery.^{1,5,12} Our case has demonstrated that with careful multidisciplinary planning, vaginal delivery can be safely achieved following AMI in pregnancy. Practical points in planning vaginal delivery in such cases include consideration for elective induction of labour to allow timely discontinuation of Clopidogrel for about five days before delivery; continuous

maternal ECG monitoring and elective epidural in early labour, shortened second stage with elective forceps delivery and avoidance of ergometrine in the third stage. Post partum monitoring in a critical care setting on the delivery suite is advisable for the first 24 h and Clopidogrel should be recommenced in the post partum period. In view of the lack of data regarding the secretion of Clopidogrel in breast milk, it is advisable to avoid breastfeeding.^{6,13}

In conclusion, the reality of modern obstetric practice suggests that increasing cases of AMI are likely to be encountered in pregnancy, and hence, the need for obstetric, midwifery and emergency physicians staffs to have an increased awareness. Preventable causes of AMI such as those induced by unlicensed use of Amphetamines as described in this case can be prevented through antenatal education and targeted support for those patients who are most at risk.

Declaration of conflicting interests

None declared.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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