CLINICAL REVIEW

Myocardial infarction during pregnancy: report of two cases with a review of the literature

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Summary

Two further cases of myocardial infarction during pregnancy are reported.

From the review of the literature of forty-three cases of myocardial infarction during pregnancy and labour, it appears that myocardial infarction in the last trimester and labour is frequently fatal.

Short-term anticoagulant therapy to suppress any thrombo-embolic tendency is desirable.

Termination of pregnancy is indicated for patients in cardiac failure or persistent angina. For patients who are well, either assisted vaginal delivery or Caesarean section are equally good.

Introduction

Myocardial infarction is uncommon in women of childbearing age (Weinreb, German & Rosenberg, 1957; Oliver, 1970). In several reviews of heart disease during pregnancy myocardial infarction is only occasionally listed as a cause of heart disease (Hamilton, 1947; Jones, 1951; Mendelson, 1960; Rothe & Schlawe, 1965; Mather, Abbas & Mehta, 1966).

It is probable that some cases of shock or chest pain are misdiagnosed (Maternal Health in Ohio, 1958; Brown, 1960).

In this paper two patients are described who developed myocardial infarction during pregnancy.

Case reports

Case 1

H. M., age 40, was admitted under the care of Dr S. Oleesky on 6 August 1963, gravida three, para two, last menstrual period 1 May 1963. For 6 weeks she had been having pain in the left arm with a feeling of heaviness across the chest produced by climbing uphill, forcing her to stop, with relief in a couple of minutes. She had hypertension during the two previous pregnancies in 1954 and 1960, but

after each pregnancy her blood pressure fell to normal. There was no albuminuria during pregnancies.

On examination her blood pressure was 210/120 mmHg, otherwise no abnormality was detected. Haemoglobin, white cell count, blood urea and electrolytes, urinary catecholamines and 5-hydroxy-indoles, chest X-ray and ECG (Fig. 1) were normal. Three days later she developed sudden severe chest pain and appeared pale and shocked, with low volume pulse and BP 130/100 mmHg; ECG now showed evidence of inferior myocardial infarction with ST depression V3-V6. Serum glutamic oxaloacetic transaminase (SGOT) was elevated to 190 and serum glutamic pyruvic transaminase (SGPT) to 92 units. Phenindione therapy was commenced. Her general condition improved, BP settling to 110/70 mmHg. Fourteen days after admission she had a recurrence of chest pain, and BP fell to 90/60 mmHg. Further ECG showed no evidence of a fresh ischaemic episode. Subsequent progress was uneventful and she was discharged 1 month later on anti-coagulant therapy. During follow-up she remained well. The serum cholesterol on 9 October 1963, was 365 mg/100 ml, BP 120/70 mmHg.

During the seventh month of gestation she was re-admitted with exertional dyspnoea, peripheral oedema and BP 170/95 mmHg. Anticoagulants were discontinued. There was little response to digoxin, diuretics, salt restriction and hypotensive drugs, and the BP remained around 180/100 mmHg.

On the 18 January 1964, a live infant was delivered by Caesarean section, with rapid relief of symptoms. Oedema resolved and BP fell to 110/80 mmHg. One week later she developed deep vein thrombosis of the left leg, and phenindione was given for 5 weeks. One month after Caesarean section BP had risen to 150/95 mmHg. She was maintained on digoxin and diuretics as an out-patient. Three months post-partum she collapsed and died suddenly.

Necropsy (Dr Allison): the heart weight was 340 g. There was no hypertrophy or dilatation of atria or

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FIG. 1. ECG Case 1.

ventricles. Extensive coronary artery atheroma was present, with occlusion of the right coronary artery by calcified atheroma and several areas of narrowing of each of the main branches of the left coronary artery. No recent thrombosis was found. Atheroma was also present in the thoracic and abdominal aorta. Numerous cholesterol stones were present in the gall bladder.

Histology. Posterior wall of left ventricle replaced by fibrous tissue. No evidence of recent muscle

necrosis. Recanalized occlusion of right coronary artery. Hypertensive changes were not seen in the renal arteries.

Case 2

J. D., age 32, was admitted under the care of Dr A. Morgan Jones on the 14 December 1966. Gravida four, para one, last menstrual period 8 April 1966. She had been depressed for some years and received diazepam 5 mg t.d.s. intermittently. In November, she had an influenza-like illness, which increased her depression.

On the 19 December 1966, she felt hot, and then cold, with shivering and palpitations. She complained of a chest pain like a constant tightness under the sternum radiating to the back and associated with pain in the left arm. This persisted intermittently for a few days. She developed dyspnoea, cough and orthopnoea, but no paroxysmal nocturnal dyspnoea.

On examination her blood pressure was 120/70 mmHg, heart rate 110/min, jugular venous pressure not raised. There was no oedema and no basal crepitations. ECG (Fig. 2) on 14 December 1966, showed inferolateral myocardial infarction. Other investigations on 14 December 1966, were: total WCC 11,200/mm³, differential normal, Hb 9.8 g/100 ml, ESR 61 mm in first hour. Serum albumin 2.8 g/100 ml, serum globulin 2.9 g/100 ml, serum cholesterol 190 mg/100 ml, serum lactic acid dehydrogenase, SGOT, SGPT, bilirubin, alkaline phosphatase, thymol turbidity, blood urea and electrolytes normal. No lupus erythematosus cells detected. Antinuclear factor test was negative.

She was treated with bed rest and digoxin. On 27 December 1966, she had a normal spontaneous delivery of a live infant.

X-ray of chest after delivery showed prominent left cardiac border, but on fluoroscopy no aneurysmal changes were seen. She was discharged home well on 25 January 1967. Intermittent chest pains of atypical nature persisted post-partum.

She became pregnant again about the beginning of June 1967. In view of endogenous depression, previous infarction and frequent chest pains of indeterminate nature, therapeutic evacuation of the uterus and sterilization were carried out in the beginning of September 1967. Subsequently she continued to complain of vague symptoms but there was no heart failure and no enlargement of the heart.

In 1968 she was admitted with an attack of ventricular tachycardia which necessitated cardioversion. No fresh changes of cardiac infarction were discovered.

Discussion

Forty-three cases of proved myocardial infarction during pregnancy or labour have been reported in

FIG. 2. ECG Case 2, 14 December 1966.

the world literature (Table 1). In addition, Gordon (1955) in a post-mortem series of 176 maternal deaths in Brooklyn found three cases of myocardial infarction; as no clinical details are available these cases have not been included in the Table. Three cases reported by Phillips (1962) and one case reported by Ginz (1970) are excluded from the discussion as neither the ECG nor enzyme studies are recorded in the publications. Froehlich (1963, 1968) has reported one case of generalized T-wave inversion in a pregnant woman who was admitted with a history of loss of consciousness and convulsions followed by purposeless movement of the left upper arm. This case is excluded because of uncertainty of diagnosis.

The month of gestation at the onset of cardiac infarction, the presence of hypertension, method of

FIG. 3. The month of gestation at onset of cardiac infarction, presence of hypertension, method of delivery and mortality. Open columns, normal blood pressure; diagonally hatched columns, raised blood pressure; ?, blood pressure not known; C, Caesarean section; †, did not survive; UN, month of gestation not known; N, same patient—see text.

delivery and mortality are shown in Fig. 3. Thirteen died during pregnancy or soon after delivery, eleven of these deaths occurring amongst those who suffered infarction during the last months of pregnancy or during labour. Sixteen of the forty-five had hypertension; four of these died, all of the latter sustaining cardiac infarction during the last months of pregnancy (Mendelson, 1952; Stewart, 1952; Committee on Maternal Welfare, 1957), and expiring soon after infarction. The blood pressure was normal in twenty-five. Seven of these died. One patient (Brown, 1960) who had cardiac infarction in the sixth month of pregnancy died at home, 24 hr after discharge against medical advice. Another patient (Lyng, 1961) who had infarction near term died soon after Caesarean section. A third patient (Curry & Quintana, 1970) after having survived an infarction during the sixth month of pregnancy had a recurrence during the tenth month and died (patient N, Fig. 3). Four patients (Magner, 1961; Krawczuk & Jakowicki, 1965; Pantev & Piperkov, 1968) had cardiac infarction during labour and died rapidly with severe shock and pulmonary oedema. One other reported case of myocardial infarction during labour (Katz, 1922) died; the blood pressure of this patient is not known. The blood pressure of the patient who had cardiac infarction during the fifth month of pregnancy and died (Muir, 1960) is also not known.

The patient of Pantev & Piperkov (1968) is interesting as infarction was considered to be due to

TABLE 1. Reported cases of myocardial infarction during pregnancy showing age and important complications

Author	Year of publication	Age of patient	Complications
Katz	1922	33	—
Reiss & Frankenthal	1935	45	Nephrosclerosis; moderate heart failure
White, Glendy & Gustafson	1937	22	—
Jensen	1938	39	—
Hamilton & Thompson	1941	?	—
		42	—
Goldberger & Pokress	1950	37	Rheumatic heart disease; heart failure; tertiary syphilis
Mendelson	1952	42	Known coronary artery disease; toxæmia previous pregnancy
		42	Marked heart failure
		35	Oedema+
Stewart	1952	?	Acute pulmonary oedema
		42	—
Brock <i>et al.</i>	1953	34	Diabetes; toxæmia second pregnancy
Antonius <i>et al.</i>	1955	36	Basal rales; albuminuria + +
Siegler <i>et al.</i>	1956	38	Diabetes mellitus; cholelithiasis, renal lithiasis, thrombophlebitis
Forssell & Brunila	1957	44	Pulmonary oedema day after infarction
		38	—
Myers & Sharpe	1957	39	—
Committee on Maternal Welfare	1957	41	—
Brown	1960	24	—
Watson <i>et al.</i>	1960	22	—
Muir	1960	26	—
Jacobs & Moress	1961	34	—
Lynge	1961	25	Previous rheumatic fever; familial hypercholesterolaemia
Magner	1961	29	Acute pulmonary oedema
Naden <i>et al.</i>	1961	39	—
Shapiro <i>et al.</i>	1962	33	Mild failure
Bechtel <i>et al.</i>	1963	35	Post-partum thrombophlebitis
Bedford	1964	36	Two previous myocardial infarctions; pre-eclampsia in one of the previous pregnancies
Pfaffenschlager	1964	21	Severe shock
Jewett	1965	35	Presented as C.C.F.
Krawczuk & Jakowicki	1965	23	Severe shock; pulmonary oedema
		27	Severe shock; pulmonary oedema
Listo & Björkenheim	1966	28	Severe shock; pulmonary oedema
Fletcher	1967	38	No failure
		36	Toxaemia during previous pregnancies
		36	No failure
		38	No failure
		41	No failure
Adler, Lash & Barrash	1968	40	No failure
Pantev & Piperkov	1968	26	Paroxysmal tachycardia
Capra Marzani & Navazzotti	1969	38	—
Curry & Quintana	1970	29	No failure
This paper	1971	40	Cardiac failure
		32	Shortness of breath

paroxysmal tachycardia. The coronary arteries were normal at necropsy.

Sedation

Adequate analgesic therapy is required. These drugs help reduce shock and with reasonable care and awareness of the risk to the foetus they can be used during labour.

Anticoagulants

Anticoagulants suppress the thrombo-embolic tendency, which is considerable during pregnancy (*British Medical Journal*, 1970) with enforced bed rest. The risk to the foetus is probably over-emphasized. However, careful control and choice of anticoagulant are important (Hirsh, Cade & Sulli-

van, 1970) and after mobilization its maintenance of questionable value. Twelve patients received anti-coagulants (Mendelson, 1952; Stewart, 1952; Brock, Russell & Randell, 1953; Forssell & Brunila, 1957; Myers & Sharpe, 1957; Naden, Jackson & Murray, 1961; Shapiro *et al.*, 1962; Bedford, 1964; Capra Marzani & Navazzotti, 1969; Curry & Quintana, 1970—first admission—and Case 1). Six of these had hypertension (Mendelson, 1952; Stewart, 1952; Brock *et al.*, 1953; Forssell & Brunila, 1957; and Case 1) and five were delivered by Caesarean section (Mendelson, 1952; Stewart, 1952; Forssell & Brunila, 1957; Naden *et al.*, 1961; and author). All survived. Only one foetus was lost (Forssell & Brunila, 1957).

Method of delivery

After the acute stage is over one is faced with the question of the safest method of management of the rest of pregnancy. If failure or angina persists it appears that termination of pregnancy is of remarkable therapeutic value. Three such patients (Pfaffenschlager, 1964; Listo & Björkenheim, 1966; and Case 1) improved after Caesarean section. The patient of Listo & Björkenheim is unique since infarction occurred during labour; the patient developed severe shock, yet evacuation of the uterus promptly relieved shock.

Eleven patients (Mendelson, 1952; Stewart, 1952; Forssell & Brunila, 1957; Watson *et al.*, 1960; Lynge, 1961; Naden, Johnson & Murray, 1961; Bechtel, Lanford & Mangone, 1963; Pfaffenschlager, 1964; Listo & Björkenheim, 1966; Fletcher, Knox & Morton, 1967; and Case 1) were delivered by Caesarean section. Ten survived, one (Lynge, 1961) died a few hours after Caesarean section. The surgical and anaesthetic risks to cardiac patients are small with present techniques. If asymptomatic patients are allowed to progress to labour spontaneously, then, as in other heart diseases the second stage should be assisted. The burden of work of labour should be as short as possible.

Post-partum management

Early mobilization is to be preferred unless there are cardiological reasons to the contrary. If prolonged bed rest is envisaged then there might be an indication for recommencement of anticoagulant therapy. Case 1 of this series developed thrombophlebitis after Caesarean section and received anticoagulants. The patient of Bechtel *et al.* (1963) who developed mild thrombophlebitis after Caesarean section did not receive anticoagulants.

Of the patients who sustained a cardiac infarction before labour and made a good recovery from the acute episode before reaching the time of delivery none failed to survive the remainder of pregnancy and labour. It appears, therefore, that once a patient has made a satisfactory recovery from an episode of acute myocardial infarction the remainder of the pregnancy does not have any deleterious effect on the cardiac state. This assumption is supported when one takes account of some of the reported cases (Leff, 1950; Lyons & Lyons, 1954; Smith & Gatenby, 1958; Jackson *et al.*, 1965; Rhen & Salokannel, 1967; Canning, Green & Mulcahy, 1969) of women who had myocardial infarction before becoming pregnant and surviving it. However, the case of Offergeld (1931) who suffered from angina, died suddenly during the middle of the pregnancy, the case of Bedford (1964) who had had two previous cardiac infarctions developed a third infarction during pregnancy, the case of Curry *et al.* (1970) who had an

infarction before pregnancy had two further infarctions during the same pregnancy and died from the last, and Case 1 of the author was re-admitted for treatment of heart failure after having been discharged well. Coupled with the fact that according to the published cases cardiac infarction during the last trimester and labour is frequently fatal, pregnancy would be contra-indicated in a patient who suffers from ischaemic heart disease.

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