Postnatal Treatment in Antenatally Diagnosed Meconium Peritonitis

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Abstract

Meconium peritonitis is a rare prenatal disease with an increased rate of morbidity and mortality in the neonatal period. Distinctive features revealed by prenatal and postnatal ultrasound may be present: abdominal calcifications, ascites, polyhydramnios, meconium pseudocyst, echogenic mass and dilated bowel or intestinal obstruction. Establishing clear postnatal treatment and prognosis is difficult because of the heterogeneity of the results obtained by ultrasound. The aim of the study is to determine how prenatal diagnosis of meconium peritonitis is associated with perinatal management and further evolution. Clinical results are different depending on the presence of antenatal diagnosis of meconium peritonitis and its form, which can be mild or severe. Surgical treatment and management of meconium peritonitis depend on the clinical presentation of the newborn. Meconium peritonitis diagnosed prenatally differs from that of the newborn, not only concerning the mortality rates but also through reduced morbidity and overall better prognosis.

Key words: meconium peritonitis, neonatal surgery, prenatal diagnosis

Introduction

Meconium peritonitis is defined as a localized or generalized peritonitis, aseptic, chemical or foreign body, due to leakage of meconium into the peritoneal cavity correlated with antenatal perforation of the digestive tract. Meconium, in the peritoneal cavity, may or may not be calcified. The syndrome was first described by Morgagni in 1761, and the first surgical correction...
was performed successfully in 1943, by Agerty.

Perforation is usually caused by obstruction induced by meconium ileus, intestinal atresia, stenosis, volvulus, internal hernia, congenital peritoneal bands, intussusception, gastrochisis, Hirschsprung disease (1).

Meconium peritonitis associated with cystic fibrosis was classified as a complication of meconium ileus.

In rare cases, chemical peritonitis heals spontaneously without any clinical manifestations. Attention to this pathology was drawn by findings presented as calcifications, intraperitoneal, inguinal or scrotal masses (2, 3).

The incidence of meconium peritonitis is about 1:30,000 (4). Due to area sealing of intestinal perforation and spontaneous regression of the inflammatory process without neonatal clinical manifestations, the incidence is probably underestimated. Perinatal morbidity and mortality reached 80 % in underdeveloped countries. Early recognition of the etiology, pathophysiology, and a specific perinatal management represent the requirements necessary for optimizing the postnatal results.

Continued progress in prenatal diagnostic procedures and providing neonatal intensive care have resulted in a 10% decrease in mortality in developed countries (5).

Etiopathogenesis

Three pathological types of meconium peritonitis can be distinguished depending on the degree of the inflammatory response as described in 1966, by W. S. Lorimer, and G. Ellis: fibroadhesive, cystic and generalized (6). Fibroadhesive meconium peritonitis is the most common and results from intense fibroblastic reaction due to chemical peritonitis, caused by digestive enzymes. This type of meconium peritonitis produces obstruction by fibroadhesive bands and the place of perforation is usually covered, sealed. The cyst type occurs when the perforation site is not completely closed and thus form a thick-walled cyst, attached to the intestinal loops preventing the communication with the rest of bowel perforation. Generalized meconium peritonitis is characterized by diffuse bowel thickening of the affected segment, peritoneal fibrosis and calcium deposits. This type closely matches generalized peritonitis. Meconium spreads in the peritoneal cavity and this occurs before birth or shortly thereafter (7).

Associated malformations

Cystic fibrosis is associated with meconium ileus and succeeding meconium peritonitis in approximately 15% of cases. Newer data analyzing cystic fibrosis transconductance receptor (CFTR) suggests that the association with meconium ileus and peritonitis is higher. Characteristic sonographic findings in the second quarter and early third quarter include intense abdominal echogenic masses. In the third quarter, dilated bowel loops can be observed. In 70 % of cases, it is possible the testing in fetus for the DF508 cystic fibrosis mutation. Neonatal investigations should include repeated sweat tests to measure quantitative chloride in sweat for diagnosis of cystic fibrosis (8).

In 25-50 % cases of meconium peritonitis, polyhydramnios is present and is attributed to difficulties in swallowing, as a result of the poor intestinal peristalsis. In rare cases, fetal hydrops may be present.

Antenatal diagnosis

Antenatal diagnosis should include testing for congenital anatomical or structural abnormalities, cystic fibrosis, chromosomal abnormalities, chemical and histological analysis of fetal peritoneal fluid obtained by paracentesis (9). The results of this investigations are very important in establishing an obstetrical and perinatal management of the patient.

Antenatal investigations, consisting of fetal blood sampling should exclude cystic fibrosis by DNA analysis, diagnose chromosomal abnormalities, and exclude congenital infections by haematological, immunological and hepatic fetal investigation. Paracentesis and histological analysis of aspirated fluid offer more accurate information about fetal ascites and bowel perforation.

Antenatal and postnatal ultrasounds are the first intention imaging investigations to diagnose meconium peritonitis. Characteristic signs revealed for meconium peritonitis are intraabdominal calcifications, fetal ascites, polyhydramnios, pseudocysts and dilated intestinal loops (Fig. 1, Fig. 2).

Ultrasound examination is used to exclude other calcified abdominal masses (7, 10).

The use of ultrasound for identification meconium peritonitis in utero has significant implications for obstetric and neonatal care. In the case of meconium peritonitis, the incidence of prematurity is 20-30 % and 10-20 % for polyhydramnios.

The meconium peritonitis prenatally diagnosed differs from that of the newborn, not only by the reduced rate of mortality and morbidity, but also through better overall prognosis. In 50 % of cases of complex meconium peritonitis associated with abdominal calcifications, dilated intestinal loops, meconium cyst, ascites and polyhydramnios, surgery is

Figure 1. Prenatal ultrasound – intraabdominal calcifications with meconium peritonitis confirmed at birth
necessary, being important that these fetuses to be born in a tertiary center. Instead, a simple meconium peritonitis requires only monitoring, the incidence of complications is low, and prognosis is favorable (7).

**Fetal MRI**

Although, prenatal ultrasound is widely used to diagnose meconium peritonitis, recent studies have reported that magnetic resonance imaging provides a more precise diagnosis of meconium peritonitis compared to prenatal ultrasound (57.1% vs. 42%) (9, 10).

The advantage of fetal MRI consists in diagnosis of other associated congenital malformations.

**Postnatal diagnosis**

Postnatal diagnosis of meconium peritonitis is established after correlation of the clinical presentation of intestinal obstruction with abdominal and / or scrotum radiographs and ultrasound (Fig. 4).

The suggestive symptomatology for diagnosis of meconium peritonitis is installed in the first 1-2 days after birth.

Clinical examination of the newborn with meconium peritonitis revealed a distended abdomen, in tension, edematous wall, congestive, shiny teguments and visible blood circulation. The abdomen is characterized by generalized distension and a palpable tumor mass. Abdominal distention frequently results in compromised breath amplitude (1).

Hypovolemia may be secondary to loss of the abdominal space. In evolution appear bilious or intestinal vomiting (11).

Abdominal plain X-ray reveals the presence of calcifications in the peritoneal cavity and peritoneal recesses (Fig. 5). Occasionally, the calcifications may extend to the scrotum by patent vaginal process and presents as a firm scrotal mass. Calcifications are confirmed by ultrasound, appearing as hyperechogenic images with posterior acoustic shadowing. Less commonly, calcifications may extend into the thorax through normal anatomical communications. Clustered calcifications, especially if they are curved, may indicate the presence of a meconium pseudocyst, which represent a perforation with calcified edge.

If the patient is asymptomatic, calcifications can be discovered incidentally and does not require further investigation. If bowel obstruction is present, we can perform Barium esophageal transit or Barium enema to determine the cause of intestinal perforation in utero.
Differential diagnosis

Liver calcifications may occur in infections with cytomegalovirus and parvovirus. In case of hepatoblastoma, for establishing the diagnosis it is recommended the dosage level of alpha-fetoprotein (AFP).

Intestinal intraluminal calcifications are present in the case of multiple intestinal atresia, complete atresia of the colon, Hirschsprung’s disease and, in this case, is associated with high obstruction and calcifications are hollow. Calcified meconium occurs if fetal meconium mixes with urine. This can happen to male fetuses with high anorectal malformations or female with cloacal anomalies (12).

Antenatal and postnatal management. Surgical treatment

A key element in the further management consists in excluding chromosomal malformations, congenital infections and cystic fibrosis. Early diagnosis can change parent’s attitude towards termination of pregnancy (13).

It is no need for induction of labor if meconium peritonitis resolves spontaneously.

Postnatal, observation of the intestinal peristalsis and simple abdominal radiography should alert the pediatrician. Sometimes, surgical exploration might be necessary.

If it is observed a progressive deterioration of the fetus, with increasing amount of ascites, premature birth in a tertiary care center should be taken into account. In such cases, surgery is required immediately after birth. Regarding the birth, it hasn’t been proved that caesarean section would improve neonatal outcomes (14).

Management and surgical strategy of a patient with meconium peritonitis rely on the clinical presentation and the overall condition of the newborn. Surgery is necessary when signs of intestinal obstruction and/or sepsis are present. The presence of intraperitoneal calcifications is not an indication for surgery. It is preferable that the surgery include viscerolysis, resection of affected intestinal segment and primary termino-terminal anastomosis (10, 15).

Performing definitive surgery can be difficult initially and it is a significant risk of bleeding during viscerolysis of the inflamed and edematous peritoneal surfaces. Also, the intervention is complicated if there is severe intestinal distension or if bowel viability is uncertain. In these cases, is undertaken an enterostomy or draining the peritoneal cavity or the encysted meconium collection, definitive surgery taking place at a later stage (10).

Prognosis

The cases of meconium peritonitis diagnosed antenatal most often have a good prognosis, perinatal death rate being low. Exclusion of infectious etiology or chromosomal abnormalities can lead to a perinatal survival rate of over 80%. Chemical peritonitis can permanently cure bowel perforation.

The presence of cystic fibrosis and pancreatic insufficiency leading to digestive disorders, many respiratory infections and chronic lung disease strongly affect the long term prognosis.

Therefore, investigations should include prenatal DNA analysis of DF508 mutation located on chromosome 7 and repeated tests to measure sweat chloride levels in sweat in the neonatal period.

Material and Methods

This is a retrospective study of all patients with meconium peritonitis treated in the pediatric surgery department and neonatal intensive care of Emergency Hospital for Children “M.S. Curie “in 2004 - 2013.

The following parameters were recorded: age at presentation, place of birth, sex, antenatal care, clinical presentation and findings from investigations. It was also taken into account the intraoperative findings, operative techniques performed, postoperative complications, treatment outcomes and postoperative monitoring.

The aim of the study is to determine how prenatal diagnosis of meconium peritonitis influence perinatal management and outcomes.

Antenatal diagnosis of meconium peritonitis was based on ultrasound findings: abdominal calcifications, meconium pseudocyst, ascites, hyperechogenic bowel or intestinal loops dilatation. Antenatal and postnatal results were compared with the etiology, intraoperative findings and results.

Assessment of patients was performed using a scoring
system described by Zangheri. Score 0 describes the presence of isolated calcification, score 1A includes calcifications associated with ascites, score 1B includes calcifications associated with meconium pseudocyst, score 1C corresponds calcifications and dilated intestinal loops, score 2 is based upon the presence of calcifications and two of the events described above and score 3 embraces all. The smallest probability (0 %) for surgery corresponds to isolated cases of calcification (score 0). In other patients with a score between 1 to 3, the probability of surgery intervention increased to over 50 % (3).

The postnatal diagnosis includes clinical examination, abdominal ultrasound, abdominal radiography and screening for cystic fibrosis (screening for the most common mutations in genes and sweat test), screening for congenital infection with herpes simplex virus, parvovirus B19, toxoplasmosis and cytomegalovirus (13).

**Results**

For this study were taken into account 13 patients (8 males - 62 % and 5 females - 38 %) with gestational age between 18 and 38 weeks. Patients were divided in two groups. In the first group, represented by 5 (38 %) of the 13 fetuses, the initial prenatal ultrasound findings, that characterizes meconium peritonitis, disappeared during pregnancy. In this group of patients, prenatal ultrasound revealed intraabdominal calcifications (5/5), moderate ascites (3/5), hyperechogenic bowel (2/5) and polyhydramnios (5). These fetuses were born without any complications, with a mean gestational age of 40 weeks (range 36-41 weeks) and a mean birth weight of 2850 g (range 2240 g - 3350 g). The abdominal ultrasound performed postnatal, physical examination of the abdomen and other investigations in these patients were without pathological changes and surgical intervention was not required. Screening for congenital infections and cystic fibrosis was negative for all five patients. This group was excluded from the study because the postnatal diagnosis of meconium peritonitis was disproved.

The second group consists of the remaining 8 (62%) patients. In 3 of them, ultrasound performed during pregnancy revealed the presence of characteristic signs of meconium peritonitis. The most common sonographic findings were dilated bowel (3/3), polyhydramnios (3/3), abdominal calcifications (2/3), ascites (2/3), and meconium pseudocyst (1/3) (Fig. 6). Two of these fetuses were delivered by caesarean section, one was premature with a gestational age of 35 weeks and a birth weight of 2300g, and the other was born at 37 weeks, weighing 3000g. The 3rd patient with diagnosis of meconium peritonitis established prenatal was spontaneous vaginal delivered at 38 weeks gestation, weighing 3240g. In all these infants, surgical intervention was performed within 48 hours due to ileus. In the first two cases, there were no perioperative complications, but in the case of the third patient death occurred 46 days after the first surgery. In this patient, sweat test confirmed the presence of cystic fibrosis. At the first surgical intervention was found intraoperatively jejunal atresia due to meconium peritonitis with antenatal intestinal perforation. It was performed viscerolysis, segmental resection of the jejunum and termino-terminal jejuno-jejunal anastomosis (Fig. 7). Postoperative evolution was unfavorable, the infant developed progressive respiratory failure with right pulmonary atelectasis and without resume of intestinal transit. At 12 days after surgery, due to a marked abdominal distension, was necessary surgical reintervention with viscerolysis and anastomosis reestablishment. The evolution was still bad, whit no resume of intestinal transit, bing present a significant quantity of bilious gastric residue, which determined the surgical reintervention with jejunostomy. The evolution was unfavorable, with respiratory and general status worsening, followed by exitus due to cardiac arrest.

The diagnosis of meconium peritonitis was established postnatal in 5 of the 8 patients. Four of these fetuses were born premature, 1 of them by caesarean section, and the remaining 3 were spontaneous vaginal delivered. Mean gestational age of the 5 patients was 34 weeks (range 30-38 weeks) and mean birth weight was 2258g (1390g - 3100g range). In two of these patients, exitus occurs through cardiorespiratory arrest.

Exploratory laparotomy was performed in all 8 patients and it was observed generalized meconium peritonitis in 6 of the 8 patients, cystic form in 1 patient (Fig. 8) and fibroadhesive meconium peritonitis in 1 case. The most frequent site of intestinal perforation was the terminal ileum (4 patients) (Fig. 9), jejunum (3 patients), cecum (1 patient) and transverse colon (1 patient). The procedures performed were segmental resection and end to end anastomosis in 5 patients, surgical reinterven-

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**Figure 6.** Meconium pseudocyst – abdominal ultrasound – left, abdominal X-ray – right
tion with jejunostomy, ileostomy and colostomy being necessary in 3 of the 5 patients. In the remaining 3 patients, was performed cecostoma (1 case) and ileostomy (2 cases).

Comparing patients who required surgical intervention, showed the prenatal diagnosis of bowel dilatation as the only significant indicator for postnatal surgery. Depending on the prenatal scoring system described by Zangheri, 6 patients had score 2 and 2 patients had score 3; all of them required surgery in the neonatal period.

**Discussions**

Meconium peritonitis usually occurs due to intrauterine gastrointestinal perforation. In the beginning, it is a sterile chemical peritonitis, but if gastrointestinal tract perforation persists after birth, bacterial overgrowth occurs and worsens the prognosis. Perforation usually occurs due to intrauterine obstruction or volvulus. Often, the main problem represents the meconium ileus, a consequence of cystic fibrosis (8). If perforation heals intrauterine, calcified extruded meconium may be detected incidentally and in these cases are not necessary further investigations or surgery. However, in cases of active peritonitis with intestinal obstruction and/or pneumoperitoneum, surgery is required immediately after birth.

Meconium formation begins in the third month of gestation and is composed of amniotic fluid containing water, desquamated squamous cells of the skin, bile salts. Besides amniotic fluid, into the composition of meconium enters uric acid, bile pigment pancreatitis, mucus, intestinal enzyme secretions, cholesterol, inorganic salts and sugars. Lipases and bile acids present in the peritoneal cavity may induce the intense chemical peritonitis. (12)

Extruded meconium undergoes rapid saponification and calcification after reaching the fetal peritoneal cavity, its presence causing bowel perforation.

Meconium peritonitis diagnosis relies on imaging that can reveal the presence of intraperitoneal calcifications in the newborn. The extruded meconium can be or not calcified, and when there were no calcification, X-rays suggest the presence of fluid in the abdomen. When calcifications are seen, meconium peritonitis diagnosis is established. These calcifications can be amorphous, irregular, curved, the latter suggesting the location of the cyst.

On the pathogenesis, a decrease in blood flow to the mesentery can result in mucosal necrosis and subsequent bowel obstruction. Any further decrease in mesenteric blood flow can cause necrosis and perforation of the intestinal wall (10). Perforation usually occurs in the small intestine but can occur
in rare cases at other levels of the gastrointestinal tract (colon).
Maternal polyhydramnios can be an early sign of bowel disease and it is present in 25% to 50% cases of meconium peritonitis.

Meconium peritonitis may have a wide spectrum of clinical manifestations, ranging from apparently healthy newborn with normal gastrointestinal function, to a newborn with severe physiological disorders and intestinal obstruction.

The cases in which the infant is apparently healthy, with normal function of the gastrointestinal tract, most likely experienced in utero intestinal perforation that healed spontaneously, being imagistic visible only intraperitoneal calcifications.

These children do not usually require surgery. Giant cystic meconium peritonitis is the opposite. Massive intestinal necrosis in utero produces a cyst within which bile accumulates. Often, the intestine viability is reduced. The results are poor in these cases, and the surviving have a high risk of short bowel syndrome. (5).

Prognosis and natural evolution of meconium peritonitis, when it is diagnosed in the fetal period, are different from that diagnosed postnatally. If at the intraperitoneal fetal ultrasound are present only calcifications without bowel dilatation, polyhydramnios, ascites or pseudocyst, the prognosis is favorable. These features constitute simple meconium peritonitis. If all the features listed above are associated, the prognosis is bad and need for surgical intervention increase.

Neonates with meconium peritonitis often have cystic fibrosis (7%-40%), requiring analyzes of chromosome and sweat test to confirm the diagnosis (8).

Pneumoperitoneum seen on abdominal radiography requires further exploration. Intraoperative we should expect to find thick inflammatory tissue, adherent to intraperitoneal viscera. The viscerolysis of the intestine should be done correctly. Ideally, the perforation should be located distal to be able to make a stoma proximal to the site.

In some cases it may be difficult to delineate viable bowel to allow a stomy, when the general condition of the newborn can not allow a surgical exploration and viscerolysis. In these cases, peritoneal drainage favors the mitigation of systemic inflammatory response and allow "second look" when the newborn is stabilised.

If meconium peritonitis is diagnosed antenataly, pediatricians should monitor any signs of intestinal obstruction in the neonatal period. In cases of infants with intestinal obstruction, surgery performed within 24 hours can significantly improve their results. However, bear in mind that asymptomatic infants may develop intestinal obstruction later in childhood because of peritoneal adhesions.

Conclusions

Prenatally diagnosed meconium peritonitis differs from neonatal meconium peritonitis not only by mortality rate, but also through reduced morbidity and overall better prognosis. Natural evolution of simple meconium peritonitis without being associated with bowel dilatation, ascites and polyhydramnios only requires tracking, knowing the low incidence of complications and good prognosis.

According score described by Zangheri, the need for the emergency surgery in the neonatal period is higher in patients with a score ≥1, therefore, they should be born in a tertiary center.

Currently, only antenatal ultrasound monitoring allows an adequate surgical approach with intensive care support and favorable prognosis.

The polymorphism of condition requires a specific approach to each case and implies discussion with parents to present the vital and functional prognosis of the child.

Author's contribution

All the authors have the same contribution.

References