Successful Treatment of Hepatic Coma, Complicated by Renal Failure, with Exchange Transfusion and Peritoneal Dialysis

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SUMMARY

A successful outcome is reported in a case of hepatic coma, complicated by renal failure, with combined treatment of exchange transfusion and peritoneal dialysis.


The first successful case of liver coma treated with exchange transfusion was reported by Lee and Tink in 1958. In a review of the literature up to June 1970, Riviera et al. collected 97 cases treated by this method, with a survival rate of 34% compared with a survival rate of 25% in 263 cases treated conventionally. No statistical significance can be assigned to such figures from different authors. Davis et al. have suggested that recovery from fulminant hepatitis is unpredictable and that superiority of exchange transfusion over conventional treatment can only be proved by a controlled trial. Such a trial is not available at present.

Both haemodialysis and peritoneal dialysis have in the past been used in the treatment of hepatic coma. In 5 patients treated with haemodialysis by Kiley et al. there was good clearance of blood ammonia, but none of the patients recovered. In one case with hepatic and renal failure there was excellent temporary improvement, but the patient failed to recover.

In 1963 Nienhuis et al. treated a patient with hepatic coma successfully with peritoneal dialysis. This patient recovered after 39 hours of peritoneal dialysis with 44 litres of fluid.

Peritoneal dialysis combined with exchange transfusion was first reported by Krebs and Flynn in 1967. In their report the peritoneal dialysis was used in a prophylactic manner to counteract the possible adverse effects of a massive transfusion of citrated blood on the kidneys.

Because of the extremely grave prognosis of hepatic coma accompanied by renal failure, we wish to report successful management of such a case with exchange transfusion and very prolonged peritoneal dialysis.

CASE REPORT

A 22-year-old Indian male was admitted to hospital in coma. One week earlier he had become ill with anorexia and vomiting. This was soon followed by jaundice and 2 days later by coma. There was no drug history or contact with hepatitis.

On transfer to the Coronation Hospital he was in deep coma, holding himself in a decerebrate posture with occasional clonic contractions resembling convulsions. He was also anuric.

Blood biochemistry at the time of admission showed the following: bilirubin 64.8 mg/100 ml, direct 36.3 mg/100 ml, indirect 28.5 mg; alkaline phosphatase 20.8 SC units; SGOT 1 280 units; SGPT 980 units; LDH 9 800 units; prothrombin index 44%; blood urea 210 g/100 ml; HAA—negative.

The following conservative regimen was instituted:
1. Bowel sterilization with neomycin 500 mg every 6 hours.
2. Solucortef 100 mg intravenously every 4 hours.
3. Ampicillin 250 mg intramuscularly every 6 hours.
4. 5% dextrose water 500 ml rapidly intravenously.
5. 10% mannitol solution 200 ml intravenously.
6. Furosemide 120 mg intravenously.

There was no response to conservative treatment after 24 hours, and exchange transfusion combined with continuous peritoneal dialysis was decided upon.

Method

Ten units of fresh ACD blood were transfused via a Scribner shunt placed in the patient’s forearm, while 10 units of blood were drained from the arterial limb of the shunt. The exchange was accomplished within 2 hours. This was repeated daily for 3 days for a total of 29 units of blood. Exchanges were discontinued for 48 hours because of unavailability of fresh blood, and then resumed, the patient receiving another 27 units of blood over 3 days, a total of 56 units of blood in 8 days.

Peritoneal Dialysis

Peritoneal dialysis was performed continuously in the standard way for 18 days, for a total of 678 exchanges.

Clinical and Biochemical Course

The patient regained consciousness on the 10th day and started to produce urine on the 12th day.

On the 20th day he produced 2 melaena stools followed again by a sharp rise in the blood urea to 310 mg/100 ml.
All the clotting factors were normal at this time. No definite cause could be found for the haemorrhage, which stopped spontaneously.

Peritoneal dialysis was instituted again for 148 exchanges.

After this he made a slow, but complete recovery.

At the time of discharge his biochemical investigations showed the following: bilirubin—total 0.1 mg/100 ml; direct 0.0 mg/100 ml; alkaline phosphatase 4.4 SC units; total protein 8.4 g/100 ml; albumin 5.3 g/100 ml; globulin 3.1 g/100 ml; transaminase—normal; prothrombin index 89%: blood urea 17 g/100 ml; creatinine clearance 71 ml/min.

**Histology of the Liver**

There was a round-cell infiltrate of the portal areas. There was no cirrhosis, but only minimal fibrosis. There were signs of intrahepatic cholestasis.

**Follow-up**

Two months after discharge the patient remained well and had gained considerable weight.

**DISCUSSION**

The mortality of patients with liver coma treated along conventional lines is high. When the liver coma is complicated by renal failure, it is formidable.

Although the response of these desperately ill patients is unpredictable, exchange transfusions have increased the survival rates. Haemodialysis and peritoneal dialysis have been used sporadically in the treatment of liver coma. Both these methods are capable of cleansing blood ammonia and bilirubin from the blood of these patients, but clinical results have been disappointing.

The successful outcome in this case of liver coma with renal failure, treated by exchange transfusions and 826 peritoneal dialysis exchanges, raises the question whether this combined method should not always be used in the treatment of liver coma. Peritoneal dialysis is a simple method which is capable of clearing ammonia as well as other breakdown products of nitrogen metabolism. Exchange transfusion with fresh blood achieves the same end and replaces clotting factors which the failing liver does not synthesize. It would thus seem rational to combine these two methods in the treatment of liver coma, regardless of whether or not the condition is complicated by renal failure.

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**REFERENCES**


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**Books Received : Boeke Ontvang**


