Ventricular fibrillation and reinfarction in pregnancy

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Summary: This is the first reported case of a woman suffering a potentially fatal dysrhythmia and reinfarction during the same pregnancy and surviving to produce a healthy, live infant.

Introduction

We report a woman suffering ventricular fibrillation due to acute myocardial infarction in the middle trimester, reinfarction later in pregnancy and surviving to produce a live healthy infant.

Case report

A 39 year old para 5 + 5 housewife who smoked 15 cigarettes a day and weighed 85 kg was booked for ante-natal care at 16 weeks gestation. She had no significant medical or family history. At 18 weeks gestation she presented complaining of retrosternal discomfort and vomiting for one hour. Her pulse was 84 beats/minute and blood pressure 130/70 mm Hg, as at her ante-natal clinic visit.

While in the Casualty Department she collapsed and was found to be in ventricular fibrillation. She was successfully cardioverted with one shock of 320 Joules and transferred to the coronary care unit. Her blood pressure was 90/80 mm Hg. The fetal heart rate was 140 beats/minute and regular. The electrocardiogram showed evidence of a fresh antero-septal transmural infarction and this was confirmed by an elevation of the cardiac enzymes. A random blood sugar was normal. Fasting lipids 12 weeks post infarct were normal.

The subsequent course of the pregnancy was uneventful until 33 weeks gestation when she was re-admitted as an emergency with a further episode of chest pain. Her blood pressure was 75/50 mm Hg and pulse rate 110 beats/minute. The electrocardiogram showed fresh changes of an acute anterior myocardial infarction and cardiac enzymes were again elevated. She was re-admitted to the coronary care unit but took

her own discharge against medical advice after four days.

The remainder of the pregnancy was marked by increasing breathlessness without overt cardiac failure. Fetal growth was normal.

She was readmitted at term. Labour was induced with prostaglandin E3 pessaries and progressed satisfactorily until the fetal cardiotocograph showed decelerative changes in association with maternal chest pain. The pain was rapidly relieved by sublingual glyceryl trinitrate and the decelerative changes disappeared following this. A live male infant (3.08 kg) was delivered with Wrigley's forceps. Apgar score was 7 at one minute and 9 at three minutes.

The child had passed all his developmental milestones at twelve months. The patient remains severely breathless on exertion having an exercise tolerance of only twenty yards. The most recent electrocardiogram shows the previous infarctions but no other ischaemic changes. An echocardiograph recorded several months post-delivery showed a dilated left ventricle with an akinetic septum and reduced posterior wall movement. A resting thallium scan confirms the dilatation of the left ventricle and shows decreased uptake by the myocardium and abnormal uptake in the lungs.

Discussion

Myocardial infarction in pregnancy is a rare condition. It was first reported by Katz¹ in 1922 and has an estimated incidence of 1 in 10,000 deliveries.² There are only 68 well documented cases reported in the literature and there are only four cases of reinfarction.

The disease carries a poor prognosis with an overall maternal mortality of 28%. Mortality ranges from nil in the first trimester to 40% in the second, 21% in the third and 50% in the puerperium.² The mortality also varies with age. Surprisingly women of 35 years or older have a better prognosis than those who are

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Accepted: 30 July 1987

younger (34% mortality vs 43%).³

From the 68 cases there are five cases of successful cardioversion with delivery of a live infant in four.³⁻⁷ Our report is we believe the first report of a woman surviving cardioversion from ventricular fibrillation and a second myocardial infarction in pregnancy to produce a live infant.

The reasons for myocardial infarction and its variable mortality in pregnancy are unknown. Our patient was at risk because she was an obese smoker. However, she had no antecedent heart trouble and no other known risk factors. There have been several reports of myocardial infarction with normal coronary arteriograms and it was suggested that these were due to coronary artery spasm provoked by angiotensin stimulated by uterine renin release.⁸ All of these reports were of patients presenting in the third trimester and the proposed initiating factor (decreased uterine blood flow induced by lying supine) would not apply in this patient's first presentation although it may have had a role in the second.

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This patient's second infarction occurred in the third trimester and this accords well with the four previous reports of reinfarction in pregnancy.⁹ Reinfarction seems to have a particularly high mortality if it occurs during labour and our patient (who had chest pain at this stage) was fortunate to survive.

The management of labour is problematical. Induction with oxytocin is theoretically disadvantageous in this situation because of fluid retention and effects on myocardial function. Animal experiments have shown that prostaglandins of the E series have some cardiodilator activity so there may be some rationale for their use as labour inducers in this type of case.¹⁰

It appears that vaginal delivery is safer than Caesarian section unless there is a specific obstetric indication for the latter.^{2,3} Labour must not be prolonged because of the strain on the myocardium and it is probably wise to assist the second stage.

Our patient has been left with a greatly reduced cardiac reserve and must avoid further pregnancy. She has refused any further cardiological investigation.