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Short Communication

Splenic rupture in neonate with hemolytic anemia

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Splenic rupture is relatively a rare and life threatening situation in the newborn. The classic triad of the disease is shock, anemia, and abdominal distension. Non-traumatic etiologies are seldom and one of them is exchange transfusion which was performed due to underlying erythroblastosis fetalis as discussed. A high index of suspicion and prompt treatment are critical to the survival of these neonates.

Key words: Splenic rupture, neonate, erythroblastosis fetalis, exchange transfusion.

INTRODUCTION

A newborn can be present with abdominal distension, pallor and shock due to several etiologies and one of them is splenic rupture with uncommon presentation and various etiology (Lewis et al., 2008). The mortality rate is high especially when the diagnosis is delayed. A high index of suspicion is needed for the prompt diagnosis. Unfortunately, the diagnosis becomes more difficult when one deals with non-traumatic delivery. Splenic rupture after performing exchange transfusion to neonates whose diagnosis was erythroblastosis fetalis as we discussed here was reported as few cases in the literature (Hui et al., 2002; Simmons et al., 1973).

CASE REPORT

A male infant of 28-years-old mother was born at 35 weeks' gestation by cesarean section. The birth weight was 2480 g and the Apgar scores were 8, 9 at 1 and 5 min, respectively. Hepatosplenomegaly and jaundice were the only findings on his first physical examination. He was transferred to the intensive care unit for investigating the etiology of organomegaly and the treatment for jaundice. Parent's first child was healthy and had no medical history. Second child died within a week; he had jaundice but did not undergo medical examination. Exchange transfusion was given to the third child due to jaundice. Patient and his mother's blood group could not be determined. Direct Coombs test was positive. White blood cell count was 114.700/mm^3 with 80% normoblasts. Erythroid series in various stages were seen on blood smear. He did not have anemia on the first day [hemoglobin (Hb): 14.7 gm/dl, haematocrit (Htc): 50%) and platelet count: 87.000/mm^3]. Biochemical analysis was normal. Abdominal ultrasonography performed on first day revealed hepatosplenomegaly. Double exchange transfusion with 0 Rh (-) erythrocyte suspension and fresh frozen plasma was performed to decrease total bilirubin level (23.7 mg/dl) immediately. The patient had bradycardia, hypotension, and lethargy with pale appearance following the exchange transfusion. He was put on the mechanical ventilation with nasal continuous positive airway pressure (n-CPAP) mode. Hemodynamic support and antibiotic therapy was started. Hb was 5.6gm/dl within a day, therefore erythrocyte suspension was transfused. Mother's blood group examination was repeated and reported as AB Rh1(+). Although indirect hyperbilirubinemia regressed, progressive direct hyperbilirubinemia was presented (Total bilirubin: 17.4 mg/dl, direct bilirubin: 11.3 mg/dl).

Progressive abdominal distention was determined on the third day. There were nonspecific findings on abdominal radiograph with no evidence of necrotizing enterocolitis. Diagnostic abdominal tap results showed the presence of hemoperitoneum. Abdominal ultrasonography was reported as excessive hepatosplenomegaly, 23 × 16 mm hypoechoic area on the lower edge of the spleen compatible with hematoma (Figure 1). Peritoneal drainage was performed. Various blood pro-ducts were transfused and homeostasis was maintained clinically.

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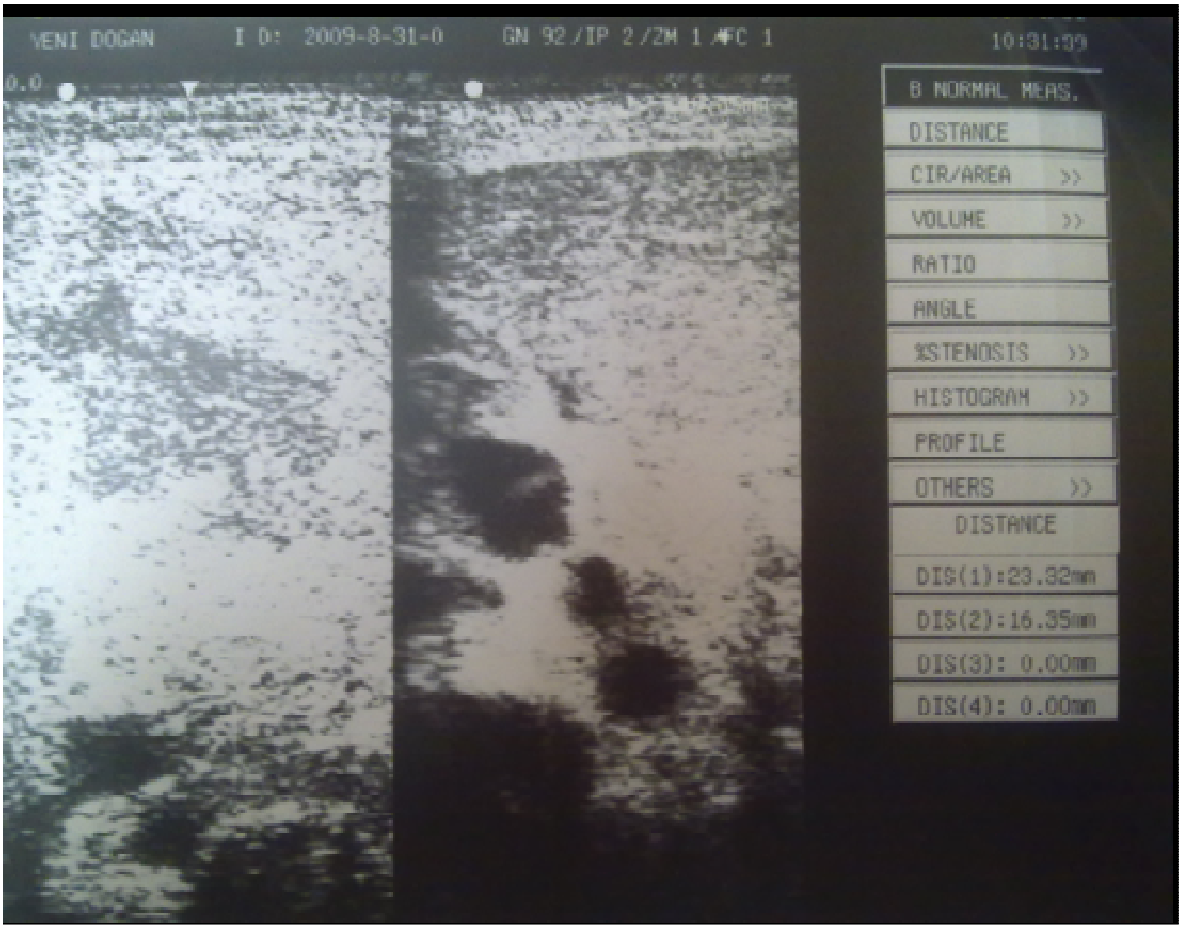

Figure 1. Abdominal ultrasonography finding of the case.

On the fifth day after the drainage was performed, bowel evisceration developed and reduction was done. He remained stable afterward and milk feeding was started on 7th day. Follow-up abdominal ultrasound scan on 12th day showed subcapsular collection at the lower pole of spleen compatible with resolving hematoma. Parents were consulted to hematology and anti-C subgroup incompatibility was determined. Subcapsular collection at the superior aspect of spleen had decreased in size (16.1× 6 mm) at 43rd day. He remained clinically well and was discharged at day 43 with a body weight of 3.1 kg.

DISCUSSION

Splenic rupture is relatively rare, but life threatening situation in the newborn (Lewis et al., 2008). The main underlying predisposing factor is traumatic delivery. The other rare etiologies are past syphilis, erythroblastosis fetalis or coagulopathy (Hui and Tsui, 2002). We reported an infant who had splenic rupture with underlying risk factor as erythroblastosis fetalis. The hemoperitoneum was developed after performing exchange transfusion in this case. Pathology can be explained in erythroblastosis as congested spleen may more easily rupture due to mechanical distension of the capsule and hypoxic damage of endothelium (Simmons et al., 1973).

Perinatal splenic rupture can be present either within the first few hours of life or as late as the second week of life, following rupture of the splenic capsule. The classical presentations are shock, anemia, and abdominal distension as we have seen in our case (Opalach et al., 1996, Tengsupakul et al., 2010). It is important to remember that the classic triad of the disease is not always present (Hui and Tsui, 2002). In our case, the diagnosis of intra-abdominal bleeding was made on the basis of clinical and laboratory findings. The abdominal distension was an important sign of intraabdominal bleeding, and it appeared as early as at the third day in our case. Diagnostic paracentesis was suggestive of hemoperitoneum which was confirmed by ultrasonography. Ultrasonography signed that the source of bleeding was spleen, also. Abdominal computed tomography or ultrasonography were useful tools with high sensitivity in

confirming the diagnosis and the follow up (Simmons, 1973, Lewis et al., 2008).

Splenectomy is one of the treatment options but due to the potential risk of post-splenectomy sepsis, the efforts to preserve the spleen are indicated (Ting et al., 2006). We preferred to perform peritoneal drainage to our patient after the diagnosis as the recent literature suggested (Ye et al., 2009, Bickler et al., 2000).

In conclusion, the majority of patients with splenic rupture died because of delayed or missed diagnoses. A high index of suspicion, early recognition, and prompt treatment are critical to the survival of these neonates. Non operative management is the first choice for the treatment due to high survival rates.

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