Case Report

Gastric perforation

An unusual cause of ascites in a newborn baby

Omer B. Abdelbasit, *FRCP, FRCPCH,* ***Meeralebbae M. Shaheed,*** *FRCP, FRCPCH,* ***Mohammad S. Al-Omari,*** *MD,* ***Abdullah M. Abomelha,*** *FACHARZT,* ***Mirghani O. Babiker,*** *FRCR, DMRD.*

ABSTRACT

We report a case of a low birth weight asymmetrical small for gestational age baby, who presented at the age of 20 hours with sudden abdominal distension. Since birth he has been breastfed and was kept with his mother. Absence of radiological findings of necrotizing enterocolitis or perforation at the time of presentation delayed the diagnosis for 48 hours. At laparotomy the baby was found to have perforation of the stomach with no evidence of other gastrointestinal disorder.

Keywords: Gastric perforation, unusual ascites, newborn baby.

Saudi Medical Journal 2000; Vol. 21 (11): 1085-1087

This male baby was delivered spontaneously at term. He had evidence of intrauterine growth retardation according to the antenatal ultrasound finding of reduced foetal growth at 28 weeks, 32 weeks, 35 weeks and 37 weeks of gestation. There was no maternal hypertension, diabetes, or other medical disorders. There was no history of maternal herpes simplex, cytomegalovirus or toxoplasma infections. Mother was rubella immune. Placenta appeared complete and normal and weighed 460 grams. The baby had an apgar score of 9 at 1 minute and 9 at 5 minutes and did not require any resuscitative measures, and no suction was applied. Birth weight 2095 grams, length 49 cm and head circumference 33.5 cm. The weight was below the 10th centile for gestational age but the head circumference and length were appropriate for gestational age ie. asymmetrical small-for-gestational age (SGA). Clinically there were no abnormal physical findings in the cardiovascular, respiratory, alimentary or central nervous systems but because the baby was SGA he was started soon after birth on oral feeds which were tolerated well from breast and bottle. Regular glucose monitoring by dextrostix was within the normal range. The baby's condition was stable until the age of 20 hours when he was reported to develop tachypnea, grunting and mild subcostal recession. On clinical examination the baby was found to have signs of respiratory distress with a respiratory rate of 75/minute, heart rate of 160/ minute, and a normal blood pressure. Abdominal examination revealed generalized distension with generalized tenderness but with no organomegaly. Signs for fluid could not be elicited. Cardiovascular system was normal and breath sounds were heard equally in both lungs. A diagnosis of an early neonatal sepsis with possibility of necrotizing enterocolitis was entertained. Arterial blood gasses showed metabolic acidosis with a pH of 7.32, P02 of 70.8 mmHg, $PC0_2$ of 25.6 mmHg, bicarbonate of 16.7 mmol/L and a base deficit of -9.8 mmol/L.

From the Department of Pediatrics, (Abdelbasit, Shaheed, Abomelha, Al-Omari), and Medical Imaging Department, (Babiker), Security Forces Hospital, Riyadh, Kingdom of Saudi Arabia.

Received 26th June 2000. Accepted for publication in final form 26th August 2000.

Address correspondence and reprint request to: Dr. Omer Bashir Abdelbasit, Pediatrics Department, PO Box 3643, Riyadh 11481, Kingdom of Saudi Arabia. Tel. +966 (1) 477 4480 Ext. 1390 Fax. +966 (1) 476 4757 or 477 4480 Ext. 2105. Email: basit53@hotmail.com

Blood count showed a hemoglobin level of 172 gm/L white blood cell counts of 4.7 X 10^9/L with neutrophil making 73%. Platelets were 180 X 10^9/L. Blood urea nitrogen, creatinine and electrolytes were within normal. First x-rays of the abdomen revealed evidence of fluid in the abdomen but there was no evidence of intramural gas, thickening of the intestinal wall or free air. Ultrasound of the abdomen confirmed the presence of ascites. (Figure 1)

The baby was started on gentomycin, ampicillin metronidozole and intravenous fluids but continued to be lethargic and sick. At no points of management was prophylactic H2-blocker or proton pump inhibitor used. The abdomen remained distended and developed erythema of abdominal wall, tenderness and decreased bowel sounds. At the age of 48 hours it was possible to elicit shifting dullness. The baby did not open his bowel since birth. There was no increase in the gastric aspirate. Repeat blood urea nitrogen, creatinine and electrolyte, liver enzymes, and proteins were normal. Coagulation studies revealed a prolonged prothombin time of 32.3 seconds, partial thromboplastin time of 61.9 seconds and an INR of 2.6. Vitamin K was given. A repeat blood count at the age of 48 hours revealed a hemoglobin of 150 gm/L, white blood count dropped to 1.3 X 10^9/L with neutrophils of 50% but with no band forms and platelets of 107 X 10^9/L.

At the age of 64 hours the abdominal distension was noted to increase but there was no change in the clinical picture and vital signs remained stable. A left decubitus x-ray of the abdomen at that time revealed free air in the peritoneum. (Figure 2). The baby was taken to operating theatre with the diagnosis of intestinal perforation. At laparotomy a large volume of milk was suctioned from the peritoneal cavity and there were thick yellowish fibrinous filaments over the intestinal wall and stomach. After inspection of the entire gastrointestinal tract the perforation was found to be in the posterior wall of the stomach with more than 50% of the wall sloughed. The lesser sac was full of milk and fibrin filaments which were sent for analysis and culture. The peritoneal cavity was cleansed with saline, a gastrostomy tube was placed through the anterior wall of the stomach and the posterior wall was approximated with 5-0 vicryl to seal the perforation. The rest of the gastrointestinal tract looked healthy with no evidence of ulceration or necrosis. The baby had an uneventful post operative course and was started on enteral feeds 10 days post operatively after excluding obstruction and abnormalities of the gastrointestinal tract by contrast study.

Discussion. Necrotizing enterocolitis is the leading cause of gastrointestinal perforation. However recently more and more cases of idiopathic gastrointestinal perforations have been described in literature.[1] A number of mechanisms have been suggested as possible causes of perforation.[2] We report here of a low birth weight male baby born at term but was small for gestational age. This baby had no evidence of prematurity, asphyxia, trauma, hypoglycemia or other apparent insults that could have caused perforation and was not ventilated. Immediately after birth the baby had normal vital signs and normal physical examination findings and was kept with his mother.

From the operative findings it was evident that the cause of the abdominal distension and the respiratory distress was due to peritonitis or sepsis due to the milk which exuded through the perforated posterior wall of the stomach. There was no mechanical intestinal obstruction. Clinically this baby gave us concern because of the presentation of abdominal distension and respiratory distress without radiological evidence of perforation which only became apparent after 42 hours suggesting that gastric perforation could remain radiologically silent

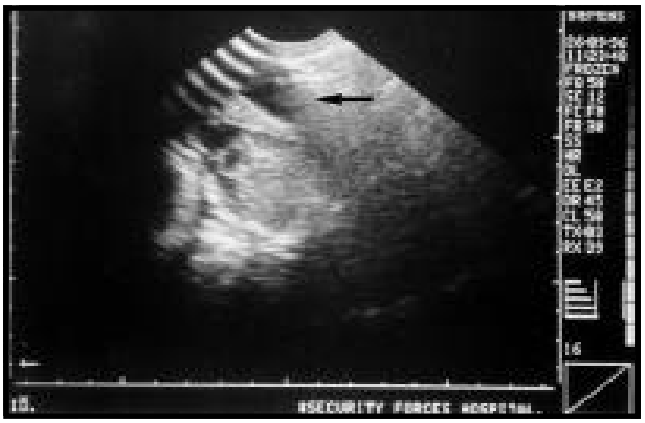

Figure 1 - Ultrasound abdomen showing fluid ascites.

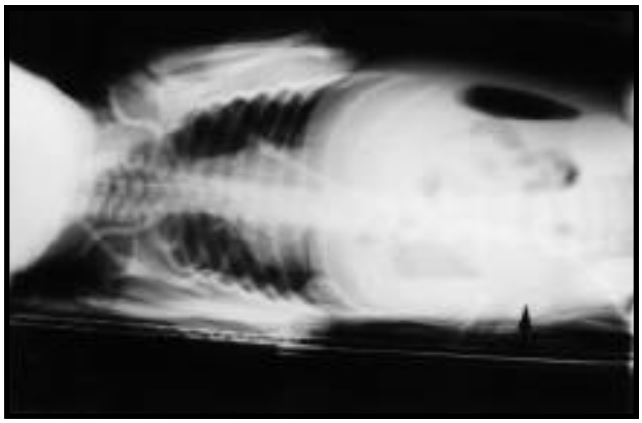

Figure 2 - Right decubitus x-ray of the abdomen showing free gas in the peritonium.

for a considerable length of time. The absence of other aetiological factors for ascites related to the heart, liver, kidneys should have directed one's attention to other causes. The necrotizing enterocolitis though not confirmed radiologically and clinically should not deter the physician from performing the valuable serial radiological evaluations of any baby presenting with sudden acute abdominal distension. Most cases of spontaneous gastrointestinal perforations were reported in very low birth weight babies. Given the clinical, microbiological and histopathological findings in this case the aetiology of gastric perforation could only be speculated. The fact that the baby was small for gestational age might have predisposed him to episodes of intrauterine hypoxia, at different times during pregnancy, and associated stresses which may have produced intrauterine gastric ischemia, necrosis and hence perforation.[1,3]

The role of congenital infection as a cause of gastric perforation cannot be ignored in this baby.[4] The blood culture grew candida and the peripheral blood count revealed leukopenia and neutropenia in the immediate postnatal period. However the culture from peritoneal fluid obtained at laparotomy did not grow candida but only enterococus species. The histopathological examination of the material also did not reveal candida. It is still possible that the infection could have predisposed to ischemic necrosis of the stomach. The baby did very well after treatment with amphotericin B and fluconazole. Over distension of the stomach with air or fluid is a possible cause of perforation and this has been linked to congenital deficiency of musculature in the stomach wall.[5] This baby did not undergo any active resuscitation and had received several feeds before the development of abdominal distension. Still this could be a predisposing factor.

In conclusion, gastrointestinal perforation presents in the majority of patients with normal bowel gas pattern followed by rapid development of paucity of bowel gas in very low birth weight babies. In our patient, perforation did not cause paucity of the bowel gas, rather it produced milk ascites with delay of radiological signs of perforation for almost 2 days. A high index of suspicion is necessary in babies especially low birth weight babies who present with unexplained ascites without clinical and radiological evidence of necrotizing enterocolitis. Retrospectively diagnostic paracentesis may have helped in establishing the diagnosis in this case.[3] The outcome in gastric perforation is excellent as the lesion is localized and not associated with necrotizing enterocolitis.[6]

Acknowledgment. The authors would like to thank Ms. Bing Borromeo for typing the manuscript.

References

1. Zamir O, Goldberg M, Udassin R, Peleg O, Nissan S, Eyal F. Idiopathic gastrointestinal perforation in the neonate. J Ped Surg 1988; 23: 4; 335-336.
2. Berseth CL. Disorders of stomach: Gastric perforation in Taeush, Ballard Eds. Avery's diseases of the newborn, 7th Ed. Philadelphia: WB Saunders Co. 1998; 916.
3. Weinber G, Kleinhaus S, Boley SJ. Idiopathic intestinal perforation in the newborn. An increasingly common entity. J Ped Surg 1989; 24: 10; 1007-1008.
4. Mintz AC, Applebaum H. Focal gastrointestinal perforations not associated with necrotizing enterocolitis in very low birth weight neonates. J Ped Surg 1993; 28:6: 857-860.
5. Desa DJ. The alimentary tract: The stomach. Gastric perforation in the newborn in. Jonathan's Wigglesworth and Don B. Singer Eds. Textbook of fetal and perinatal pathology. Boston Blackwell Scientific Publications 1991; 918-919.
6. St. Vil D, Le Bouthillier G, Luke FI, Bensoussan AL, Blanchard H, Yousef S. Neonatal gastrointestinal perforation. J Ped Surg 1992; 27: 10: 1340-1342.