CARDIOMYOPATHY IN PREGNANCY: 2 CASE REPORTS

S Krishnamurthi, S V G Reddy, M Mohamed

ABSTRACT

Cardiomyopathy developed in the peripartum period in two healthy primigravida at 41 and 42 weeks of gestation are presented. In the first case, the patient presented with pulmonary oedema and had cardiac arrest immediately after admission. After resuscitation, a live baby was delivered by Caesarean section. Second cardiac arrest occurred after 12 hours of operation and the patient was resuscitated again. On the 8th post operative day, patient died of D.I.V.C. and renal failure.

The second case was a twin pregnancy associated with pregnancy induced hypertension. Caesarean section was performed for foetal distress. She developed pulmonary oedema with left ventricular failure on the second post operative day, was resuscitated and discharged on the 15th day.

These are the first two cases to be reported from Hospital University Sains Malaysia. Reference is made to three other cases in which a similar pathological process might have occurred.

Key words: Cardiomyopathy, Pulmonary oedema, Peripartum.

SING MED J. 1989; No 30: 208 — 209

INTRODUCTION

Peripartum cardiomyopathy is defined as a syndrome of cardiac failure occurring in the later part of pregnancy or in the puerperium without any obvious cause or prior evidence of heart disease (1). Diagnosis of this condition is usually one of exclusion (2). Invariably these could be misdiagnosed as a case of heart failure due to pregnancy induced hypertension. There were 3 other similar cases, with identical fatal outcome, in the four year history of this hospital. They were diagnosed as a case of cerebrovascular accident or left heart failure.

The cases presented here differ from other cases reported, in developing hypertension at term and they were post dated.

CASE NO. 1

A 26 year old Malay teacher, primigravida at 41 weeks, was admitted with generalised swelling of body, and difficulty in breathing for three days.

The symptoms of breathlessness worsened prior to admission and she developed cough with blood stained sputum. There was no family or personal history of heart disease or hypertension. She had attended the antenatal clinic from 20 weeks onwards, and her blood pressure was 110-120 systolic and 70-80 diastolic. Oedema was noted only at 39 weeks of pregnancy and the urine was protein free throughout except at 40 weeks when a trace of protein was detected. The total weight gain was 14.5 kg. and there was 2.5 kg. increase of weight in the two weeks prior to admission.

On examination she was cyanosed, severely dyspnoeic and there was generalised gross oedema present. Blood pressure was 200/130 and pulse was 120/min. Jugular venous pressure was raised and coarse crepitations were heard all over the chest.

No cardiac murmurs were detected. She had cardiomegaly. Deep reflexes were brisk. Abdominal examination showed uterus was term size, longitudinal lie and cephalic presentation, head not engaged. Pelvic examination revealed an unfavourable cervix. She was diagnosed to be in left ventricular failure with pulmonary oedema secondary to severe preeclampsia. She was intubated and ventilated with 100% oxygen. Morphine injection, frusemide, hydralazine infusion was also started simultaneously and the fetal heart rate was 146/min with no abnormalities noted with cardio tocography monitoring. Decision was made to deliver the baby by lower segment Caesarean section, because of severe preeclampsia and the unfavourable cervix. While preparation was under way to transport the patient to the theatre, she had a cardiac arrest. Patient required frequent endotracheal suction as she had excessive pink frothy fluid. Immediate resuscitative measures were carried out with cardiac massage, and E.C.G. showed ventricular fibrilation initially and later showed significant T wave inversion. After the emergency resuscitation patient's blood gas analysis showed a considerable improvement. The continuous fetal monitoring showed the fetus was alive, hence the lower segment Caesarean section was carried out. Post operative management was carried out in the intensive care unit. The blood pressure remained between 170-180 mmHg systolic and 120-130 diastolic. Her proteinuria which was 4+ on admission came down to 3+. The blood urea remained high from the time of admission and on the third day the patient developed D.I.V.C. controlled with fresh blood and F.F.P. Patient's renal profile deteriorated and she remained comatose. On the 8th post

Department of Obstetrics and Gynaecology
University Hospital
University Sains Malaysia
Kota Bharu
Kelantan
Malaysia

S Krishnamurthi, MBBS, DGO, MRCOG

Department of Anaesthesiology
University Hospital

S V G Reddy, MBBS, MD

Department of Medicine
University Hospital

M Mohamed, MBBS, MRCP, M Med,

Correspondence to: Dr Krishnamurthi

operative day, the patient had irreversible cardiac arrest.

CASE NO. 2

A 26 year old primigravida at 41 weeks of gestation, was referred to University Hospital for suspected multiple pregnancy. Her antenatal history was unremarkable except for the mild oedema and difficulty in feeling the fetal parts. Her blood pressure was 120/80 mmhg. and there was slight oedema over her legs.

Examination of abdomen showed that the uterus was term size and it was difficult to feel the fetal parts. There was fluid thrill. Ultrasound examination showed two fetal parts and both were presenting as cephalic. The biparietal diameters were 85mm. corresponding to 33 weeks of gestation, and the placental maturity was grade III. Pelvic examination showed a bishop score of 8. Since her haemoglobin was 9.5 gms/dil, she was transfused one unit of blood.

Patient went into spontaneous labour and ruptured her membranes with meconium stained liquor. Since the fetal monitoring showed type II deceleration, lower segment Caesarean section was carried out as an emergency measure under general anasthesia. Two female babies weighing 2550 gms and 2250 gms were delivered with the apgar score of 6 at one minute, 8 at 5 minutes and 5 at one minute, 9 at 5 minutes respectively. Post operatively she had moderate post partum haemorrhage, which was controlled with intravenous oxytocin and blood transfusion. Her post operative haemoglobin was 11.5 gms percent. On the second post operative day, she suddenly developed dyspnoea, tachycardia and her blood pressure increased to 190/110 mmHg. There were basal crepitations and auscultation of the heart showed triple rhythm with no murmurs. E.C.G. showed sinus rhythm, T wave inversion in Lead I and II, VI to V6 suggestive of anterolateral ischaemia. Immediate resuscitative measures were carried out. Echo cardiography showed enlargement of heart with hypokinetic segment. She was started on prazosin, oxygen and intravenous frusemide. On the ninth post operative days, the patient responded to the treatment and repeat E.C.G. showed sinus rhythm with no T wave inversion in any of the leads. The post operative period was otherwise uneventful. She was seen in the postnatal clinic five weeks after discharge, and had no complaints except for the weakness. The E.C.G. showed no abnormality and the echo cardiogram showed cardiomegaly with left ventricular dilatation with some hypokinetic segment. She was given an appointment at the cardiology clinic and advised barrier method of contraception.

DISCUSSION

Three elements are required to establish the diagnosis of peripartum cardiomyopathy. One, occurence during the peripartum period. Second, no previous history of heart disease. Three, no specific aetiology found (1). The three criteria are fulfilled by the two cases reported here. More advanced investigational techniques, such as endocardial catheter biopsy, immunological studies of this disease and echo cardiography would help us understand the pathophysiology of this syndrome better (1). Except for echo-cardiography the other investigations are not available in our hospital.

The incidence of cardiomyopathy in pregnancy is reported to be one in three thousand to four thousand pregnancies (1, 4, 5). Universiti Sains Malaysia Hospital data show one in 2900 deliveries. From a survey of the literature, it is noted that this syndrome occurs more frequently in the older multi-parous women, but is by no means restricted to these groups (1). In our series all the five patients were primigravida of younger age group and Malay in ethnic origin. Hull and associates reported a high percentage of hypertension and a survey of published cases shows that peripartum cardiomyopathy can occur even in patients whose blood pressures are normal, as was observed in our second case.

Twin pregnancies are at greater risk of developing peripartum cardiomyopathy (3, 7). These patients have greater haemodynamic perturbations and greater demand on nutritional reserves, both of which could be involved in the pathophysiology of the disease (1).

These patients have a higher incidence of embolic phenomena from intramural cardiac thrombi. Thus, in such patients anticoagulants are indicated (1, 2). As the M mode echo cardiography did not show any emboli in our second case, no anticoagulant therapy was started.

Although the actual mortality rate is not known, it has been estimated to be in the range of 30% to 60% for the mother and close to 10% for the infants whose mothers have congestive heart failure (1).

The prognosis of these patients are variable and appears to be related to persistence of cardiac failure and cardiomyopathy. Postnatal checkup of the second case showed no significant changes in her symptoms, signs, E.C.G., or in the echo cardiogram. She will be followed up in the cardiology clinic because of the tendency for recurrence.

ACKNOWLEDGEMENT

We thank the Deputy Dean and the Director for the permission granted and to Mrs. Roseni Ahmad for the typing of this manuscript.

REFERENCES

1. Jean-Claude V. Peripartum cardiomyopathy: A review Am J Obstet Gynecol March 15. 1984; 805-18.
2. Raman S, Rachagan SP, Marzuki A: Peripartum cardiomyopathy in a Malay female. Asia-Oceania J Obstet Gynaecol 1988; 1: 17-20.
3. Rand RJ, Jenkins DM, Scott DG: Maternal cardiomyopathy of pregnancy causing stillbirth. Br J Obst Gynaecol 1975; 82: 172-5.
4. Meadows WR: Post partum heart disease. Am J Cardiol 1960; 6: 788.
5. Seftel H, Susser M: Maternity and myocardial failure in African women. Br Heart J 1961; 23: 43.
6. Warembourg H, Niquet G, Ducloux G, Stainikiewicz G, Thibaut M. L'insuffisance cardiaque primitive gravidopuerperale: A propos de deux observations. Lille Med 1964; 9: 468.