PREGNANCY IN PATIENT WITH CIRRHOSIS OF LIVER

(A Case Report)

by

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Pregnancy is considered as an uncommon event in a woman with cirrhosis of the liver, since majority of the patients with this type of disorder, in general, belong to an age group when pregnancy is rather unlikely (Barnes, 1974; Sherlock, 1974; Niven *et al*, 1971). It has also been pointed out by Moore and Hughes (1960) that women with cirrhosis of liver suffer from infertility due to anovulatory menstrual cycles resulting from their inability to metabolize oestrogens in the usual way.

The two varieties of nodular cirrhosis with a recognised female preponderance are active chronic hepatitis and primary biliary cirrhosis (Niven *et al*, 1971). Whelton and Sherlock (1968) reviewed 54 pregnancies amongst 47 women with cirrhosis in the literature and also reported 16 pregnancies amongst 13 patients of their own. In view of rarity, their study appeared interesting.

A case of pregnancy associated with cirrhosis of liver is presented herewith.

Case Report

Mrs. N. R. S. aged 35 years, P 4 + 0 was admitted to Eden Hospital on 1-12-77 at 22-50 hours. She was referred from Chittaranjan Seva Sadan, Calcutta, as a case of acute respiratory distress in cirrhosis of liver with ascites and oedema associated with 30 weeks' pregnancy.

History of Present Illness

The patient stated that she did not undergo any antenatal check-up during her present pregnancy till she noticed swelling of her ankle about a month ago while carrying pregnancy of 26 weeks' duration. She was admitted to Mathurapur Primary Health Centre, 24-Parganas, where she was diagnosed as a case of severe anaemia with hydramnios and was treated as such. The treatment continued for about a month without much improvement. She was then referred to Chittaranjan Seva Sadan on 27-11-77, where she was diagnosed as a case of pregnancy with ascites, oedema and anaemia associated with cirrhosis of liver and was treated with Injection lasix 40 mg., I.M. daily; Mist Pot Chlor 2 T.S.F. T.D.S.; Inj. liver extract 2 ml. I.M. daily, etc. She developed symptoms and signs of acute respiratory distress on 5th day of her stay there and was referred to this hospital on 1-12-77 due to lack of facility for availing of the expert opinion of a physician in that hospital.

Menstrual History: Menarche-12 years, cycle 3-5 days—L.M.P. 14-4-77, E.D.D. 21-1-78. 28-5

Obstetrical History: She had 4 previous pregnancies out of which consecutive first 3 were uncomplicated and ended at term by normal deliveries at her home with 2 boys and 1 girl who are alive and well. The fourth pregnancy

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Accepted for publication on 31-7-78.

was complicated by swelling of feet and abdomen on and from 30th weeks' of her gestation resulting in a premature delivery at 34th week about 2 years ago. The premature female baby died after a month. She also noticed mild icterus during her early puerperium after last confinement which cleared up gradually without treatment.

Past Medical or Surgical History

The patient stated that she was having an enlarged liver without much discomfort for about last 3-4 years as diagnosed by local practitioner of her village for which she did not have any further investigation and treatment.

Personal History: Housewife in farmer's family, not addicted to any drugs or alcohol. She was having low protein diet.

Condition on Admission

General condition was poor with mild degree of anaemia, and oedema of legs. Her pulse, respiration, blood pressure and body temperature were 100/min., 40/min., 105/70 m.m. of Hg and 37°C respectively. There was functional soft systolic murmur in mitral area of the heart but lungs were clear. Abdominal examination revealed engorgement of superficial anterior abdominal wall veins with flow away from the umbilicus and moderate degree of ascites. The liver was just palpable in the epigastrium. It was firm in feel, with sharp margin. The spleen cou'd not be palpated by dipping method. Height of the fundus of uterus corresponded to 30-32 weeks' size of pregnancy. Presentation was vertex (floating). Foetal heart sounds were regular. Vaginal examination showed no abnormality. The provisional diagnosis of pregnancy with cirrhosis of liver with oedema and ascites was made, in collaboration with physician and management of the case was undertaken accordingly with Inj. lasix 40 mg. I.M. stat followed by tablets Lasix (20 mg.) once daily Mist Pot Chlor 2 T.S.F., T.D.S., Liv. 52, 2 tablets T.D.S., salt and fat free diets, glucose drink, Vit. B complex tablets and Haematinic capsules. Investigations done on the day of admission and subsequently: 2-12-77—Hb 9 gm%, T.C. 6600/CCM, D.C. p-65%, l—30%, m—1%, and eosino—4%. Urine-albumin—trace, pus cells +, epithelial cell + blood sugar 80 mg%, blood urea 39 mg%.

Liver function tests—Total bilirubin—0.7 mg%
Vandenberg—Delayed direct positive.

6-12-77—**Liver function tests**

Total bilirubin—1.4 mg%
Vandenberg—Direct immediate positive.
Total protein—5.9 gm% Alb—3.8 gm% globulin 2.1 gm%.
Alkaline phosphatase 20.4 K.A.Units
S.G.P.T. 60 I.U./L
S.G.O.T. 100 I.U./L

Biochemistry of Ascitic Fluid

Sugar 35 mgm%
Chloride 580 mg%
Total protein 1 gm%

Microscopic examination of ascitic fluid showed plenty of epithelial cells and inflammatory cells (Both poly and lymphocytes).
Malignant cells—not found.

Urine: Albumin-trace, sugar and bile salts—positive, bile pigment—nil, clotting time—10 min. 12 seconds, bleeding time—4 min. 38 secs.

Progress in the Hospital: For the first 2 days after admission to this hospital, the condition of the patient remained unchanged. She suffered from abdominal pain and dyspnoea on the 3rd day of her admission (4-12-77) which did not subside inspite of treatment with propped up position, O_2 inhalation and inj. Baralgan 2 c.c. I/M in addition to her previous treatment. The severity of the symptoms aggravated. The patient became drowsy (pre coma) 24 hours after the onset of pain (5-12-77), when I/V drip with 10% dextrose solution followed by Ringer's solution and monosodium glutamate solution along with cap Neomycin (250 mg.) 4 caps 4 hourly, Inj. B Complex, Inj. Vit K, Inj crystalline penicillin 5 lacs I/M. B.D., bowel wash, etc. were given according to the advice of the physician. The full picture of coma developed 8 hours after the onset of pre-coma with severe dyspnoea when we were forced to perform paracentesis and slow drainage of ascitic fluid (2 litres) during 12 hours to give relief from respiratory distress as suggested by physician knowing fully well that drainage of ascitic fluid might deteriorate the condition of coma in cirrhosis of liver. There was oliguria with the onset of hepatic coma. The patient delivered spontaneously a premature stillborn female foetus 14 hours after the onset of coma. The third stage was uncomplicated. The foetus weighed 0.9 kg.

Her condition gradually deteriorated. Investigations such as liver function tests, urine examination on 6-12-77 as shown above, also indicated hepatic decompensation. Ultimately the patient died on 8-12-77 at 16.40 hours.

Needle biopsy of liver was taken immediately after death to confirm the diagnosis, which showed structure of liver tissue disrupted by irregular fibrous bands, liver cells showing fatty change (Fig. 1).

Diagnosis-Cirrhosis of liver.

Discussion

A case of pregnancy in a patient with cirrhosis of liver is presented. Although the patient did not give any definite history suggestive of the etiology of cirrhosis, yet history of liver enlargement and Jaundice following previous childbirth, clinical findings, laboratory investigations and post mortem liver biopsy confirmed the diagnosis. Probably the patient had an attack of acute infective hepatitis in a pre-existing liver disorder during or after her 4th pregnancy and the process of cirrhosis started since then. The spleen could not be palpated in this case during the 5th pregnancy. Probably the portal hypertension was not so marked as to give rise to palpable spleen. Other signs of severe portal hypertension such as haematemesis and malaena were also absent. Maternal and foetal outcome was fatal in the present case. However, successful results with healthy babies and live mothers have been reported (Whelton and Sherlock, 1968). In a pregnancy with cirrhosis of liver, the following points should be emphasised.

Young patients with active chronic hepatitis may conceive during relatively inactive phase of the disease (Sherlock, 1974). Whelton and Sherlock (1968) reported 8 such cases with pregnancy with ages varying between 28 to 39 years. The age of the present case was within these limits.

Fluid retention due to hypoalbuminaema of cirrhosis may appear or aggravate during the course of pregnancy. (Hasseltine, 1930; Abrams, 1957; Shattuck and Spellancy, 1965). In this case, the plasma protein level was rather lower though above the critical level for oedema. This added with anaemia and anoxia of the capillaries may explain the fluid retention in the present case.

Deterioration of liver function is said to be common during pregnancy. Transient bouts of intra-hepatic obstructive jaundice may occur (Barnes, 1974; Niven *et al*, 1971). Bleeding from oesophageal varices is uncommon during pregnancy and changes in portal pressure during second stage of labour do not predispose to bleeding (Whelton and Sherlock, 1968). There was no abnormal bleeding in the case under discussion. There may be mild toxaemia of pregnancy in these cases (Whelton and Sherlock, 1968; Barnes, 1974; Niven *et al*, 1971). Therapeutic abortions are not justified on the ground of liver disease (Whelton and Sherlock, 1968).

The incidences of thrombotic complications in pregnancy in cirrhotic patients are surprisingly few inspite of high level of circulating oestrogen (Niven *et al*, 1971).

Supervention of fetal hepatic coma during labour (Kleckner, 1960) and after parturition (Scaglone, 1923; Saave, 1954; Dehalleux *et al*, 1965; Whelton and Sherlock, 1968) is reported, but more usually the liver is unaffected or recovered postpartum (Whelton and Sherlock, 1968).

Pregnancy as such does not affect the management of liver disease (Niven *et al*, 1971). Folic acid should be supplemented during pregnancy. Steroid should be continued if they are already on it inspite of the risk of congenital deformity of the

foetus. Immuno-suppressive drugs such as 6-mercaptopurine which interferes with protein synthesis should be avoided during pregnancy (Whelton and Sherlock, 1968).

Vaginal delivery is usually preferred as these patients tolerate laparotomy poorly but caesarean section may have to be considered in hepatic coma to save foetuses (Whelton and Sherlock, 1968) when they are otherwise viable.

Pregnancy after successful portacaval shunt operation with compensated liver function does not carry increased risk for mother and foetus (Niven *et al*, 1971).

Whelton and Sherlock (1968) stated that most of the patients with cirrhosis of liver died of liver failure and not of any complications due to pregnancy—as was also our erperience in this case.

Factors which precipitate the deterioration of liver function should be avoided whenever possible e.g. haemorrhage, especially from genital tract, hypotension, sepsis, constipation, general anaesthesia and drugs such as morphine, barbiturates and tetracyclines (Niven *et al*, 1971).

Summary and Conclusions

A case of pregnancy in cirrhosis of liver has been presented. Diagnosis posed a problem because the condition was not detected earlier. Both maternal and foetal outcome was fatal though survival of both mother and foetus are not unlikely as reported in the literature.

Had the patient been under proper medication in the interval between 4th and 5th pregnancy or in the earlier weeks of 5th pregnancy, the progress of the liver disease migth have been checked. Prevention of 5th prgnancy might have at least prolonged her life.

Acknowledgement

The authors are grateful to the Principal-Superintendent and Head of the Dept. of G & O, Medical College, Calcutta for allowing to publish this case. They are also indebted to Dr. B. R. Sengupta, M.D. (Cal.), M.R.C.P. (Edin.), Dept. of Medicine for his valuable advice in the course of management of the case.

References

1. Abrams, F. R.: Obstet. & Gynec. 10: 451, 1957.
2. Barnes, C. G.: Medical disorders in Obstetric Practice, Fourth Edition, Blackwell Scientific Publication, p. 172, 1974.
3. Dehalleux, J. M., Masson, J. C. L., Burger, J. H., Dryfus. J. Gynec. & Obstet. 64: 651, 1965.
4. Hesseltine, H. C.: Am. J. Obstet. & Gynec. 20: 77, 1930.
5. Kleckner, M. S. (1960) Quoted by Reference 12.
6. Moore, R. M. and Hughes, P. K.: Obstet. & Gynec. 15: 753, 1960.
7. Niven, P., Williams, D. and Zeegen, R.: Am. J. Obstet. & Gynec. 110: 1100, 1971.
8. Saave, J. J., (1954) Quoted by Reference 12.
9. Scaglione, S. (1923) Quoted by Reference 12.
10. Shattuck, C. A. and Spellancy, W. N. 1965—Quoted by Reference 12.
11. Sherlock, S.: Diseases of the liver and Biliary system, 5th Edition, Blackwell Scientific Publications, P. 576, 1974.
12. Whelton, M. J. and Sherlock, S.: Lancet 2: 995, 1968.

See Figs. on Art Paper VI