Acute Myocardial Infarction in Pregnancy

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Obstructive coronary artery disease is the rarest form of primary organic heart disease encountered during pregnancy. Myocardial infarction occurring during pregnancy was confirmed for the first time at necropsy by Katz (1921). The first case of recurrent myocardial infarction during pregnancy and labour, with electrocardiographic evidence, was published in 1935 by Reis and Frankenthal. Since then a few further case reports have been published by various authors. Bedford (1964) was able to collect only four cases in the British literature, to which he added one of his own. In a recent review Watson *et al.* (1960) collected 23 authentic cases of myocardial infarction during pregnancy or labour. To this number may be added the case of Magner (1961), that of Bedford referred to above, and the five cases described in this paper, a total of 30 in the world literature.

This paper presents the clinical and electrocardiographic data of five women who sustained major episodes of acute myocardial infarction while pregnant. During the period 1 January 1955 to 31 December 1966 at least 40,000 patients

attended the antenatal clinics of the Belfast City Hospital. The five patients already mentioned were selected from approximately 800 pregnant women referred to the cardiac department for assessment. They were all multigravidae, with an age range of 36-41 years. There was no maternal mortality in connexion with the pregnancies or puerperia. All but one (Case 5) pro-

FIG. 1.—Case 1. A (29th week of 4th pregnancy). Sinus rhythm. Abnormal S-T segment and T-wave displacement with grossly abnormal T waves, most pronounced in leads V2-V6. The tracing is consistent with the acute phase of antero-septal ischaemia or infarction. B (16 days after A). In the interval between the tracings there has been some correction of repolarization, but the underlying myocardium remains abnormal.

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Summary of Relevant Data in Five Cases

Case No.	Age	No. of Pregnancies	Week of Onset	Symptoms	Preceding Hypertension	Pre-eclamptic Toxaemia	Biochemical Findings				Follow-up
							S.G.O.T. (units)	S.G.P.T. (units)	Cholesterol (mg./100 ml.)	β-Lipo-proteins (mm.)	
1	38	4	29	Dyspnoea for one week, followed by severe crushing retrosternal pain radiating to neck and left arm. Pain lasted 1½ hours	No	No	150		280		Normal delivery of living child at 38 weeks. Puerperium uneventful. Persistent angina of effort over 7 years. Remains normotensive
2	36	7	30	No symptoms referable to C.V.S. before onset of this pregnancy. Sudden attack of severe substernal pain lasting several hours. Pain accompanied by sweating and dyspnoea	No	Yes. All previous 6 pregnancies resulted in stillbirths before 7th month on account of hypertension oedema and albuminuria	50		280		Living child by caesarean section at 40 weeks. Puerperium uneventful. Persistent angina of effort over 2 years 3 months Sudden death after severe episode of retrosternal pain. E.C.G. a few hours before death confirmed a fresh extensive myocardial infarction
3	36	4	32	Epigastric discomfort on exertion relieved by rest, followed 10 days later by crushing retrosternal pain radiating to both arms. Pain awakened patient from sleep and lasted 3 hours	No	No	95		280		Normal delivery of living child at 40 weeks. Puerperium uneventful. No recurrence of ischaemic cardiac pain during 2-year follow-up
4	38	2	30	Dyspnoea for 2 weeks followed by sudden onset of retrosternal tightness associated with flatulence	No	No	50				Normal delivery of living child at 38 weeks. Puerperium uneventful. No follow-up after discharge
5	41	22	13	2-year history of angina of effort before onset of this pregnancy. Awakened from sleep with crushing retrosternal pain radiating to left arm. Pain lasted five hours	Yes	Yes (onset at 28 weeks)	107	12	412	4.2	Intrauterine death at 29 weeks. Normal delivery of macerated foetus at 33 weeks. Puerperium uneventful. Still angina of effort during three-month follow-up

duced healthy children. The cause of the single foetal death was severe pre-eclamptic toxæmia. It was not related to the cardiac status of the mother. Two patients (Cases 2 and 5) had severe pre-eclamptic toxæmia of pregnancy, and this might be a contributing factor for the occurrence of coronary atherosclerosis. The other three women had no general medical condition likely to predispose them to coronary artery disease. The relevant clinical obstetric and biochemical data for each patient are summarized in the Table. Serial electrocardiograms were recorded in each case, and these are shown in Figs. 1-5.

Discussion

In a review of 45 cases of coronary artery disease associated with pregnancy by Mendelson (1960) 19 had systemic hypertension, 9 had cardiovascular syphilis, and 2 had diabetes mellitus. Two of our patients (Cases 2 and 5) had a systemic disease likely to predispose to coronary atherosclerosis—that is,

a long history of recurrent toxæmia of pregnancy with hypertension. The underlying disorder in all five cases appears to have been coronary atherosclerosis. In all but one of them the acute infarctions occurred in the third trimester, when the oxygen demand of the tissues is increased and the work load of the heart is at its maximum. During pregnancy the blood chemistry alters significantly; the total cholesterol increases and more is attached to the beta-lipoproteins. The pattern resembles that found in men with ischaemic heart disease (Oliver and Boyd, 1955). These changes are maximal about the thirty-third week, and they take place in spite of the great increase in circulating oestrogens, known effective agents in restoring the non-atherosclerotic plasma lipid pattern (Barr, 1953).

The dynamic factors and biochemical changes referred to above may have predisposed some of our patients to coronary occlusion at the critical weeks of gestation, on the assumption that they already suffered from coronary atherosclerosis. They are not, however, the whole answer, since one patient (Case 5)

FIG. 2.—Case 2. A (20th week of 7th pregnancy). Normal tracing. B (10 weeks after A). Sinus rhythm. Abnormal S-T segment depression in leads I, II, and V4-V6. An abnormal tracing when compared with A. C (two weeks after B). Abnormal S-T segment displacement in the limb leads with increased polarity of T waves in leads I, AVL, and V2-V6. The tracing is consistent with recent anterolateral infarction.

FIG. 3.—Case 3 (32nd week of 4th pregnancy). Sinus rhythm. Abnormal Q-S deflections in leads VI-V4. S-T segment and T-wave displacement upwards due to injury current in these leads and also in leads II, III, and AVF. The tracing is consistent with acute myocardial infarction.

FIG. 4.—Case 4 (30th week of 2nd pregnancy). Sinus rhythm. P-R interval 0.22 second. Abnormal S-T segment and T-wave displacement upwards, due to injury current in leads II, III, and AVF, and downwards in leads I, AVL, and V1-V6. The tracing is consistent with acute postero-diaphragmatic infarction and anterior-wall ischaemia.

FIG. 5.—Case 5. A (10 months before B). Normal tracing. B (13th week of 22nd pregnancy—10 months after A). Sinus rhythm. Abnormal S-T segment and T-wave displacement with increased polarity of T waves in leads I and AVL. Abnormal Q-S deflection in lead AVL. The tracing is consistent with recent anterolateral infarction. C (4 months after B). In interval following B considerable correction of repolarization has taken place but the tracing still remains abnormal.

developed her episode of coronary occlusion as early as the thirteenth week of gestation and she was the only one in our series in whom the serum cholesterol was significantly raised.

The diagnosis of myocardial infarction during pregnancy presents clinical and electrocardiographic difficulties. Accessory laboratory investigations for myocardial necrosis have to be interpreted with reservations. Upper abdominal pain, flatulence, and even chest discomfort are so common during normal pregnancy that they may mask ischaemic heart pain. The true nature of such symptoms may be obvious only retrospectively when an incidental electrocardiogram is recorded, as in the case reported by Watson *et al.* (1960). Normally the erythrocyte sedimentation rate increases progressively, reflecting changes in plasma proteins. Leucocytosis occurs, and at term the white cell count may be as high as 15,000/cu. mm. (Tysoe and Lowenstein, 1950). There is evidence that normally the serum aspartate aminotransferase activity does not deviate, though it may be elevated in toxæmia of pregnancy (Crisp *et al.*, 1959). Serum lactic dehydrogenase is a much less reliable index of myocardial necrosis, as it may be raised in normal pregnancy, and in particular during the puerperium. According to Smith and Gatenby (1958) this enzyme is elevated in 18% of antepartum patients and in 100% of postpartum patients.

Objective evidence of myocardial infarction, therefore, will depend on electrocardiographic analysis, and account must be taken of the electrocardiographic changes which may occur during pregnancy when the heart is not primarily involved. In the later months the mean QRS axis may rotate leftwards owing to elevation of the diaphragm (Hollander and Crawford, 1943) and a deep Q deflection may appear in lead III (Thomson *et al.*, 1938). Acute pulmonary embolism may produce abnormal Q deflections, especially in lead III and S-T segment and T-wave changes, which may mimic postero-diaphragmatic infarction. In addition, electrolyte imbalance may alter the voltage and polarity of the T waves. Intrinsic myocardial

disease other than infarction, such as myocarditis of pregnancy, may produce electrocardiographic changes indistinguishable from infarction. In short, electrocardiographic abnormalities must be correlated with all the clinical data. The subsequent history of our patients supports the view that the grossly abnormal tracings were in fact due to myocardial infarction.

Summary

Acute myocardial infarction is rare in pregnancy. The cases of five elderly multigravidae suffering from this condition are described. The diagnosis during pregnancy may present clinical and electrocardiographic difficulties. The increased load on the heart and changes in the blood lipids may predispose to acute infarction in late pregnancy in women with pre-existing coronary atherosclerosis.

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Oil Mist Hazard and Piped Air Supplies

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During recent years there has been a considerable increase in the use of piped air supplies in hospitals. In intensive therapy units this air may often be used to ventilate patients directly.

A system of this type was installed in the intensive therapy unit at Barnet General Hospital and has been in regular use from January 1965. The supply was provided by duplicate oil-lubricated Lacey-Hulbert compressors, each capable of delivering 5½ cu. ft. (0.156 cu. m.) of air per minute at 100 lb./sq. in. (7 kg./sq. cm.). These compressors were installed in the cellar below the intensive therapy unit, together with a reservoir of 33 cu. ft. (0.935 cu. m.) capacity. A Hymatic "rectifier" was installed to remove water. The compressed air was delivered by copper piping to five bedside outlets each equipped with B.O.C. flowmeters.

Some months later it was noticed that some of the flowmeter bobbins were inclined to stick. One of the flowmeters appeared to be slightly oily, and the possibility of oil mist contamination

was considered. When the valve of this flowmeter was completely opened to a point far beyond its normal setting, a ripple of oil could be seen on the side of the meter tube (Fig. 1). This excessive air flow had presumably dislodged a quantity of oil trapped below the valve.

An investigation to find the extent of this oil contamination was started. In view of the extremely small quantities of oil mist likely to be present in the air, it was decided to use a fluorimetric technique for oil estimation (Parker and Barnes, 1960).

Methods

Oil mist was collected by passing a known volume of air through filter paper held in a special device consisting of two recessed flanges fitted with hose connexions between which pairs of filter paper discs were held by washers and a spacer. The filter papers used in this work were Whatman paper No. 41, which will cope with low-pressure flows of at least 10 litres per minute. Owing to the rather open nature of the paper, two discs were used in each flange. The papers were

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