Myocardial Infarction in Pregnancy
Case Report and Brief Review

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A 40 YEARS OLD Chinese lady, gravida nine, para eight, suffered two episodes of myocardial infarction in pregnancy, one at eight weeks and the other at seventeen weeks of gestation. She survived both episodes to give birth to a healthy male infant at terms. Despite medical advice and contraception, she became pregnant again two years later. This time the patient agreed to termination and an abdominal hysterotomy with tubal ligation was performed at 14 weeks gestation.

Case Report

K.Y., a 40 years old Chinese lady, gravida nine, para eight, was admitted to the University Hospital on 6th March 1968 with the diagnosis of recurrent myocardial infarction in pregnancy. Her last menstrual period was on the 7th of November 1967. On 2nd January 1968, at eight weeks gestation, she suddenly experienced a severe epigastric pain which radiated to the root of her neck. This was associated with cold sweat and dyspnoea. Soon after, she collapsed and was admitted to the Assunta Hospital where she was warded for fifty days. The pain subsided slowly after three days of injections and medications. One week after discharge, she again had severe epigastric pain, now associated with palpitations. The epigastric pain was aggravated by activity and relieved by rest. There was no orthopnoea, paroxysmal nocturnal dyspnoea or ankle oedema. She was admitted to the University Hospital on 6th March 1968, at seventeen weeks of gestation.

On examination, her blood pressure was 100/70 mm Hg. and her pulse was 120 per minute with extrasystoles. The jugular venous pressure was not raised and there was no ankle oedema. The apex beat was 12 cm from the mid-sternal line outside the mid-clavicular line in the 5th intercostal space. There were no thrills and the heart sounds were normal. There were no heart murmurs. The uterus was enlarged to eighteen weeks gestational size. Electrocardiagram showed Q waves with raised S-T segments and inverted T waves in the chest leads best seen in V3 and V4, indicating acute anterior myocardial infarction. Chest X-ray showed an increased transverse diameter of the heart with prominence of the left ventrivular segment consistent with left chamber enlargement; but on fluroscopy after delivery, showed no aneurysm. Serum glutamic oxalacetic transaminase (SGOT) was normal (10 IU/1) and so were her haemoglobin, white cell count, blood urea and electrolytes. The Kahn and Wasserman tests were negative. The serum cholesterol was 302 mg per cent and the erythrocyte sedimentation rate was 27 mm per hour. She was treated by bed rest, sedated with phenobarbitone and the pain relieved by injection pethidine, codeine phosphate tablets and glycerine trinitirte. No anticoagulants were given. She made an unevenful recovery and was discharged forty days after admission to be followed up in the antenatal clinic. Her pregnancy progressed uneventfully and her cardiac status remained satisfactory.

At the 29th week of her pregnancy, she was readmitted for bed rest. On 2nd June 1968, four days after admission, she developed severe retrosternal pain radiating to the back of her chest. An electrocardiogram showed further elevation of the S-T segments in leads I, AVL and V2 to V5 suggesting further ischaemic episode. She was again

treated with analgesics, sedation, bed rest and glycerine trinitirte. She improved and on the 1st of July 1968, she discharged herself against medical advice.

On 7th August 1968, she went into spontaneous labour and was admitted to the labour ward. The first stage of labour which lasted a total of eleven hours was uneventful. She had a spontaneous delivery to a live male infant weighing 2640 grammes after a very short second stage of seven minutes. Throughout labour, her heart was well compensated and she suffered no ischaemic episodes. No oxytocics was given and the blood loss was minimal. The puerperium was uneventful and she was discharged fourteen days after delivery. She was advised sterilization but she refused.

At the post-natal follow up, an intra-uterine contraceptive device was inserted. She was followed up in the cardiac clinic and remained well. However, she removed the contraceptive device and became pregnant again. In view of her previous myocardial infarction and her present cardiae status, her pregnancy was terminated by abdominal hysterotomy and tubal ligation on 14th December 1970 at 14 weeks gestation. She has since remained well.

Discussion

So far, there are forty-five reported cases of myocardial infarction in pregnancy and labour confirmed by electrocardiagram or enzyme studies (Hussaini 1971). Myocardial infarction is rare in women of the child-bearing age (Weinreb, German & Rosenberg 1957; Oliver 1970), since this condition occurs among elderly women. Also, circulating oestrogens in the pre-menopausal women is protective against atherosclerotic changes (Eilert 1949; Barr 1953). On the other hand, the total cholesterol increases and its distribution changes in normal pregnancy to resemble that found in patients with ischaemic heart disease; more cholesterol being attached to the p lipoprotein (Oliver & Boyd 1955). This is inspite of the greatly increased amounts of circulating oestrogens in pregnancy.

The diagnosis of myocardial infarction in pregnancy presents a clinical and electrocardiagraphic difficulty, since upper abdominal pain, flatulence and chest pain are common in normal pregnancy. Those symptoms may mask ischaemic heart disease and electrocardiographic diagnosis of the condition has to be corelated with the clinical picture (Fletcher 1967). In the later months of pregnancy, the QRS axis may rotate leftwards owing to the elevation of the diaphragm and a deep Q wave deflection may appear in lead III. Similarly, active pulmonary embolism may produce abnormal Q wave deflection, especially in lead III and S-T segment and T wave changes which may mimic postero-diaphragmatic infarction.

Management consists essentially of supportive treatment to prevent cardiac decompensation, suppressing the thrombo-embolic tendency which is considerable during pregnancy and management of the pregnancy itself. The patient should be adequately sedated and potent analygesics given. These drugs will help to reduce the shock. Angina can be treated with glycerine trinitrite and cardiac failure by digitalisation. In the presence of shock, congestive cardiac failure, embolism or recurrent myocardial infarction, anticoagulants should be employed. This is even more so in the pregnant patient because of venous stasis in the lower limbs and the pelvis and the increased thrombo embolic tendency in pregnancy (British Medical Journal 1970).

Subsequent management of the pregnancy depends on the cardiac status. If failure and angina persists, it appears that termination of the pregnancy is of remarkable therapeutic value (Hussaini 1971). Of forty-six patients with myocardial infarction in pregnancy and labour, thirteen died and of these, eleven were cases of myocardial infarction occuring at or after thirty two weeks of gestation. The other two occured at five and six months respectively and died soon after the episode. It would seem that myocardial infarction in the later part of pregnancy carries a far worse prognosis than if it occured in early prgnancy. All patients who recovered from the initial infarction survived the rest of pregnancy and labour. This may be because in early pregnancy, there is time for the patient to convalesce before being subjected to the strain of labour.

Management of labour depends entirely on the cardiac status and the prospect of a short and easy vaginal delivery. In a multiparous woman in whom reasonably easy vaginal delivery is expected, this is the route of choice. On the other hand, if her cardiac status is decompensated and labour might be expected to be prolonged by say disproportion, then Caeserean section is prefered. The anaesthetic risks involved and the danger of hypotension should be weighed against the hazards of permitting a patient who has just suffered a myocardial infarction to labour relentlessly. Of nine cases who underwent Caeserean section, one died. She had a myocardial infarction near term (Lynge 1961). Ergometrine should not be given routinely in the third stage in patients who have recently suffered a myocardial infarction. The ergot group of drugs has long been known to cause generalized arterial and coronary vasoconstriction. Stein and Weinstein

(1950) concluded that Ergometrine B.P. in doses of 0.2 – 0.6 mg had a direct tonic effect on the coronary vasculature and Canning (1969) attributed a case of myocardial infarction immediately after delivery, to the administration of Syntometrine (ergometrine 0.5 mg; Pitocin 5 i.u.) at the time of delivery.

In the post-partum period, early mobilization is preferred. If this is not possible due to her cardiac status, one should consider anticoagulant therapy. The patient should be advised against further pregnancy and sterilization offered.

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