PERIPARTUM CARDIAC FAILURE

(A Case Report)

by

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Peripartum cardiac failure is an uncommon, syndrome except in Northern Nigeria. Some consider it synonymous with peripartum cardiomyopathy (Stuart, 1968). It occurs in women during the last month of pregnancy or in the first 5 postpartum months. Few cases have been reported from India (Ghosh *et al,* 1968; Talwalkar *et al,* 1978). Such a case is reported and its various aspects are discussed.

CASE REPORT

A 28 year old female was hospitalized for breathlessness and swelling of the feet of 2 weeks' duration. Her 2 previous deliveries as well as the last delivery, about a month prior to the onset of her illness were normal. About 2 weeks after the delivery she developed breathlessness and oedema of the feet. Dyspnoea was progressive and she became orthopnoeic and was hospitalized. She also had attacks of paroxysmal nocturnal dyspnoea. She never had rheumatic fever or hypertension and there was no history of drugs or alcohol.

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On examination, she was well nourished and was orthopnoeic. Her pulse was 150/min. regular, respiration 44/min. and the blood pressure was 120/80 mm.hg. She had oedema of the feet. Her jugular venous pressure was raised. The examination of chest revealed cardiomegaly and a gallop. There was no murmur. Rales were present at both bases. The liver was enlarged 5 cm. below the costal margin and was tender. Rest of the examination was unremarkable.

Investigations: CBC, ESR, fasting blood sugar, urea, electrolytes, serum proteins, SGPT, SGOT, LDH and urine were normal. X-Ray of the chest showed cardiomegaly with basal congestion. The electrocardiogram showed sinus tachycardia, left axis deviation and left ventricular enlargement. The vector cardiogram showed left axis deviation and left ventricular hypertrophy. The echocardiogram showed enlargement, both of the left ventricular outflow tract and of the left ventricle. The pattern was suggestive of poor left ventricular compliance. The lung scan was normal. Cardiac stress imaging with TC labelled RBCS was also done. It showed changes of congestive cardiomyopathy.

She was treated for the heart failure with bed rest, salt restricted diet, digoxin and diuretics. In about 10 days the heart failure was controlled and she was discharged. She did not come for followup.

Discussion

Peripartum cardiac failure is more common in multipara than in primipara.

The familial occurrence of the disorder has been noted. Its exact aetiology is not known. Various hypothesis have been put forward to explain its occurrence. Toxaemia of pregnancy, malnutrition, and hypertension were considered as possible aetiological factors but none has been proved. Eventhough it may be associated with toxaemia of pregnancy it has not been proved to be an aetiological factor (Demakis, 1971). The association between deficiency of any specific nutritional factor and peripartum cardic failure has not been demonstrated and it has been reported in women who are not malnourished (Ghosh *et al*, 1974). High sodium diet and post partum practice of hot bath and lying on hot bed have been responsible for peripartum cardiac failure in Nigeria (Davidson and Parry,) but this does not explain its occurrence in other races and in other geographical areas. Hypertension is not present in all such cases and when present in few, it is transient and seems unlikely to be a causative factor. The pathological changes in peripartum cardiac failure are non specific and not much different from seen in idiopathic cardiomyopathy (Demakis, 1971).

It seems likely that pregnancy plays a more precise role than nonspecific precipitation of cardiac failure. Probably late stage of pregnancy and/or the metabolic effects of involution in the puerperium may have an adverse effect on so far asymptomatic myocardial disease. Conditioning factors may be necessary in its development and these could be metabolic, immunologic, infective or hypertensive (Goodwin, 1976).

The clinical features the radiological and electrocardiographic changes in peripartum cardiac failure are well described (Davidson and Parry, 1978). The diagnosis of peripartum cardiac failure can be made with certainty when there is: (1) Development of heart failure in the last month of pregnancy or the first five post partum months. (2) Absence of determinable aetiology for congestive failure. (3) Absence of demonstrable heart disease prior to last month of pregnancy (Demakis *et al*, 1971).

In patients of peripartum cardiac failure, the factors considered of bad prognosis are: (1) age more than 30 years, (2) more than fifth para, (3) presence of hypertension or arrythmia, (4) failure of the heart size to return to normal after the failure is controlled (Davidson and Parry, 1971). In about one fifth of women having peripartum cardiac failure there may be recurrence of the failure or of hypertension occur. Relapse usually occurs during the following pregnancy, however, it may occur without pregnancy. Prolonged bed rest is very essential and is strongly recommended (Burch *et al*, 1971). Digoxin should be given to control the heart failure and should be continued in maintainance dose to reduce the risk of relapse. If residual cardiomegaly persists, it is advisable to avoid further pregnancy since that may produce increase myocardial damage. If the peripartum heart returns to normal size after the failure is controlled subsequent pregnancy may sometimes be well tolerated.

Summary

A case of peripartum cardiac failure is reported and discussed.

References

1. Burch, G. E., MacDonald, D. D. and Walsh, J. J.: Ame. Heart J. 81: 187, 1971.
2. Davidson, M. and Mc.Parry E. H. O.: Quart. J. Med. 47: 431, 1978.

3. Demakis, J. G., Shahabuddin, S. and Rahimtoola, M. D.: Circulation 44: 964, 1971.
4. Editorial—Peripartum Cardiomyopathy. Brit. Med. J. 1: 303, 1976.
5. Gosh, J. C., Neelkanthan, M. K. and Chhetri, M. K.: Indian Heart, J. 26: 212, 1974.
6. Goodwin, J. F.: In "The Heart". Edi. J. W. Hurst. McGraw Hill Co., New York, Fourth Ed. 1978 p. 1556.
7. Stuart, K. L.: J. of Med. 37: 63, 1968.
8. Talwalkar, P. G., Narulla, D. V., Mistry, C. J. and Wagholikar, U. L.: Jr. Ass. Phy. Ind. 26: 793, 1978.