Neonatal Hemoperitoneum: Unexpected Birth Trauma with Fatal Consequences

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ABSTRACT

We report a case of fatal intra-abdominal bleeding in a term newborn delivered by vacuum extractor and Kristeller manouvre. Although autopsy was not performed in compliance with the parents' wishes, there is strong evidence of a massive abdominal haemorrhage due to injuries of the hypochondriac organs probably leading to disconnection of a vascular pedicle.

Key words:
Abdominal bleeding, birth injuries, birth trauma, newborn

INTRODUCTION

Abdominal haemorrhaging in newborns is a dramatic event that may be caused by several obstetrical and foetal factors including breech or complicated delivery, hepatosplenomegaly, macrosomy and anoxic liver congestion. It is an uncommon yet severe and life-threatening birth trauma. Birth injuries can be divided into two categories based on their aetiology: 1- injuries deriving from hypoxia and ischemia; 2- injuries deriving from mechanical stress during labour and delivery.[1] In this second group intra-abdominal injuries are rare and involve rupturing or haemorrhaging in the liver, spleen, or adrenal glands. Liver injury is the most common of these injuries. Three potential mechanisms lead to intra-abdominal injury: 1- direct trauma; 2- compression of the chest against the surface of the spleen or liver; 3- chest compression leading to tearing of the ligamentous insertions of the liver or spleen.[2,3] Clinical symptoms are correlated with the appearance of shock, the intensity of which is proportional to the severity of the trauma. Especially in the first hour of life, the clinical signs of impending shock could be misunderstood or mistaken for asphyxia. A central hematocrit may be misleading immediately after acute blood loss and hypotension may also be a late sign.

CASE REPORT

A 3400-gram neonate was delivered by vacuum extractor in a country hospital after a normal pregnancy in a young healthy woman. The baby cried and was put on the mother's abdomen however his clinical status deteriorated unexpectedly and required cardiopulmonary resuscitation (CPR) with intubation and external cardiac massage. An umbilical venous catheter (UVC) was inserted, three doses of adrenaline were administered. Apgar score was 1 and 3 at the first and fifth minute of life. The neonatal transport team transferred the baby to our NICU with a presuntive diagnosis of perinatal asphyxia. At 1 and half hours of age, pH was 7.29 pCO_2 22.4 EB –14, arterial haemoglobin 15.5 g/l. He was breathing spontaneously, with oxygen saturation levels 92-95%, and was administered dopamine. On admission the infant was mottled, pale, hypotonic, and hypoperfused. The abdomen was soft. Pulse rate was 150 beats per minute and cuff blood pressure was not detectable. Blood gases showed normal respiratory exchange and a severe metabolic acidosis (pH 7.08 pO_2 112 pCO_2 22 EB –21.6). Haemoglobin was 13.4 g/l. The chest X-ray [Figure 1] at the birth hospital showed a slim cardiac silhouette and a mal-positioned UVC. Fluid replacement therapy was initiated. An echocardiogram showed a normal heart, however during the exam, ten minutes after arrival, there was the onset of bradycardia which failed to clear up. At the positioning of a new UVC, the umbilical stump bled profusely and the catheter tip entered too easily and too deeply. An X-ray was requested while the infant was administered three doses of colloide, dopamine, and dobutamine in increasing

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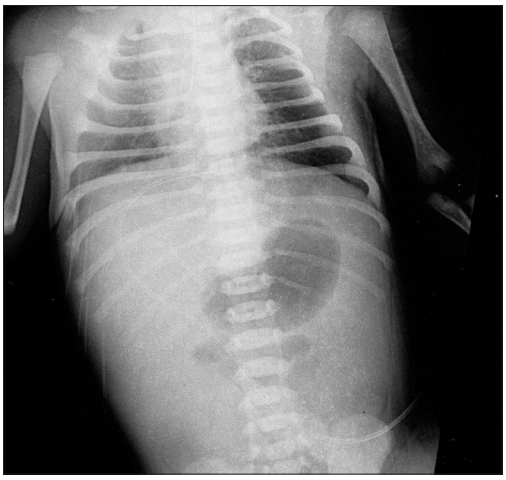

Figure 1: Anteroposterior roentgenogram of chest and abdomen obtained in the delivery room, 30' of age: Bowel gas pattern suggests presence of peritoneal fluid. Configuration of catheter is not consistent with the usual course of the umbilical vein. The cardiac silhouette is very slim

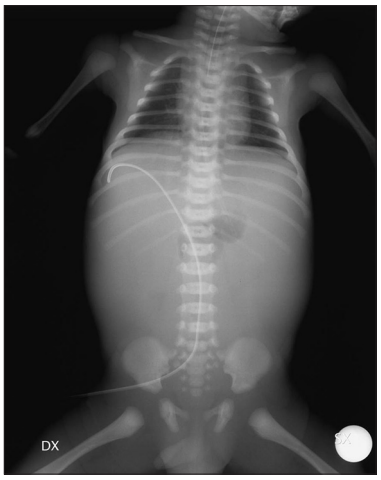

Figure 2: Anteroposterior roentgenogram of chest and abdomen obtained in the intensive care unit, 4 hours of age: Bowel gas pattern suggests presence of peritoneal fluid. The catheter is still deviated from its expected course. The cardiac silhouette is very slim

doses, and bicarbonate through the new catheter. He was totally non-responsive and his heart rate started dropping. The X-ray showed a white abdomen with the catheter tip, once again in an unusual position [Figure 2]. The haemoglobin level dropped to 10 g/l. An ultrasonography confirmed the clinical and radiological suspicion of a massive abdominal haemorrhage. An anaesthesiologist and surgeon were immediately summoned, however the infant, in irreversible shock, died 50' after admission. An autopsy was not performed due to the parent's opposition.

DISCUSSION

In the short clinical history of our patient there were several significant features: all our efforts to establish a positive cardiac function were ineffective; shock was not amenable to fluid replacement therapy; both the first and second X-rays showed a very slim cardiac silhouette, with a bowel gas pattern suggesting the presence of peritoneal fluid, and a configuration of both the UVCs that was inconsistent with the usual course of the umbilical vein; the bleeding from the umbilical stump was too profuse. The drop in the haemoglobin level, hypotension and presence of free blood in the peritoneal cavity confirmed the occurrence of a major intra-abdominal haemorrhage. Abdominal haemorrhaging in newborns is an uncommon yet severe and life-threatening event. Direct or indirect trauma of an abdominal organ during delivery represents the major pathogenetic factor. Clinical symptoms are correlated with the appearance of shock. A central haematocrit, immediately after acute blood loss, may be misleading, so serial hematocrits should be obtained, and hypotension may also be a late sign. Perforation of the peritoneum and intra-abdominal haemorrhaging has been described as a complication of umbilical vein catheterizations[4] however in our case the UVC was inserted after deterioration of the clinical status. Liver angioma is the most common congenital condition that could lead to abdominal bleeding during birth[5] due to a different tensile strength of the tissue in comparison with the rest of the liver. In our case, foetal ultrasonographies were normal. Disconnection of the splenic vascular pedicle causing hemoperitoneum has also been described[6] as well as rupturing of the liver in term newborns of normal weight after uncomplicated delivery.[7] In our case the infant presented immediate and severe shock, and was totally unresponsive to fluid replacement therapy. From the appearance of both the UVCs' positions we believe that both the catheters in our patient were positioned in the peritoneal cavity, outside of the vascular bed. This hypothesis would explain why all our efforts to reinstall a positive blood pressure were ineffective as the infusions were running through the UCV. On reviewing the infant's obstetric history, the delivery was complicated by the use of a vacuum extractor and the repeated use of the Kristeller manoeuvre. We are aware that a lateral X-ray could have helped in defining the localizations of both the UCVs, moreover, an autopsy would have been decisive in demonstrating the interruption of the vascular pedicle, however we believe that there is a strong evidence of this aetio-pathogenesis in the clinical evolution of our patient. Even without evidence of birth trauma, intra-abdominal bleeding must be suspected in a newborn with a sudden onset of shock symptoms. Interruption of the vascular tree of the hypochondriac organs in the case of shock and unresponsiveness to fluid replacement therapy through umbilical vessels must be kept in mind to plan the therapy and select a different infusion's route.

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