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Haemocarboperfusion treatment of neonatal haemolytic jaundice

by

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Haemocarboperfusion was used with success in two cases for the treatment of hyperbilirubinaemia of newborns with haemolytic disease. The method will be useful in the lack of the necessary amount of blood for exchange transfusion and will successfully prevent the CNS damage.

Exchange transfusion with 170 to 200 ml/kg body weight of blood is the generally used procedure in the treatment of severe hyperbilirubinaemia in haemolytic disease of the newborn. In severe cases repeated exchange transfusions may be necessary.

In developing countries where a blood supply has not been organized or is against the laws and religious habits, there is not always enough blood available for exchange transfusion [3, 4]. Blue light therapy or D-penicillamine [6] are advantageous but only in the mild forms of hyperbilirubinaemia.

Since in two cases we had not enough group specific blood available for exchange transfusion, and the serum bilirubin level increased in spite of blue light and D-penicillamine treatment, we decided to apply haemocarboperfusion. According to previous experience [2], this method reduced the serum direct and indirect bilirubin levels in hepatic coma.

Case Reports

Case 1. M.Md.M. was the 11th child of the mother. Birth weight was 3600 g and the baby was admitted with jaundice in the 12th hour after delivery. The baby had 8 living siblings. His general condition was good, the skin was yellow, there were no neurological symptoms. At admission the total serum bilirubin level was 31 mg/dl, the direct bilirubin level was 2.8 mg/dl. Blood group of the mother was B Rh negative, and that of the baby was 0 Rh positive. The direct Coombs test was positive.

Other laboratory findings were, Hb 17.8 g/dl, PCV 56%, WBC 28 900/mm^3, 30 nucleated red blood cells for 100 white blood cells, thrombocyte count 120 000/mm^3. Since 0 Rh negative blood was not available, an exchange transfusion could not be performed. Blue light treatment was started and 250 mg/kg body weight of D-penicillamine was administered together with 5% glucose infusion. The serum bilirubin level increased to 37 mg/dl by the 36th hour after birth and the direct bilirubin level increased to 4.5 mg/dl. There were no disturbances in the acid-base or ion balance, and there were no symptoms of nervous system damage. Hb was reduced to 15.7 g/dl and PCV to 50%. Proteus was

cultivated from the throat and ear swabs.

In the 60th hour of life total serum bilirubin level was 41 mg/dl and the direct bilirubin level increased to 10.7 mg/dl. Hb decreased to 13.0 g/dl, and PCV to 43%, The platelet count was 85 000/mm³.

On the fourth day we received 250 ml 0 Rh negative blood. Since this amount would have sufficed only for the exchange of 67 ml/kg of blood but was enough for the filling of an Absorba 300 haemocarboperfusion cartridge, we continued the treatment by haemocarboperfusion.

Haemocarboperfusion treatment was performed by a Gambro AK-5-HMD unit using infant blood lines. Veno-venous perfusion was carried out by cannulation of the umbilical and saphenous veins. Blood clotting in the cartridge was prevented by the addition of heparin-protamine. Perfusion was continued for 2.5 hours, while PCV, platelet count, blood clotting and bilirubin level were monitored. The baby was placed into an incubator during and for 24 hours after the perfusion. Heart function and body temperature were monitored continuously by a Siemens Sirecast 302 D apparatus.

The perfusion was tolerated well, there were no disturbances in the circulatory, respiratory or blood clotting systems. The total serum bilirubin level decreased significantly to 27.5 mg/dl, but the direct bilirubin level did not change significantly (7 mg/dl). Hb was 10.6 g/dl, and PCV was 32%. There were no changes in acid-base or ion balance. Sodium concentration of the blood leaving the cartridge was 138—139 mEq/l, while the potassium level was 0.4 mEq/l at the beginning and 3.0—3.2 mEq/l at the end of perfusion. There occurred no postperfusion hypokalaemia. ECG was normal. The WBC in the blood leaving the Absorba 300 cartridge showed a decrease during perfusion: in the blood entering the cartridge WBC was 7600/mm³ while in the blood leaving the cartridge it was 3500/mm³. The platelet count did not change during the 2.5 hr perfusion period: it was 45—50 000/mm³.

Twenty-four hours after perfusion Hb was 9.0 g/dl, PCV was 28%, WBC 10 300/mm³ and DLC was normal. Total serum bilirubin level was 20.8 mg/dl and the direct bilirubin level 6.3 mg/dl. Total serum protein was 5.4 g/dl. The ionogram, serum glucose and BUN values were normal. The general condition was good, there were no neurological symptoms and the baby could be fed orally.

Two days later, the serum total bilirubin level was 8.7 mg/dl, while at 12 days it was 5.7 mg/dl. The direct bilirubin level was 2.9 mg/dl and the platelet count 270 000/mm³. The patient was discharged in a good condition and with increasing body weight, without any neurological symptoms.

Case 2. Sz.M.D. was the 6th child of the mother. He was born with 2000 g, and was admitted with deep jaundice on the 4th day following delivery. At admission the baby was crying vigorously, but the reflexes were feeble, and he had diarrhoea.

Total serum bilirubin level was 53 mg/dl and the direct bilirubin level 40 mg/dl. Other laboratory tests revealed Hb 5.8 g/dl, PCV 19% WBC 19 700/mm³, DLC 20 nucleated red blood cells per 100 white blood cells; the platelet count was 163 000/mm³. The patient's blood group was 0 Rh positive, the direct Coombs test positive, Australia antigen negative in the serum of the patient. Total serum protein was 7.1 g/dl, GOT 4 IU/L, GPT 18 IU/l, LDH 1854 IU/l, alkaline phosphatase 322 IU/l, BUN 50 mg/dl, sodium 148 mEq/l, potassium 5.8 mEq/l, serum glucose 50 mg/dl. The blood pH was 7.2, pCO_2 44 mm Hg, pO_2 32 mm Hg, SBE — 7.7 mEq/l, Sat 52%, SBC 17.3 mEq/l.

Since there was no specific blood for exchange transfusion, blue light and D-penicillamine treatment were started in combination with 10% glucose infusion.

Twenty-four hours later the total serum bilirubin level was 50 mg/dl, direct bilirubin was 45 mg/dl, BUN was 42 mg/dl, the serum sodium 138 mEq/l and potassium 2.9 mEq/

l. The earlier detected metabolic acidosis persisted.

By that time we received 250 ml of 0 Rh negative blood. This sufficed for haemocarboperfusion; it was done similarly as in Case 1. Perfusion lasted for 2.5 hours. In the 30th min of recirculation the outflow was disturbed and the heart rate increased to 160—180/min. The disturbance was corrected with 30 ml of 3.5% plasma protein. At the end of the perfusion severe metabolic acidosis was observed. At that time the values in umbilical venous blood were pH 7.09, pCO_2 35 mm Hg, pO_2 49 mm Hg, BE 18.9 mEq/l, Sat. 67.5%, SBC 9.2 mEq/l. The acidosis was assumed to be the consequence of the transient circulatory failure and the persisting enteritis; it was successfully corrected by an infusion of sodium bicarbonate solution.

Total protein content and albumin level were reduced by 2 g/dl in the blood leaving the cartridge as compared with the blood entering the Absorba 300 cartridge. The 46—49 mg/dl BUN value of the blood entering the cartridge was significantly reduced to 2—7 mg/dl in the blood leaving it, and in the serum it was reduced to 21—25 mg/dl by the end of the perfusion.

Five hours after the perfusion the total serum bilirubin level was 25 mg/dl, serum glucose was 60 mg/dl, and the acid-base balance had normalized. The ion balance was normal. Twenty-four hours later a rebound occurred; total serum bilirubin was 31 mg/dl and direct bilirubin was 25 mg/dl. At 48 hours the bilirubin value showed a decreasing tendency: total serum bilirubin was 23 mg/dl, direct bilirubin was 22 mg/dl and the BUN 15 mg/dl. Acid-base and ion balances were normal.

The platelet count was 80 000/mm³ at the beginning of perfusion. During the next 48 hours it was 25—50 000/mm³, then next 4 days it increased to 130 000/mm³. WBC was 3—5000/mm³ during the 48 hours after the perfusion.

The serum bilirubin content decreased slowly after the perfusion. Total bilirubin was 9.0 mg/dl on the third day. The patient became anaemic during this period; Hb fell to 6.4 g/dl, PCV was 20%. We received blood only on the 6th day, when the patient was transfused.

Bacteriological and parasitological examinations were negative. In spite of the antibiotic and dietary treatment the diarrhoea persisted and dehydration and acidosis set in. This was treated by fluid and bicarbonate. Then intravenous feeding was performed for one month and under its effect the enteritis disappeared, only to return after the introduction of oral feedings. It did not respond to antibiotics nor to Lactobacillus treatment.

The patient died with symptoms of intestinal paralysis, peritonitis and sepsis 70 days after admission. Haemocultures were repeatedly negative. A post mortem examination could not be performed being forbidden by Libyan laws and habits.

Discussion

If the amount of blood necessary for exchange transfusion is not available, haemocarboperfusion seems to be an appropriate method for the reduction of the serum bilirubin level. The drawback of the method is that it needs a special equipment and practice. The pump of the Gambro AK-5 apparatus is easily mobilized so that the perfusion can be performed in the baby incubator. Monitoring of the treatment and the occasionally necessary resuscitation need a team with adequate practice in intensive care.

The main danger of the technique are the changes of blood volume occurring when the outflow is inhibited. This is mostly due to clotting of the blood in the perfusion cartridge. In order to avoid this complication, hepa-

rin and protamine sulphate should be added to the system by perfusion pumps.

The action mechanism of haemocarboperfusion has not been yet clarified. Resin and charcoal perfusion of animals in acute experimental hepatic coma caused a 19—25% reduction of the serum bilirubin level. Beside bilirubin, considerable quantities of serum proteins, mostly albumin, adhere to the carbon column of Absorba 300. Active carbon binds in addition carbamide, white blood cells and thrombocytes [1].

A further reduction of the necessary blood volume can be expected from the use of the Absorba 150 haemocarboperfusion cartridge which needs only 140 ml of blood. In neonates the tolerated blood volume reduction is 10% [5], so the minimum blood volume required for the treatment is 130 ml. Further blood is needed for the correction of anaemia after the hyperbilirubinaemia had been decreased. On the basis of our experience, the haemocarboperfusion method by itself may suffice for protecting the nervous system from the damage induced by hyperbilirubinaemia.

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