Gall bladder distention in newborns

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SUMMARY Three cases of gall bladder distention in asphyxiated newborns are described. Clinical and ultrasound examination showed this to be a benign, transient phenomenon. A causal relation between tissue hypoxia and gall bladder distention is proposed.

Gall bladder distention in neonates has only recently been described by several authors,¹⁻⁶ but considerable conjecture regarding the pathophysiology of this finding exists as reflected by the numerous hypotheses proposed. Lack of enteral feeding,¹⁻³ sepsis,⁴ hepatic vein engorgement,⁵ and transient functional disturbance of gall bladder motility⁶ have all been implicated as causes of gall bladder distention.

Transient gall bladder distention occurring in three asphyxiated neonates is described and an hypothesis linking this phenomenon with tissue hypoxia is proposed.

Case reports

Case 1. An asphyxiated boy, weighing 4100 g, was delivered by caesarean section at 42 weeks' gestation. Apgar scores were 3 and 4 at one and five minutes, respectively. The initial blood pH was 7-16 with a base deficit of 12-2. The immediate neonatal course was complicated by seizures, acute tubular necrosis, ileus, and urinary retention. On the third postnatal day, a large, firm mass was palpated in the right hypochondrium extending to below the level of the umbilicus. Ultrasound examination showed a distended gall bladder measuring 4-6×2 cm (fig 1). Nasogastric feeds of expressed breast milk were introduced on the fourth postnatal day. The mass remained palpable until the 13th day. A repeat ultrasound examination on day 11 confirmed resolu-

tion of the enlargement. An unconjugated hyperbilirubinaemia was prolonged, but not severe. Blood cultures were sterile.

Case 2. A boy, weighing 2350 g, was delivered by caesarean section after fetal distress at 36 weeks' gestation. The Apgar scores were not accurately recorded, but the blood pH was 7-09 and base deficit 14-5. There was no conclusive evidence for acute tubular necrosis, ileus, or urinary retention. The infant was severely hypotonic, however, and remained so during the period of hospital care. A firm, regular mass was palpated in the right hypochondrium on the third day. Ultrasound examination showed a distended gall bladder measuring 4×1-3 cm (fig 2). The mass was not clinically

![Ultrasound picture taken on third postnatal day showing distended gall bladder containing debris in case 1. (L=liver, gb=gall bladder, and D=debris).](image-url)
A mild hyperbilirubinaemia was of short duration. Nasogastric feeds of expressed breast milk were introduced on the fifth day. Aggressive resuscitation was required. An early blood gas analysis was not performed. The immediate postnatal course was complicated by apnoeic spells and acute tubular necrosis. On the third day, a right sided abdominal mass was palpated. An ultrasound examination confirmed the presence of a distended gall bladder, measuring 4.9 x 1.6 cm. A grade III intraventricular haemorrhage was observed on cranial ultrasound. Blood cultures were sterile. Nasogastric feeds of expressed breast milk were initiated on the fourth day. By the 10th day the abdominal mass was no longer palpable. A moderate unconjugated hyperbilirubinaemia was short lived.

**Case 3.** A boy, weighing 1100 g. was delivered vaginally at 30 weeks’ gestation. Apgar scores were 2 and 6 at one and five minutes, respectively. Aggressive resuscitation was required. An early blood gas analysis was not performed. The immediate postnatal course was complicated by apnoeic spells and acute tubular necrosis. On the third day, a right sided abdominal mass was palpated. An ultrasound examination confirmed the presence of a distended gall bladder, measuring 4.9 x 1.6 cm. A grade III intraventricular haemorrhage was observed on cranial ultrasound. Blood cultures were sterile. Nasogastric feeds of expressed breast milk were initiated on the fourth day. By the 10th day the abdominal mass was no longer palpable. A moderate unconjugated hyperbilirubinaemia was short lived.

**Discussion**

Asphyxia neonatorum is known to reduce blood flow to expendable, splanchnic organs, in favour of flow to more vital organs. The hypoxia suffered by these splanchnic organs may result in an ileus, necrotising enterocolitis, acute tubular necrosis, and atomic distention of the urinary bladder. We believe this mechanism to be causal in the three cases of gall bladder distention described above.

The hypoxia produced by reduced splanchnic blood flow may cause hollow organ dysfunction in two ways. Firstly, a direct effect upon smooth muscle function or secondly, by autonomic nerve dysfunction.

Most previously described cases have occurred in the setting of an ill newborn with respiratory distress syndrome or septicaemia in which tissue hypoxia was most likely. Two authors have described gall bladder distention in the asphyxiated newborn, however, they did not link the findings. We conclude that tissue hypoxia may have an important pathophysiologic role in producing transient gall bladder distention in ill neonates.

**References**


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