0749-5161/91/0701-0021/$03.00/0
PEDIATRIC EMERGENCY CARE
Copyright © 1991 by Williams & Wilkins

Vol. 7, No. 1
Printed in U.S.A.

Intrahepatic hematoma: A rare complication of exchange transfusion

REJIN KEBUDI, MD, SERPIL UĞUR, MD, ASUMAN ÇELENK, MD, NEDRET UZEL, MD

A four-day-old male neonate who underwent three exchange transfusions for jaundice owing to ABO incompatibility on three consecutive days is presented. A large intrahepatic hematoma developed as a complication of the third exchange transfusion. The baby fully recovered.

INTRODUCTION

The primary aim of exchange transfusion is to prevent the toxic effects of bilirubin by removing it from the body. The most widely used technique is the one described by Allen and Diamond[1] in 1957, which entails catheterization via the umbilical vein. Although this procedure is a simple one, catheterization of the umbilical vein or artery is not without hazard.[2,3]

We report a neonate who underwent three consecutive exchange transfusions and who developed intrahepatic hematoma as a complication of the third exchange transfusion.

CASE

A four-day-old, white, full-term male baby born at home by a vaginal delivery with the help of a midwife presented to the Pediatric Emergency Unit with jaundice, fever, and poor sucking which had developed the same morning. It was the mother's fifth delivery. Two siblings had died at ages three and five years, from other causes. Membrane rupture time was reported as three hours. No asphyxia was noted at birth. The baby's weight on admission was 4,000 g. Physical examination revealed an icteric male newborn with a fever of 38.5°C. Spontaneous activity was slightly diminished. Heart rate was 160 beats/min, and respiratory rate was 40 breaths/min. The liver was tender and palpable 2 cm below the costal margin. No splenomegaly or abdominal mass was noted. Examination of the heart and lung was unremarkable. Normal newborn reflexes were present, except for poor sucking. Initial laboratory evaluation revealed ABO incompatibility. The mother's blood group was O Rh(+), and the baby's was A Rh(+). The direct and indirect Coombs tests were negative. Hematocrit value was 44%; WBC was 10,000/mm^3 with a differential count of 22% bands, 50% PMNs, and 28% lymphocytes. Normoblasts were present in the peripheral blood smear, spherocytes being less than 16%. Reticulocyte count was 6%. Spinal tap disclosed normal chemistry with no cells. Urine analysis was unremarkable. Antibiotics (penicillin 50,000 U/kg/day and amicasin 15 mg/kg/day) were initiated after obtaining blood, CSF, and urine cultures. Blood urea, glucose, and electrolytes were within normal limits. Initial total bilirubin level was 29.2 mg/dl; indirect bilirubin level was 26.8 mg/dl. The first exchange transfusion with O Rh(+) fresh whole blood (180 ml/kg) was performed with no complications. Subsequent to the transfusion and while under phototherapy, the indirect bilirubin continued to rise and reached a level of 24.5 mg/dl the following day, necessitating a second exchange transfusion. On the third day, the indirect bilirubin level again increased to critical levels, and a third exchange transfusion was initiated. As in the previous two exchange transfusions, the catheterization of the umbilical vein was performed by the discontinuous technique described by Allen and Diamond[1] in 1957, and the place of the catheter was confirmed by measuring the length of the catheter inserted. A French 6 polyethylene catheter (Pharma-Plast A/S DK) with multiple side holes was advanced 10 cm from the skin surface of the umbilical vein. One hundred eighty ml/kg of blood was transfused in aliquots of 10 ml, at a rate of 10 ml/2 min. Toward the end of this third intervention, an obstruction of the catheter, which interfered with the transfusion, was noted. This obstruction could not be overcome when the catheter was gently moved forward and backward. The catheter was pulled out, a new one was inserted, and the exchange transfusion was completed. Toward the end of the transfusion, ecchymoses appeared in the periumbilical area. The ecchymoses progressed rapidly and, within two to three hours extended to the abdominal wall, scrotum, penis, right leg, and right foot. The liver was palpable 5 cm below the costal margin. Abdominal distension and a decrease in hematocrit value were observed. Vital signs remained normal. A significant increase in direct bilirubinemia occurred. Total bilirubin reached a level of 32.9 mg/dl (direct bilirubin 17.4 mg/dl). An upright abdominal x-ray revealed a shift of intestinal gases to the left (Fig. 1). SGOT and SGPT values were slightly elevated. Other routine biochemical values, as well as bleeding and clotting times, PT and PTT were all within normal limits. The platelet count was 120,000/mm^3. Antibiotics were discontinued when CSF, urine, and repeated blood cultures were found to be negative.

After two weeks, while the liver enlargement persisted, the ecchymoses began to disappear, leaving some pigmentation,

From the Department of Pediatrics, the Istanbul University School of Medicine.
Address for reprints: Rejin Kebudi, MD, Salih Tozan Sokak, No. 2/22 Polat Apt, Gayrettepe, Istanbul, Turkey.
We are grateful to Professor Dr. Olcay Neyzi, Director of the Department of Pediatrics, Istanbul University, School of Medicine, for helpful discussions.
Key Words: Exchange transfusion, intrahepatic hematoma

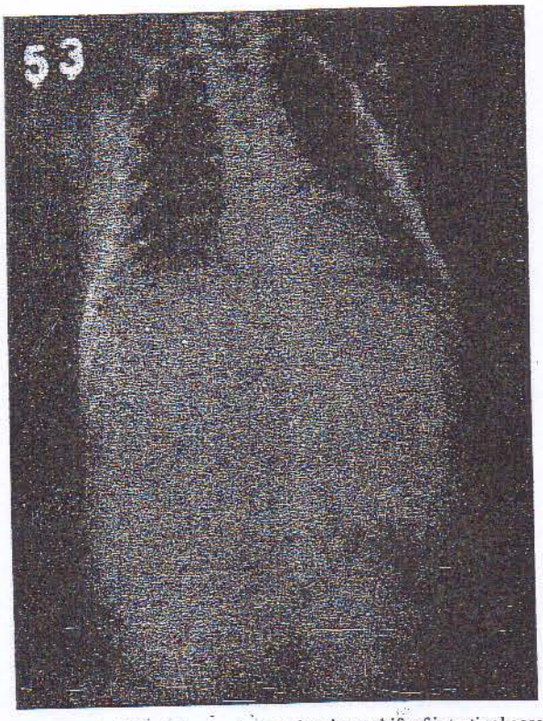


FIG. 1. Upright abdominal x-ray showing a shift of intestinal gases to the lower left quadrant.

and the bilirubinemia decreased to normal. Abdominal ultrasound revealed hypoechogenic areas in both the right and left lobes of the liver, which were interpreted as intrahepatic hematoma (Fig. 2). Slight hypotonia was noted in the right leg, and physiotherapy was initiated. The patient was discharged on the twenty-fourth postnatal day but was followed closely. Three months later the hepatomegaly had disappeared, and ultrasonography findings were normal (Fig. 3). Neuromotor development was normal for age. Hypotonia in the right leg persisted, and EMG revealed partial denervation signs in muscles innervated by the two subbranches of the sciatic nerve. Physiotherapy was continued with some improvement.

DISCUSSION

The mortality caused by exchange transfusion is defined as mortality that occurs within six hours of the exchange transfusion. Postexchange transfusion mortality rate is reported to be less than 1% in most publications, varying between 0.53% and 3.2%.[4–8] The incidence of serious complications owing to exchange transfusion is reported as 4.2% by Keenan et al.[9] Postmortem data indicate that serious complications of umbilical catheterization are encountered at a rate of 1.5 to 7% in newborn autopsies. However, these complications appear as a significant cause of death in less than 2% of cases. The lesions include embolization, thrombosis of the hepatic vein, liver necrosis, aortic thrombi, and infarcts. A serious complication is thrombosis of the renal arteries and renal shutdown, leading to death.[7,8] Hemorrhagic necrosis of the liver has also been observed in asphyxiated newborn infants. In hemorrhage



FIG. 2. Abdominal ultrasound revealing hypoechogenic areas in the liver.



FIG. 3. Abdominal ultrasound showing a normal liver structure.

of the liver of any cause, the hemorrhage may be mild and subcapsular, or abundant hemorrhage into the peritoneal cavity may occur, owing to the rupture of the capsule or to trauma involving hepatic parenchymal tissue. The general state of the baby usually remains unaffected during the progressive subcapsular hepatic hemorrhage unless the capsule ruptures, leading to hemoperitoneum and hypovolemic shock.[2–4]

In our case, we can only make speculations about the etiopathogenesis of the complication we have encountered since the baby survived, and since laparotomy was not attempted, owing to gradual regression of the clinical findings

When obstruction was noted in the course of the transfusion, the catheter was reported to be pushed in and pulled out in an effort to reposition. During the attempts at repositioning, the catheter probably went into the entry point of the vena porta into the liver. The vein rupture which occurred at that point led to hemorrhage into the liver and partly into the abdomen. Part of this hemorrhage was absorbed by the peritoneum and passed to the superficial tissues causing the ecchymoses that were seen. The intraabdominal hemorrhage probably was not abundant enough to cause signs of shock, which were not observed at any time in this baby. The second catheter that was placed was probably in the right place; thus, transfusion could be completed. Meanwhile, a clot formed at the site of the rupture. The degradation of the extravasated blood led to the observed increase in the indirect bilirubin level. The compression of the intrahepatic biliary tract by the intrahepatic hematoma and of the extrahepatic biliary tract by the intraabdominal hematoma was probably the cause of the increases involving both direct and indirect bilirubin fractions. As the hematoma was resorbed, the abdominal distention and hepatomegaly regressed, and the direct bilirubin level progressively decreased. The shift of the intestinal air to the left on the upright abdominal x-ray was also probably caused by an intraabdominal hematoma or by the hepatomegaly secondary to the intrahepatic hematoma. An intraabdominal hematoma was not present in the abdominal ultrasound taken on the eighteenth day, probably owing to the resolution of the hematoma. Hypotonia of the right leg and the EMG findings are thought to be related to a compression of the nerve roots contributing to the sciatic nerve (L_4–S_3) by an intraabdominal hematoma. Intrahepatic hematoma alone would not cause such a finding.

This case is reported because it is a rare example of a surviving baby who has suffered from intrahepatic hematoma caused by umbilical catheterization. During insertion of the catheter, and especially during attempts at repositioning, the position of the catheter must be confirmed, not only by measuring the length of the catheter inserted, but also by x-ray when available and possible. Every effort at repositioning must be very gentle. When too much difficulty is encountered during umbilical venous catheterization, insertion of the catheter via another vessel such as vena saphena may be tried. This example emphasizes once more that, although catheterization for exchange transfusion is a simple maneuver, the process should be applied with great caution, with all possible consideration of complications.

REFERENCES

1. Allen FH Jr, Diamond LK. Erythroblastosis fetalis. Boston: Little, Brown, 1957.
2. Stanley J. Procedures for infant resuscitation. In: Fanaroff AA, Martin JR, eds. Neonatal-perinatal medicine. St. Louis: CV Mosby, 1987;365–369.
3. Driscoll JM, Jr. Care of the newborn—The umbilical vessels. In: Fanaroff AA, Martin JR, eds. Neonatal-perinatal medicine. St Louis: CV Mosby, 1987;442.
4. Scott JM. Iatrogenic lesions in babies following umbilical vein catheterization. Arch Dis Child 1965;40:426.
5. Boggs TR, Westphal MC. Mortality of exchange transfusion. Pediatrics 1960;26:745–755.
6. Kitchen WH. Neonatal mortality in infants receiving an exchange transfusion. Aust Paediatr J 1970;6:30–40.
7. Panagopoulos G, Valars T, Doxiadis SA. Morbidity and mortality related to exchange transfusions. J Pediatr 1969;74:247–254.
8. Weldon VV, Odell GB. Mortality risk of exchange transfusion. Pediatrics 1968;41:797–801.
9. Keenan WJ, Novak KK, Sutherland JM, et al. Morbidity and mortality associated with exchange transfusion. Pediatrics 1985;(suppl):417–421.