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Answer

A rare case of persistent fetal intestinal hyperechogenicity – Answer

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Answers

1. In the anteroposterior (AP) radiography, we can see a significant reduction in the lung volume also due to the increased abdominal volume. There is a marked widespread increase in lung density, especially on the right side. The heart figure is hardly visible. The endotracheal tube is a little above the carina of the trachea. The nasogastric tube is correctly positioned. The abdomen is significantly increased in volume and almost completely opaque, with air only in the epigastric region. These findings also appear in the translateral view. The radiographic images are compatible with a respiratory distress syndrome and with the presence of a massive abdominal effusion.
2. An abdominal ultrasound should be performed.
3. A hyperechogenicity of the intestinal loops was present on fetal ultrasound starting from the 12 weeks of gestation. The most common causes, aneuploidies and cystic fibrosis, were excluded. Another possible cause of persistent fetal intestinal hyperechogenicity is intestinal obstruction. The appearance of intestinal and gastric dilation in the second fetal ultrasound confirms this diagnosis.

Discussion

Bowel hyperechogenicity has been defined [1] as similar or greater echogenicity of the bowel than surrounding bone [2] or fetal liver [3, 4].

In the second trimester of gestation, when meconium begins to accumulate in the intestinal lumen, a transient hyperechogenicity can be found, which disappears in the following weeks. In most newborns, intestinal hyperechogenicity is resolved upon the establishment of a normal intestinal function [2, 5].

The persistence of hyperechogenicity in the third trimester is generally linked to an underlying disease [5, 6], although in some rare cases it is not associated with any pathological condition [7]. It is often linked to aneuploidy [8, 9], especially trisomy 21, 13 and 18, or to Turner syndrome. It is frequently found in fetuses with cystic fibrosis [10], due to an increase of meconium consistency for alteration in the secretion of the pancreatic enzymes. Another cause of persistent hyperechogenicity is represented by intestinal obstruction and atresia. Other less common associations are growth restriction and infections such as cytomegalovirus (CMV), toxoplasmosis and parvovirus [3, 4].

Midgut volvulus is a life-threatening condition and represents a surgical emergency [11]. It is characterized by the torsion of the loops of the small intestine or of the proximal colon around the fetal mesenteric artery and its branches with consequent mechanical obstruction, vascular compromise and secondary intestinal necrosis, which can extend to the entire intestine, from Treitz ligament to the colon transverse. Most fetal or neonatal cases are associated with intestinal malrotation, less frequently with congenital malformations, such as omphalocele, gastroschisis, intestinal atresia or annular pancreas [12].

Midgut volvulus not associated with malrotation or congenital anomalies is extremely rare [13-16]. Vascular compromise can cause intestinal infarction and eventual perforation, with the development of hemorrhagic fetal ascites and fetal anemia [16-19]. A further complication of intestinal necrosis can be intestinal atresia.

Volvulus should be suspected when fetal ultrasound shows persistent intestinal hyperechogenicity followed by dilation of the loops, especially in association with fetal ascites [20, 21].

With a worsening of the condition, a progressive obstruction with polyhydramnios is observed; fetal anemia resulting from hemorrhagic ascites leads to a reduction in fetal movements and heart variability, which requires an emergency cesarean section [17, 20]. Sometimes the constriction of the intestinal loops, caused by the volvulus, can lead not only to a dilation of the bowel, but also to a distension of the stomach [22].

After birth, the final diagnosis of intestinal volvulus can be made. At the clinical examination, the abdomen can appear increased in volume with poor treatability, and the X-ray shows the presence of an abdominal effusion.

The survival of newborns with this disease is influenced by the gestational age at birth, the extent of intestinal necrosis and the ability to tolerate surgery.

Clinical course

In our case, the hyperechogenicity of the fetal intestine was observed for the first time during the 12th week sonography and was not resolved later. At the ultrasound checkup at 27 weeks, there was intestinal and gastric dilation; during the following one, at 29 weeks, a peritoneal effusion was also

evident. The conditions of the fetus worsened rapidly, and the cardiotocographic trace showed a very reduced cardiac variability that required an urgent cesarean section.

The abdominal ultrasound performed immediately after birth revealed corpuscolar abdominal free fluids (**Fig. 1**). The bowel loops were dilated, with very thick walls, filled with corpuscle material.

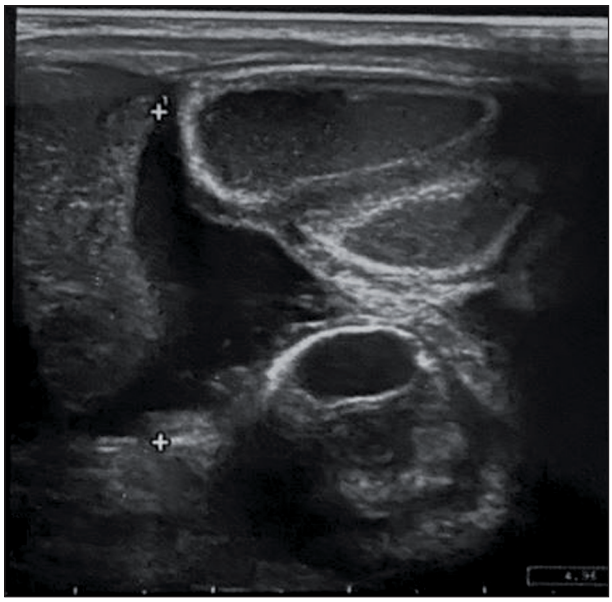

Figure 1. Abdominal ultrasound.

The child's clinical condition worsened rapidly. The oxygen saturation was at the lower limit even after the administration of Curosurf®. For this reason, it was not possible to transfer the patient to the Pediatric Surgery Department. So the surgeon performed a paracentesis in the Neonatal Intensive Care Unit (NICU), subtracting about 100 cc of hemorrhagic fluid. After a transitory improvement, the little girl died.

The autopsy confirmed the presence of an intestinal volvulus with extensive hemorrhagic necrosis of the whole ileum, with dilated loops filled with hemorrhagic fluid, in the absence of intestinal malrotation.

Declaration of interest

The Authors declare that there is no conflict of interest.

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