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Meconium Peritonitis: Antenatal Diagnosis and Management. Two Case Reports

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Abstract

Meconium peritonitis (MP) is a sterile chemical peritonitis resulting from intestinal perforation in utero. We present two cases of preterm neonates weighing 2580 g and 3100 g respectively, in whom fetal meconium peritonitis was diagnosed by antenatal ultrasonography (US) and magnetic resonance imaging (MRI). Both patients required surgery. As the literature suggests, newborn surgery for meconium peritonitis is sometimes very difficult owing to severe adhesions and bleeding. The aim of these case reports is to underline that close collaboration between neonatologists and pediatric surgeons is essential for the timely diagnosis and prompt management of MP, but also to reveal the benefit of primary anastomosis (PA) for MP as well as to demonstrate the importance of the imagistic techniques in diagnosing and managing MP, both before and after birth.

Keywords: meconium peritonitis, pediatric surgery, preterm, primary anastomosis

Introduction

Meconium peritonitis (MP) is defined as an aseptic, localized or generalized peritonitis due to leakage of meconium into the peritoneal cavity, secondary to bowel perforation in utero. Peritonitis caused by postnatal gastro-intestinal perforations even if meconium is involved is not included in the diagnosis of MP [1].

The incidence of MP is reported as 1/30000, which might be an underestimation due to spontaneous recovery of bowel perforation and regression of the inflammatory process before the time of birth, in some cases without clinical manifestation [2]. While the mortality rate was approximately 70% in the 1960s, in these days it is around 10-15%, but still with significant perinatal morbidity [3].

Meconium is an intestinal content, starting to form within the 3rd gestational month. It is composed of amniotic fluid with bile salts, cell debris and proteins, uric acid, intestinal enzymes, cholesterols, inorganic salts and sugar. Bowel perforation will induce chemical peritonitis triggered by bile salts and enzymes (such as lipase) spilled into the abdominal cavity [1]. The immediate result is inflammation through the release of chemical mediators causing phagocytosis, especially TNF- production which is reported to remarkably increase when the peritoneum encounters meconium. The result is fibrin deposits and severe intra-abdominal adhesions [4]. Meconium was demonstrated to give rise to a peritoneal reaction accompanied by fibroblastic proliferation that covers the lesion, followed by foreign body granulomas and calcifications [5]. The result is represented by adherences of intestinal loops, with a fibrous tissue that is difficult to dissect. The exact location of the perforation is hard to find due to the calcifications and disseminated meconium inclusions.

There are many hypotheses regarding the intrauterine intestinal perforation, neither of which has been substantiated yet. Bowel hypoxia seems to be the main factor, but no consensus has been achieved. The main etiological factors are: intestinal atresia, intestinal volvulus and meconium ileus related to cystic fibrosis [1].

The fetus is able to swallow amniotic fluid in the 12th gestational week, the same time the bile secretion begins in the fetal liver; in the 16th gestational week, meconium reaches the ileum for the first time. Whether or not the necrosis or perforation of the intestine occurs before or after this week, it would result in intestinal atresia with or without meconium peritonitis. The timing of this event will also determine the pathological type of MP: fibro-adhesive, generalized or cystic type. If the perforation site is sealed off before meconium passes, there will be no spillage of meconium in the peritoneum, but the digestive enzymes

reaching the peritoneal cavity will cause chemical peritonitis, which eventually will lead to a local fibroblastic reaction and the perforation effectively seals off, resulting in the fibro-adhesive type of MP. If the perforation cannot be contained and the intestine presents an intense inflammatory process followed by its fixation to the adjacent structures, it will lead to the formation of a cystic meconium-filled cavity by fixed intestinal loops, resulting in the cystic type of MP. Sometimes meconium is spread in the entire peritoneal cavity, resulting in the generalized type of MP [6].

As immediate diagnosis and surgery are crucial after birth, termination of pregnancy is unnecessary [7]. However, an elective preterm delivery by cesarean section at 35th gestational week could be recommended to prevent disease progression and enable an early intervention [8].

Antenatal US is the primary investigation used for the diagnosis of MP, revealing intraabdominal calcifications, fetal ascites, pseudocyst formation, dilated bowel loops or collapsed intestines. Based on the US criteria, Zangheri et al. developed a grading system used for international standardization of prenatal MP diagnosis [9]. Even though US has a high sensitivity for cystic lesions, there are many diseases to be considered in the differential diagnosis of cystic masses, such as: ovarian cyst, mesenteric cyst, digestive duplication cyst - which render the diagnosis difficult.

Differential diagnosis may include multiple bowel atresia, colonic atresia, Hirschsprung's disease, high anorectal malformations and cloacal anomalies. Adrenal and liver calcifications can also be identified with cytomegalovirus, parvovirus infections or neuroblastoma, hepatoblastoma [10-12].

Fetal MRI can be a useful tool in describing the exact pathology and comorbidities, even if it's not needed in all cases [1].

Meconium peritonitis can also be identified prenatally by diagnosing meconium through open processus vaginalis. The diagnosis of MP in the postnatal period is based on clinical findings and scrotal or abdominal radiographs and ultrasonography [10]. In patients without prenatal suspicion or diagnosis, the evidence of MP in the postnatal period is based on clinical presentation of intestinal obstruction: newborn revealing abdominal distension, vomiting, failure to pass the meconium, respiratory distress. Local signs, such as color modifications (red, cyanotic, brown) of the abdominal skin are an optional feature.

Abdominal X-Ray might reveal pneumoperitoneum or calcifications in the peritoneal cavity, sometimes including scrotal calcifications [13]. Because the meconium bacterial overgrowth begins just after birth, immediate and proper diagnosis is fundamental for the prognosis of the neonate. MP might also be present at birth as a scrotal mass with or without inguinal involvement, due to the presence of calcified meconium in the scrotum. Scrotal calcifications could also be demonstrated on the radiography.

Prenatal US findings are a good indicator in deciding a candidate for surgery and selecting them for delivery in a tertiary center, which disposes of both neonatal and pediatric surgery departments [14].

A management for the prenatal period is not yet unanimously accepted. There were some attempting trials such as the injection of trypsin inhibitor into fetal abdominal cavity, in order to reduce meconium-induced chemical peritonitis and diminish intraabdominal pressure to improve mesenteric vascular supply and also remove inflammatory debris [15].

Clinical signs of intestinal obstruction and/or bowel perforation are clear indicators for surgery, while simple presence of intraabdominal calcifications is not mandatory for surgical explorations but requires close follow-up. After admission, intensive care unit monitoring is necessary, along with prophylactic antibiotics. After laparotomy, adhesiolysis should only be attempted in the purpose of discovering the perforation site or relieving a major obstruction. After bowel resection, end-to-end anastomosis is recommended, the other possibility being a temporary enterostomy with delayed reconstruction of intestinal continuity [15].

Complications related to the surgical treatment are common: anastomotic leakage, necrosis of the enterostomy, enterocutaneous fistula, postoperative ileus, sepsis, short bowel syndrome [11,16].

Case Report #1

A five-hour old female patient was referred to our clinic after being born in another medical center through cesarean section. The pregnancy was partially supervised and the diagnosis of polyhydramnios was determined using an ultrasound examination. At 33 weeks of pregnancy the ultrasound examination revealed bowel distension. At birth, the patient weight was 2850 grams and she was born at 35 weeks of gestation with an Apgar score of 8. The postnatal examination revealed axial hypotony, acrocyanosis, subcostal retractions, nose flaring, an abdomen that was distended, cvasi-rigid and with a stretching tendency, which presented collateral circulation, pallor and a periumbilical ecchymosis. After the nasogastric tube was inserted, it revealed, on aspiration, that the stomach contained meconium.

The patient was admitted in the ICU where a peripheric venous catheter was placed and through which we administered glucose 10% at a rhythm of 6.5 ml/h.

The abdominal ultrasound on admission revealed distended bowel loops with echogenic walls, inhomogeneous content and accelerated peristaltic movements. Also, the examination unveiled liquid in small quantity localized in-between the bowel loops, near the liver (Figure 1-3).

The abdominal radiography on admission showed no signs of pneumoperitoneum, but revealed a few hydro-aeric levels. The transfontanellar ultrasound showed no abnormalities, but the echocardiography showed an associated patent foramen ovale, minimal mitral valve insufficiency and pulmonary hypertension (Figure 4).

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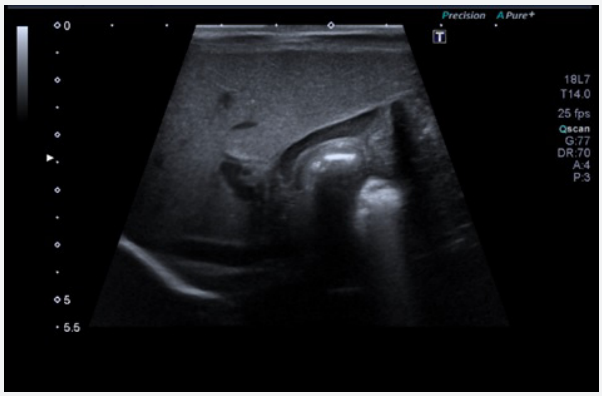

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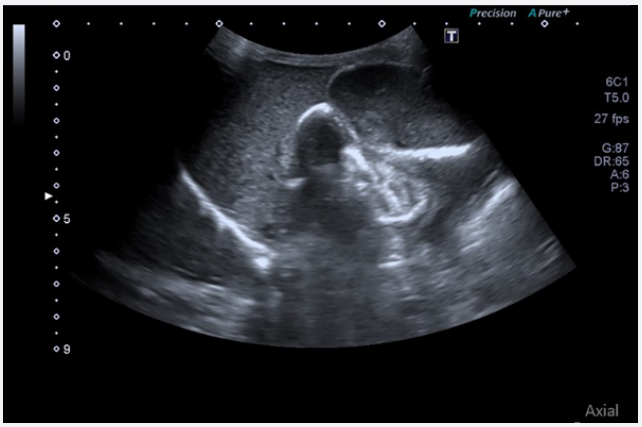

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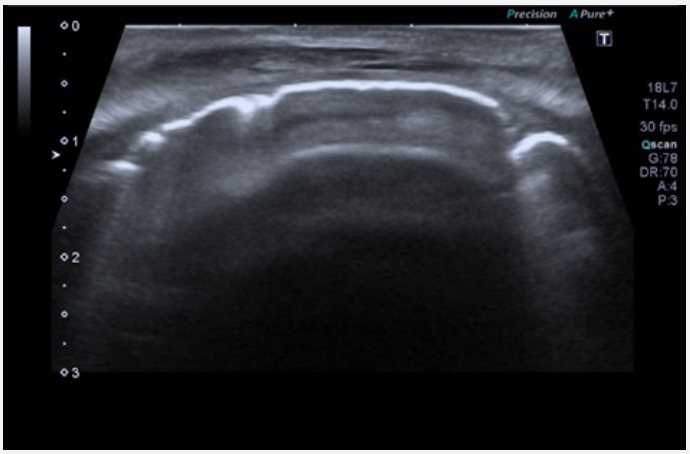

fig 3.

Patient monitoring showed a respiratory rate of 54/min, SpO2 of 92% without any oxygen input; also, a moderate degree of subcostal retractions was present. Because of the low hemoglobin levels and in order to complete the pre-operative preparation, the patient was administered 60 ml of Rh 0 Negative blood.

The surgical procedure took place 4 hours after admission, with a proper preoperative preparation beforehand. The access

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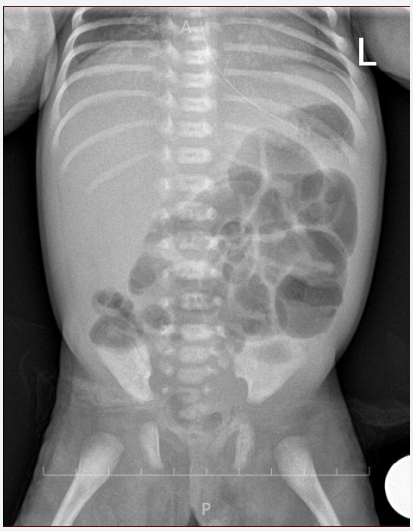
incision was represented by a median xipho-umbilical laparotomy that was ulteriorly extended downward under the umbilicus. The abdominal wall was thickened and edematous. After the umbilical vein was identified, ligated and sectioned, the peritoneal cavity was opened and the inspection showed ileal bowel loops that were necrotic, adherent and dilated (diameter of approximately 3 cm). The bowel wall was thickened and friable and when opened, it contained a large amount of hemorrhagic liquid mixed with meconium (cca. 600 ml) (Figure 5 and 6).



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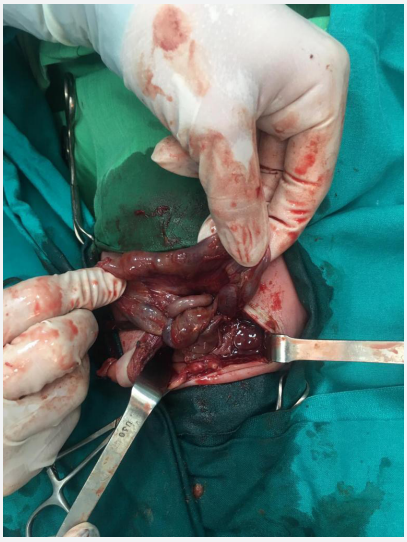

fig 5.

The necrotic bowel loops were identified with difficulty and, at inspection and manipulation, presented multiple calcifications. Also, the aspect was characteristic for midgut volvulus and malrotation.

After the isolation of the necrotic bowel loops, we performed an extended ileal resection (approximatively 30 cm) and ileo-ileal termino-terminal anastomosis (Figure 7).

Proximally to the anastomosis (approximatively 12 cm) the jejuno-ileal wall was thickened and edematous with a distended lumen, whilst the distal ileum, (approximatively 12-13 cm) containing discolored meconium and pellets, had a thin wall and a narrowed lumen specific for meconium ileus (Figure 8).

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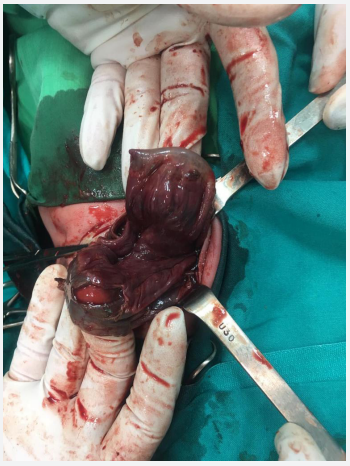

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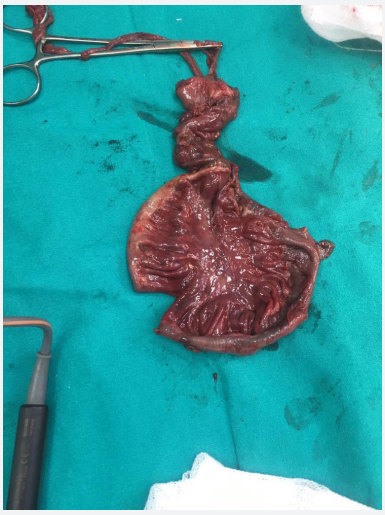

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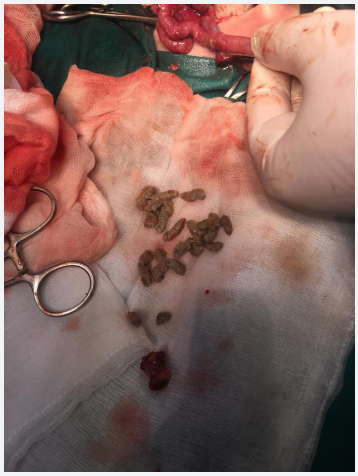

Fig. 8:

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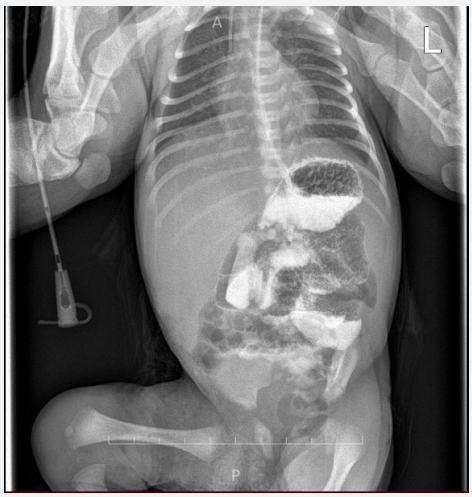
The colon had a normal length but the aspect was characteristic of a microcolon due to the lack of bowel content. A drain was put in place and the abdominal wall was sutured plane by plane. The patient was stable throughout the surgery and after the closure of the abdominal wall, a central venous catheter was inserted via the right internal jugular vein.

On the 3rd day after surgery oral contrast (Gastrografin) was administered with the purpose of examining the bowel loops (Figure 9 and 10).



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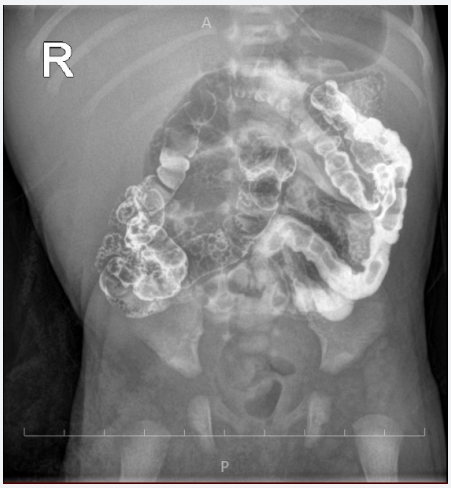

Fig. 10:

The postoperative evolution was favorable and the first normal stool was registered on the 6th day after the surgery.

After the oral feeding was started, the patient was transferred in a pediatric service, where cystic fibrosis was confirmed.

Case Report #2

A four-hour-old female patient was referred to our clinic after being born via cesarean section at another center. The patient was born prematurely (36 weeks of gestation). An ultrasonography performed at 34 weeks of gestations revealed splenomegaly and distended bowel loops.

As a result of these findings, fetal MRI was recommended, identifying distended bowel loops in the right hemiabdomen and peritoneal calcifications (Figure 11).

The weight at birth was 3500 g and the Apgar score was 9. Upon arrival, the clinical examination revealed a distended abdomen, altered general status, discreet dyspnea but without respiratory distress.

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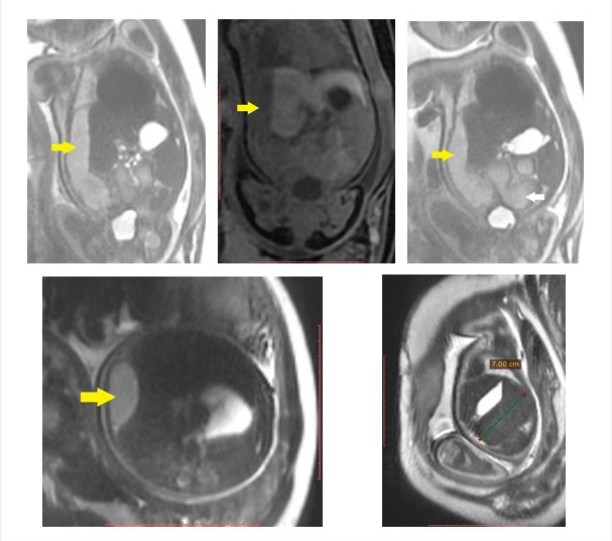

Fig. 11:

Biological evaluation suggested an intense inflammatory syndrome with a possible infectious cause, mild dehydration, cholestatic syndrome, hepato-cytolytic syndrome.

Abdominal echography showed a subhepatic collection that measured 40/11 mm, had a transonic impure content with a few intralesional septa and presented a mass effect onto the hepatic parenchyma. Also, the dimensions of the spleen were above normal for the patient's age.

Echocardiography revealed patent foramen ovale, patent ductus arteriosus and a minimal left pulmonary artery stenosis alongside with a first-grade pulmonary hypertension.

The patient was transferred in the intensive care unit for monitoring and preoperative treatment.

A xipho-umbilical laparotomy was performed. At exploration, the intestinal loops presented a brown color and extended adherences; intraoperatively we could diagnose the volvulus of the midgut. The spleen measured 4 cm underneath the costal ridge. The liver had a dark-grey color. Under the liver we found an irregular membranous mass, which had a yellow-green color, adherent to the lower hepatic margin and to the bowel loops, which had the characteristics of a ruptured subhepatic meconial pseudocyst. When attempting to mobilize the bowel loops in order to reduce the malrotation, meconial fluid passed through the multiple bowel perforations. Ileal resection was then performed (length of bowel resection: 15 cm; during the resection, some meconial plugs were also revealed) and subsequently termino-terminal ileo-ileal anastomosis.

The subhepatic pseudocyst was dissected carefully and excised. A drainage was put in place and the abdominal cavity was closed using a plain-by-plain suture.

As the suspicion of cystic fibrosis was high, we tested the patient and the result was non-conclusive. Histopathology report indicated that the specimen had a conjunctive-vascular structure, that resembles a cystic pseudo capsule and which contains meconium. Although the meconium was found to be rich in epithelial cells and desquamated keratin (lanugo), it did not contain biliary pigment. The lack of bile in the Meconium could suggest a barrier, which was another reason for further testing (genetic test for cystic fibrosis).

After surgery, a contrast study was performed using Gastrografin (Figure 12).

Postoperative care included daily CBC and biochemistry. The hepato-cytolytic syndrome was present until day 3 and during this period the patient presented sporadic bilious vomiting. The cholestatic syndrome appeared in day 5 and disappeared after discharge. The patient passed acholic meconium in the 5th day after the surgery. The inflammatory syndrome and total bilirubin normalized in day 10, but direct bilirubin maintained high values (ten times the normal value). Before discharging the patient, ultrasonography revealed perihepatic calcifications, a small liquid collection under the liver, homogenous and without impurities and another perisplenic collection. The bowel loops were found to be normal with peristaltic movements present. The patient was discharged in the 20th day after surgery, and referred to a pediatrician. On the one-month follow-up, the icteric syndrome disappeared and from the biological point of view, the direct bilirubin was close to normal values (under 2 times the normal value). Genetic tests did not confirm cystic fibrosis.

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Discussion

MP is a rare condition, with a reported frequency varying between 1:30000 [15] and 1:35000 [10]. However, as some cases which occur in utero can be clinically inapparent at birth, the actual frequency might be higher [10]. MP is often associated with cystic fibrosis, increasing the gravity and influencing the vital prognosis of these patients [7]. The incidence of cystic fibrosis in infants with MP has been reported to be between 15% and 40 %; furthermore, patients that present MP as a complication of cystic fibrosis show symptoms of varying severity, involving both the gastrointestinal tract and the pulmonary system [2]. The patient presented in the second case report, although diagnosed with cystic fibrosis, did not have pulmonary involvement pending admission, which played an important role in her post-operative outcome. The median gestational age of MP patients is similar: 35-36 weeks of gestation, as well as the median birth weight (around 2700g) [1,11,17], characteristics that were also found in our case reports. Although the survival rate has improved in the last decades to over 90%, the mortality remains at around 50% some reports [15]. As the diagnostic modalities evolve, the accuracy of prenatal diagnosis has improved and so the survival and morbidity rates could be greatly ameliorated if MP is detected antenatally and appropriately managed during the immediate postnatal period [15]. The literature displays a conflict of opinions regarding the prenatal diagnosis influencing the outcome. Ionescu et al. [18] detected a better prognosis in patients with antenatally diagnosed MP; also [4,19] some studies have described the relationship between prenatal US findings and postnatal outcomes. However, Chen et al. finds no statistically significant change in outcomes between prenatally and postnatally diagnosed MP [15]. The prognosis depends on the pathology having caused the perforation [20]: MP without gastrointestinal tract abnormalities generally has a favorable outcome, but because of bowel atresia requires postnatal surgery, it is important to identify the presence of such abnormalities (MRI) [4].

MP can be simple or complex. The sole presence of intraabdominal calcifications indicates simple MP, whereas an association with ascites, pseudocyst or bowel dilatation constitutes the complex variety [10]. Zangheri et al. created a classification system (Table 1) based on the 3rd trimester US findings (which were related to the perinatal outcome) that resembles Dewan's concept, grade 0 being the equivalent of simple MP and grades I, II, III being the equivalent of complex MP.

Furthermore, Saleh et al. demonstrated that all grades above I have the highest probability of urgent neonatal surgery [21]. However, some reported a case series describing MP with fetal ascites and intraabdominal calcifications (grade II a) which was followed by complete resolution in utero [22,23].

Among the risk factors that also influence the outcome of these patients, Chandrasekaran el al. includes: viral infections, gastrointestinal malformations causing either bowel or biliary atresia, hemochromatosis, cystic fibrosis [24].

The most common postnatal manifestations of MP include abdominal distension (77,8%), respiratory distress (44,4%), delayed passage of meconium (27,8%), bilious aspirate (16,7%), vomiting (5,6%) [15,17]. Although meconium is aseptic, bacterial overgrowth occurs and worsens the prognosis if gastrointestinal tract perforation persists after birth [15]. Ping et al. in his study of 18 MP patients showed that 60% presented respiratory distress postnatally, which, the study says, is more likely a consequence of diaphragmatic splinting from the ascites rather than due to the respiratory distress syndrome [17].

US is the most widely used primary tool for the diagnosis of MP [1,25]. The characteristic US findings in the prenatal MP diagnosis are: fetal ascites (54%-57%), dilated bowel loops (27%-29%), intraabdominal calcifications, echogenic bowel, polyhydramnios (64%-71%) and pseudocysts [10,15]. Knowing that polyhydramnios is associated with MP in 25 % to 50 % of cases [1], it becomes clear that US is essential. Furthermore, up to 86% of MP cases may have intraabdominal calcifications that can be detected as early as 18 weeks of gestation. These calcifications are characteristic of MP and usually appear as linear or plaque-like lesions in the pelvis, peritoneum and scrotum, and may even appear on the undersurface of the liver [10] as was the case of our second patient. When the calcifications are isolated, the neonatal outcome is usually favorable and surgical intervention is not required.

US can have some inevitable disadvantages: small field of view, low image contrast, operator dependence [4,26-28]. For this reason, other studies report a large variability – 0% - 94% [3,29,30] in finding calcifications, mainly because they take time to form and only large aggregated calcific plaques can demonstrate acoustic shadowing. If the calcifications are finely distributed in the peritoneal cavity, they remain undetected by the US, or appear just as hyperechoic lines or sports without acoustic shadow [2].

The "snowstorm" sign is also described in patients with MP and is caused by the echogenic character of the meconium in the sterile peritoneal fluid. Features such as microcolon, lumen obstruction with meconium pellets, as was the case of our second patient – image x) and pseudocyst formation can be seen on water soluble contrast studies [10].

Although US findings are not MP-specific, preparations for the presence of possible disorders, including cesarean section delivery and consultation with a pediatric surgeon, have made management easier [31].

Recently, it was reported that MRI can afford higher prenatal diagnostic accuracy of MP (57%) than US (42%) [1,2,4]. Because of the low sensitivity of prenatal MRI in detecting calcifications, MP can be diagnosed by using findings of meconium ascites,

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meconium pseudocysts and dilated bowel loops on MRI. In diagnosing fetal bowel abnormalities, it is important to notice whether or not meconium is present in the bowel, and MRI is more sensitive than US for this purpose. Also, in fetuses with bowel atresia, it is more useful to assess disease entities by fetal MRI; research has shown that us can detect only the dilated loop proximal to the first atresia, whereas MRI can show whether the post-atretic small bowel is also abnormal, thus providing more accurate findings than US [4].

Abdominal radiography is another useful tool in the diagnostic process of MP. It could be able to detect fine calcific spots [2] that elude US techniques. In the case of healed intestinal perforation and asymptomatic MP patients, multiple sites of calcification can show up in the scrotal, abdominal or thoracic cavities in postnatal X-ray studies [15]. In 2009, Tsai et al. revealed that the plain abdominal film showed calcifications in 4 out of 10 patients. In our case reports, both patients demonstrated these calcifications on the abdominal radiography [13].

Currently, resection and primary anastomosis is the preferred operative method [15]. Miyake et al. suggests primary anastomosis instead of multistep operation in cases with MP, except in neonates with very low birth weights because of their vulnerability. Even though initial operative time was significantly longer in patients who underwent primary anastomosis, times of initiation of enteral feeding and discharge (length of hospital stay) was significantly shorter Miyake et al. demonstrate [31]. In spite of this, the mortality and complication rates were similar in patients who underwent primary anastomosis and multistep operation. Nam et al. also reported that they preferred primary resection and anastomosis of the intestinal segment involved [11].

In some cases, when the newborn's condition is too critical to allow surgical exploration and viscerolysis, peritoneal drainage or stoma creation is favored for mitigating a systemic inflammatory response. Definitive surgery is subsequently performed after the newborn has stabilized [15].

Uncomplicated MP may be managed conservatively, under close monitoring. However, if signs of a surgical abdomen (severe metabolic acidosis, hemodynamic instability, pneumoperitoneum or other non-reassuring signs) which indicate sepsis or extensive bowel necrosis are observed, any delay can be catastrophic [15].

Fetal shunt placement can be considered for massive ascites and MP, which are associated with a risk of pulmonary hypoplasia. However few reports have described fetal shunt operations for MP [23]. Won et al. described 3 patients with MP who underwent a fetal shunt operation [32]. Although the perinatal survival rate in neonates with MP and massive ascites is low, these patients can survive after the fetal shunt operation and early postnatal surgical intervention, including ileostomy and primary repair of the small bowel [23].

Shyu et al. shared the experience of 17 cases managed by intrauterine therapy (US guided aspiration and repeat paracentesis) and found persistent ascites, pseudocyst or dilated bowel loop were correlated with the need of postnatal surgery (sensitivity rate of 92%) [23,29]. In addition, persistent ascites and postnatal persistent pulmonary hypertension were associated with neonatal mortality [23].

It is reported that early detection is not associated with poor neonatal outcomes therefore, we should avoid selective termination, except those accompanied with severe malformation and uncorrected genetic mutations which could be diagnosed by advanced technology, such as conventional cytogenetic or modern genetic study (noninvasive prenatal testing and high-resolution imaging systems) [23].

Conclusion

Close collaboration between neonatologists and pediatric surgeons is essential for the timely diagnosis and prompt management of MP. Even if a two-stage operation is indicated for complicated meconium peritonitis, performing primary anastomosis has good results, with shorter time of recovery and low morbidity. The surgical technique decision should rely on the general status of the patient and surgeon's experience.

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