INFECTIVE HEPATITIS IN PREGNANCY—THE ROLE OF EXCHANGE TRANSFUSION IN HEPATIC FAILURE

(Preliminary report)

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SUMMARY

Hepato cellular Jaundice in pregnancy is a fatal combination. The problem is that a patient who comes with Jaundice in pregnancy especially in the last trimester develops either an acute hepatic failure or a severe recovery in rather unusual. The incidene of Jaundice complicating pregnancy in our hospital varies from 1/275 to 1/375 total birth. The mortality as a result of Jaundice is 1/20. The problem of hepatitis in pregnany still continue and hepatic failure that ensures in some cases has a bad prognosis but recovery can occur provided there is intensive care and adequate hepatic support. Here an analysis of 5 cases of hepatitis complicating pregnancy is being done. Where an exchange transfusion was given in those with acute hepato cellular failure, where the prognosis is otherwise poor. It has been found that, in those with minimal liver cell damage, only supportive measure are required. In those with measure liver cell necrosis, no treatment is found to ze effective. In the intermediate group, gives adequate time and proper supportive therapy, liver cells might regenerate and start functioning satisfacory.

Hepatocellular jaundice in pregnancy is a grave and often fatal combination. The problem is that a patient who comes with jaundice complicating pregnancy especially in the last trimester develops either an acute hepatic failure or a severe postpartum haemorrhage from which recovery is rather unusual. The incidence of jaundice complicating pregnancy in our hospital varies from 1/275 to 1/375 total births. The mortality as a result of jaundice was 1/20 which gives a mortality rate of 500/10,000. The total maternal mortality rate in our hospital was 50/10,000. Jaundice in pregnancy was responsible for 10% of the maternal mortality.

The problem of hepatitis in pregnancy still continues and hepatic failure that ensues in some cases poses problems as encephalopathy due to hepatocellular failure and bleeding tendency due to disturbance of

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clotting mechanism. In both conditions the prognosis is worse but recovery can occur provided there is intensive care and adequate hepatic support.

An analysis of 5 cases of hepatitis complicating pregnancy who developed as acute hepatocellular failure with coma and who were given an exchange transfusion is given.

There is nothing new in the exchange transfusion, as this method of treatment has already been done since 1966. Although favourable results were obtained initially, over the succeeding years, results have not been satisfactory. The availability of adequate quantities of suitable blood is another problem in our situation. We initiated this procedure as the mortality in these cases is otherwise frighteningly high. By doing an exchange, the non-dialysable substances are removed to some extent and nutritional substances provided that might help in the regeneration of liver.

Technique of exchange transfusion

1. Eight bottles of fresh whole blood should be kept ready before starting the exchange.

2. All the essential drugs necessary for resuscitation of the patient should be kept ready as complications like cardiac arrest, respiratory arrest, hypotension or pulmonary oedema may occur during the procedure.

3. The general condition of the patient is assessed before starting an exchange. The pulse rate, blood pressure, respiratory rate and an assessment of the neurological state is made. These are charted on a separate sheet of paper before starting the exchange. These parameters should be recorded every 15 minutes during the procedure and also a record of urine output is made.

4. The most important thing is the maintenance of an adequate venous lines for infusion as well as for withdrawal of blood. The saphenous vein is cannulated and blood is drawn from the external iliac vein. Subclavian vein is used for infusion after cannulation through antecubital vein. After the cut down, the cannula used is the polythene infant feeding tube No. 12 size which will reduce the tendency for clotting and ensures an easy withdrawal of blood.

5. The input and output should be balanced except in cases with internal haemorrhage, where a positive balance should be maintained.

6. Inj. Calcium gluconate 10 ml given intravenously for each bottle of blood transfused and oftener if necessary (to correct the hypocalcemia which occurs in citrate intoxication).

Case analysis :

There was something in common for all the cases. Majority of the patients were primigravida, with associated toxaemia of pregnancy, high blood urea level and a shrunken liver. Most of them were admitted antepartum in a fully conscious state, delivered and went in hepatic coma.

For the purpose of prognosis *clinical grading of the consciousness* is done into Grade I to Grade V (Sheela Sherlock).

TABLE I
Clinical Grading

Grade I	Confused
Grade II	Drowsy
Grade III	Stuporous, but obeys simple commands
Grade IV	Come but responds to painful stimuli
Grade V	Deep coma with no response to painful stimuli and no involuntary movements

Patient No. 1

The patient was admitted as a case of hepatitis and toxaemia complicating pregnancy. The patient was fully conscious at the time of admission. Serum bilirubin was 16 mg% and blood urea 75 mg%. On the

2nd day of admission, the condition of the patient was deteriorating gradually and went into Grade IV coma with evidence of gastro-intestinal bleeding and a rising blood urea level. The general condition became worse after the delivery of a dead foetus. At that time, the bilirubin was 20 mg% and blood urea 110 mg%. Exchange transfusion was tried with 6 bottles of fresh whole blood. Twelve hours after the exchange, the patient was only drowsy with a bilirubin of 16 mg% and the blood urea was high. On the third day of exchange the patient again went into Grade IV coma with a serum bilirubin of 18 mg% and blood urea of 120 mg%. Second exchange was given with 6 bottles of fresh blood. Twelve hours after the second exchange, the change was remarkable. The bilirubin came down to 10 mg% and blood urea to 69mg.% Seven days after the exchange, the bilirubin was only 4 mg.%. The patient was discharged well, 21 days after admission.

Patient II

In elderly primigravida was admitted with toxaemia of pregnancy and hepatitis. At the time of admission, the billirubin was 16 mg% and the patient was in a confused state. She was managed conservatively and delivered on the 3rd day of admission. The patient's condition deteriorated after delivery with billirubin of 17.2 mg% and urea of 100 mg%. An exchange transfusion was given with 8 bottles of blood. Within 12 hours of transfusion, the patient's condition improved with serum bilirubin of 15 mg% and Grade I coma. Although the patient's condition improved, the blood urea increased upto 81 mg% with oliguria. This was managed conservatively. Although the patient was conscious, the bilirubin level was 20 mg%. This shows that bilirubin level alone is not the one which determines the neurological status of the patient. On the 9th day after exchange, the urea came down to 45 mg% and after 19th day, bilirubin was 7.2 mg% and urea levels came down to 20 mg%. Twenty days after exchange urea and bilirubin levels were within normal limits. The patient was discharged 35 days after admission.

Patient III

A second gravida, with 1st abortion was admitted as a case of jaundice complicating pregnancy. The patient was stuporous at the time of admission and the condition worsened after delivery with evidence of gastro-intestinal haemorrhage. Exchange transfusion was tried but she developed respiratory distress after the 1st bottle of blood and expired.

Patient IV

The patient was admitted from outside after delivery as a referred case of P.P.H. She was in Grade IV coma with serum bilirubin levels of 20 mg%. She was given 2 exchange transfusions one on the day of admission and another 48 hours later. She was discharged 60 days after admission.

Patient V.

A primigravida who came walking into the casualty and delivered on the same day of admission. At the time of admission, the bilirubin was 30 mg% and blood urea levels of 80 mg%. After delivery, the patient went into grade IV coma. Exchange transfusion was given, but the improvement was not that marked as seen in other patients. The bilirubin levels after exchange was 29.2 mg%. The other problem in the patient was a persistent hypertension with decerebrate rigidity and a persistently high blood urea level. Because of the persisting grade IV coma, second exchange was given 48 hours after the 1st exchange. At that time the bilirubin level was 28 mg%. But the general condition remained the same. The patient was going in for a renal failure

with rising blood urea level, 48 hours after the 2nd exchange, the bilirubin level was 22 mg% with a blood urea of 160 mg%. Although we were planning to give a third exchange the patient expired. The probable cause of death was chronic renal failure and hypertension.

The purpose of this paper is to analyse the usefulness of exchange transfusion in the management of hepatic coma. No therapy currently in use for hepatic coma is of no value in salvaging life.

The recovery of patients from hepatocellular failure depends upon the amount of liver cell necrosis and the ability of liver cells to regenerate. In patients with minimal liver cell damage, only supportive measures are necessary and they seldom go into hepatic coma. In those with massive cell necrosis, with impairment of cerebral and renal function, the prognosis is poor. In the intermediate group allowing sufficient time for the liver to regain function satisfactorily exchange transfusion might be helpful.

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