PORTAL HYPERTENSION IN PREGNANCY*

by

S. DAS GUPTA,** M.B., D.A., F.R.C.S., M.R.C.O.G.

Pregnancy with portal hypertension is very rare. Gordon and Johnston in 1963 found only 24 cases reported in the English literature and added five more cases of their own. Table I shows the number of cases recorded.

In Tata Main Hospital, Jamshedpur, two such patients were admitted in 1967.

Case 1

Mrs. B., aged 30 years, primigravida, was admitted in the gynaecological ward on 29th June 1967, with a history of two and half months' amenorrhoea. She had been admitted in the Surgical Ward about 15 months before admission as a case of hepatosplenomegaly with portal hypertension. Exploratory laparotomy, done by Dr. K. P. Misra, Surgeon, Tata Main Hospital, showed cirrhosis of the liver. The portal vein was thrombosed and the portal pressure was 400 m.m. of citrate solution. The patient had one attack of haematemesis before the operation. Her menstrual cycles were irregular, 4-5 days/1-2 months. On examination, her blood pressure was 130/99 m.m. of Hg. The heart was clinically normal. The spleen was enlarged, 6 fingers below the costal margin and the liver, 2 fingers below the costal margin. Pelvic examination confirmed 10 weeks' pregnancy.

Investigations regarding the liver functions and haematological status were undertaken as shown in table II and table III. A barium swallow revealed the presence of oesophageal varices, as shown in figure I. Urine examination did not reveal any abnormality.

TABLE I

Name of author	No. of patients	No. of pregnancies
Abrama (1957)	15	17
Nabriski (1958)	2	3
Adno (1957)	1	1
Ohio. St. Med. J. (1958)	1	1
Moore & Hughes (1960)	3	4
Fish & Mills (1961)	1	1
O'Leary & Bepko (1962)	1	1
Gordan (1963)	5	5

As the liver function tests were quite satisfactory, the pregnancy was allowed to continue under strict antenatal supervision. The patient was anaemic on admission and was treated by blood transfusion and oral iron therapy as the marrow showed normoblastic erythropoesis.

The liver function tests and the haematological investigations were repeated at about 34 weeks of pregnancy, as shown in tables IV and V. There was marked thrombocytopenia in the third trimester.

Based on a paper read by Dr. S. Gadh and Dr. S. Dasgupta at the Annual Conference of Bihar Obstetric and Gynaecological Society at Jamshedpur in February 1968.

**Prof. & Head, Dept. of Obst. & Gynec. Mahatma Gandhi Memorial Medical College and Gynaecologist, Tata Main Hospital, Jamshedpur.*

Received for publication on 16-12-1969.

TABLE II

Liver function tests during first trimester of case 1 (Mrs. B)

1.	S.G.O.T.	—	62 Units
2.	S.G.P.T.	—	14 Units
3.	Total protein	—	4.6 gm. %
	albumin	—	3.1 gm. %
	globulin	—	1.5 gm. %
4.	Van den Bergh	—	Negative
5.	Indirect bilirubin	—	0.25 mgm. %
6.	Thymol flocculation	—	1 plus
7.	Cephalin cholesterol	—	1 plus
8.	Prothrombin time—Test	—	18 Secs.
	—Control	—	13 Secs.

TABLE III

Haematological findings during first trimester of case 1 (Mrs. B)

1.	Haemoglobin	—	6 Gm. %
2.	Total white count	—	6,400/cu.m.m
	Polymorphs	—	65 %
	Lymphocytes	—	30 %
	Monocytes	—	3 %
	Eosinophils	—	2 %
3.	Peripheral smear	—	Normochromic red cells with slight apparent microcytosis.
4.	Bone marrow	—	Normoblastic erythropoesis granulopoesis—normal.
5.	Bleeding time	—	1 min. 30 secs.
6.	Clotting time	—	3 min.
7.	Platelet count	—	1.41 lacs/cu.m.m.

TABLE IV

Liver function tests during third trimester of case 1 (Mrs. B)

1.	S.G.O.T.	—	27 units
2.	S.G.P.T.	—	13 units
3.	Total protein	—	5.9 gm. %
	albumin	—	5.3 gm. %
	globulin	—	2.6 mg. %
4.	Van den Bergh	—	Negative
5.	Indirect bilirubin	—	0.25 mg. %
6.	Thymol flocculation	—	1 plus
7.	Cephalin cholesterol	—	2 plus
8.	Prothrombin time—Test	—	16 secs.
	—Control	—	14 secs.

TABLE V

Haematological tests during third thrimester of case 1 (Mrs. B)

1.	Haemoglobin	—	12 Gm. %
2.	W.B.C.	—	8,200/cu.mm.
	Polymorphs	—	72%
	Lymphocytes	—	22%
	Monocytes	—	3 %
	Eosinophils	—	3 %
3.	Peripheral smear	—	Normochromic red cells with slight apparent microcytosis.
4.	Bone marrow	—	Not done
5.	Bleeding time	—	1 min. 45 secs.
6.	Clotting time	—	4 min. 30 secs.
7.	Platelet count	—	60,000/cu.m.m.

An elective lower uterine segment caesarean section was done on 30th December 1967, and a female baby weighing 5 lbs. 10 oz in a healthy condition was born. The post-operative period was uneventful and her haemoglobin at the time of discharge was 11 Gm.% and the platelet count was 50,000/cu.m.m.

Case 2

Mrs. A. B., aged 25 years, primigravida was admitted in the Maternity ward on 20th January 1968, with six months' amenorrhoea. The previous record of the patient revealed that she had been admitted in the Medical Ward as a case of hepato-splenomegaly. Investigations indicated portal hypertension as the splenic pressure was more than 200 millimetres of water. She used to suffer from severe menorrhagia with anaemia which required blood transfusion on two occasions. An interesting feature of the menorrhagia was that soon after a blood tranfusion, she used to have normal periods for a few months followed by progressively increasing menorrhagia again. On examination at the time of admission, the heart was clinically normal, blood pressure was 130/90 m.m. of Hg., the spleen was palpable, three fingers below the level of the costal margin. The height of the uterus corresponded to the period of amenorrhoea and the foetal heart sounds were heard. A barium swallow did not reveal any abnormality. Liver function tests, as shown in table VII, were normal, and Coombs' test was negative. Bleeding and coagulation time were normal; but the platelet count was markedly diminished (Table VI). Prednisolone, 30 mgm. per day, was started and after 12 days the platelet count came up to 9,000/c.m.m. On 22nd February 1968, she had a premature spontaneous rupture of membranes. Her blood pressure was 140/90 m.m. of Hg and platelet count was 88,000/c.m.m. On 18th March,

TABLE VI

Haematological investigations during third trimester of case 2 (Mrs. AB)

1.	Haemoglobin	—	10 gm. %
2.	R.B.C.	—	3.9 million
3.	W.B.C.	—	4,300/cu.m.m.
	Polymorphs	—	70%
	Lymphocytes	—	25 %
	Monocytes	—	2 %
	Eosinophils	—	3 %
4.	Peripneral smear	—	Microcytic hypochromic with marked leucopenia, but no abnormal white blood cells seen.
5.	Fragility test	—	Normal
6.	Coombs' test	—	Negative
7.	Bleeding time	—	2 min. 5 secs.
8.	Clotting time	—	4 min. 40 secs.
9.	Platelet count	—	5,000/cu.m.m.

TABLE VII

Liver Function tests during third trimester of case 2 (Mrs. A.B.)

1.	S.G.O.T.	—	28 Units
2.	S.G.P.T.	—	7 Units
3.	Van den Berg	—	Positive
4.	Thymol flocculation Cephalin flocculation }	—	Four plus
5.	Indirect bilirubin	—	1.2 mgm. %
6.	Serum protein	—	6 gm. %
	Albumin	—	3.9 gm. %
	Globulin	—	2.1 gm. %
7.	Alkalin phosphatase	—	14.6 K.A. Units
8.	Prothrombin time—Test	—	13 Secs.
	—Control	—	12 Secs.

she started labour pains. Intravenous hydrocortisone drip 100 mgm. in one pint of 5% glucose solution was started as she was on prednisolone therapy for about 58 days. She had an easy vaginal delivery and a healthy female baby weighing 7 lbs. was born. The puerperium was normal and she was discharged on the 7th day when the platelet count was 90,000/cu.m.m. She was advised to continue prednisolone.

Discussion

The two cases reported here had portal hypertension proved in the first case by the occurrence of haematemesis, laparotomy findings and barium swallow and in the second case by the splenic pressure of more than 200 m.m. of water with hepatosplenomegaly. The first case belonged to the extra-hepatic type as the liver during laparotomy was found to be normal. In the second case it is presumed to be of intra-hepatic type although a liver biopsy which might have established the pathology was not done.

Portal hypertension of the extra-hepatic variety is usually associated with normal liver function and hence does not pose a big problem regarding the risk of hepatic failure during pregnancy. Liver biopsy in a normal pregnant patient does not show any abnormality (Schiff, 1956). Alkaline phosphatase is only slightly increased near term (Bodansky, 1939). There is slight retention of Bromsulphthalein in late pregnancy as reported by Christhilf and Bonsnes in 1951. Hence, it is only to be expected that pregnancy in patients with extra-hepatic type of portal hypertension is not associated with the risk of liver failure. The risk of haemorrhage from oesophageal varices increases during pregnancy, specially during the second stage of labour. Taylor, in 1954, showed that coughing could increase the portal venous pressure to almost three times the normal in cirrhotic and even in normal persons. Probably, the contraction of the diaphragm during labour may lead to alarming haemorrhage from the varices in portal hypertension. Our first case, having shown definite presence of oesophageal varices, was delivered by caesarean section as a prophylactic measure. There are, of course, different opinions regarding the methods of delivery in such cases. Adno (1957) and Gordon and Johnston (1963) recommended that such patients should be allowed to go into normal labour.

If haemorrhage occurs from varices, the usual recommended measures like sedatives, blood transfusion and baloon tamponade using the Sengastaken tube are to be adopted. McBeth (1955) practised injection of oesophageal varices.

The intra-hepatic variety of portal hypertension is not usually associated with pregnancy as these patients have abnormal oestrogen and progesterone metabolism leading to amenorrhoea and infertility. Our second case, Mrs. A. B., used to have severe menorrhagia before pregnancy requiring blood transfusion. This indicates some derangement of sex steroid metabolism. But if pregnancy occurs in such cases the greatest danger is that of hepatic failure. Administration of oestrogen in a patient with cirrhosis increases jaundice, as observed by Bearn *et al* (1956) and hence it is possible that pregnancy which is associated with a rising level of circulating oestrogen may upset the

hepatic function to a dangerous degree. Infective hepatitis during pregnany is likely to be more severe than in the non-pregnant patient as reported by Zondek and Bromberg (1947) and Himsworth (1950). Our patient, Mrs. A. B., was also diagnosed to be a case of intra-hepatic portal hypertension clinically. But the pregnancy proceeded without any serious complication excepting thrombocytopenia. This reduction in platelet count cannot be easily explained but it responded quite satisfactorily to prednisolone therapy. Mrs. A. B. had also an easy vaginal delivery following premature spontaneous rupture of membranes. There was no postpartum haemorrhage even though the platelet count was low Sedatives like morphine and phenothiazine derivatives and diuretics of chlorthiazide group were carefully avoided to prevent hepatic coma. In those patients where liver failure sets in, treatment should be on the usual lines, like protein free diet, administration of glucose, oral neomycin and prevention of hypokalaemia. Sodium exchange resins with purgation and enemata are helpful in preventing the absorption of nitrogenous break-down products. Infection should be carefully avoided.

One constant finding in both our cases was thrombocytopenia. In the first case it was very mild and did not require any specific treatment. In the second case the reduction in platelet count was severe enough to require steroid therapy. Probably some derangement of haemopoetic function due to auto-immune process may have been responsible for the thrombocytopenia.

Summary

1. Two cases of portal hypertension with pregnancy are reported.
2. The problems associated with portal hypertension in pregnancy are discussed.
3. Both the patients had thrombocytopenia but one had very markedly low platelet count for which she was treated by steroids.
4. Caesarean section was done in one of the patients showing oesophageal varices, as a prophylactic measure to avoid haemorrhage during labour.
5. Management of complications like liver failure and bleeding varices is discussed.

Acknowledgement

I wish to thank Dr. P. B. Bharucha, M.D., F.R.C.S., Superintendent, Tata Main Hospital, for permitting me to publish the reports of our cases. We are also grateful to Dr. X. J. Anklesaria, M.D., M.R.C.P., Physician. Tata Main Hospital, for his valuable suggestions regarding the management during pregnancy. I also thank Dr. Mrs. S. Gadh and Dr. (Miss) R. P. Dei, for their assistance in preparing this paper.

References

1. Abrams, F. W.: Obst. and Gynaec. **10**: 451, 1957.
2. Adno, J.: S. A. Med. J. **31**: 1189, 1957.
3. Bearn, A. G., Kunkel, H. G. and Slater, R. J.: Am. J. Med. **21**: 3, 1956.
4. Bodansky, M.: Am. J. Clin. Path. **9**: 36, 1939.
5. Christchilf, S. and Bonsnes, R.: Am. J. Obst. Gynec. **59**: 1100, 1951.

6. Fish, G. D. and Mills, W.: Illinois. Med. J. **119:** 20, 1961.
7. Gordeon, A. G. and Johnston, G. W.: J. Obst. & Gynec. Brit. Cwlth. **70:** 1056, 1963.
8. Mc Beth, R.: Brit. Med. J. **2:** 877, 1955.
9. Moore, R. M. and Hughes, P. K.: Obst. & Gynec. **15:** 753, 1960.
10. Nabrisky, S., Zeloo, F. D., Fleishman, P. and Lewitus, Z.: J. Obst. & Gynec. Brit. Emp. **65:** 462, 1958.
11. Ohio State Medical Journal: **54:** 917, 1958.
12. O'Leary, J. A. and Bepko, F. J.: Obst. & Gynec. **20:** 243, 1962.
13. Schiff, L.: Diseases of Liver, Philadelphia, 1956, Saunders.
14. Taylor, F. W.: Ann. Surg. **140:** 652, 1954.
15. Zondek, B. and Bromberg, Y. M.: J. Mt. Sinai Hosp. **14:** 222, 1947.

See Fig. on Art Paper V