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INTRAUTERINE MECONIUM PERITONITIS (LITERATURE REVIEW)

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ABSTRACT

Intrauterine meconium peritonitis (MP) is a rare but severe neonatal surgical condition caused by fetal intestinal perforation with meconium leakage into the peritoneal cavity. Despite advances in prenatal imaging and surgery, diagnostic and therapeutic challenges remain. To summarize current knowledge on the etiology, pathogenesis, classification, diagnosis, and treatment of intrauterine meconium peritonitis. A narrative literature review was conducted based on historical and contemporary publications describing the diagnosis and management of MP. Sources include autopsy reports, surgical case studies, and radiologic findings published between 1761 and 2024. MP is classified into fibroadhesive, cystic, and generalized forms. The fibroadhesive type often has a benign course, while cystic and generalized types require urgent surgery. Ultrasound remains the key prenatal diagnostic tool, identifying calcifications, ascites, or pseudocysts. Early surgery within 24 hours of birth improves survival. Two-stage management with initial enterostomy followed by reconstruction is considered the most effective. Meconium peritonitis, though uncommon, remains a clinically significant condition requiring timely prenatal recognition and multidisciplinary management. Advances in imaging and neonatal intensive care have reduced mortality, but further research is needed to standardize surgical tactics and improve outcomes.

Introduction. In recent years, increasing attention has been paid to the study of intrauterine infection (IUI) as a pathogenetic risk factor for perinatal and obstetric complications. According to WHO data, infectious diseases are detected in 70% of hospitalized premature newborns [4,7].

Meconium peritonitis (MP) is a rare and still insufficiently studied disease in newborns. According to various authors, the incidence of the disease ranges from 0.29 to

0.33 per 10,000 live births, with a noted tendency toward an increase in the number of newborns with this pathology. The relevance of the problem of MP in newborns is primarily due to the difficulties in diagnosing this condition, as well as the absence of a unified approach to therapeutic tactics, including optimal methods of surgical management and postoperative rehabilitation of children with this pathology [5–6,8].

Intrauterine peritonitis represents a localized or generalized inflammation of the fetal peritoneum in response to prenatal intestinal perforation and the leakage of meconium into the abdominal cavity. Most often, it is aseptic in nature and is referred to as meconium peritonitis [11]. This syndrome was first described in 1761 by Morgagni, who, based on autopsy material, suggested that the cause of MP was intrauterine intestinal perforation [13]. In 1838, J. Simpson published autopsy data on 25 fetuses and newborns who died within the first days of life, in whom peritonitis was found at autopsy as a result of intrauterine intestinal perforation [3,15].

In subsequent years, studies expanded understanding of this pathology, which develops against the background of congenital malformations of the gastrointestinal tract (GIT). At that time, mortality reached 100%, and all reports were based on autopsy findings [10]. The next stage in MP research began in the 1940s, when anesthetic management improved and the antibiotic era began. In 1943, H. Agerty performed the first successful operation on a child with MP [10]. By 1952, eight successful operations had been reported [16]. In 1953, J. Bendel and J. Michel [11] defined MP as:

“Meconium peritonitis is a non-microbial, chemical peritonitis that occurs intrauterinely or in the early postnatal period as a result of a pathological communication between the intestinal lumen and the abdominal cavity.”

In 1966, W. Lorimer et al. [26] proposed a classification of MP into three types: fibroadhesive, cystic, and generalized. Most authors still adhere to this classification. Some researchers later suggested adding two more forms — self-healing and microscopic [23,33], though this remains debatable.

Despite advances in surgery, mortality in MP remained high (>50%) [32] due to late diagnosis and treatment outside specialized centers. Since the 1980s, the introduction of sonography significantly improved prenatal diagnosis. Modern imaging, refined surgical techniques, and improved neonatal care have reduced MP mortality from 50% to 11% [25].

Methods. This paper is a narrative literature review based on the analysis of national and international scientific publications, clinical observations, and historical data describing intrauterine meconium peritonitis. The sources include works from 1761 to modern publications, referenced according to the original numbering system (1–43).

Data were extracted from peer-reviewed journals, surgical case reports, and radiological studies, emphasizing prenatal diagnosis, classification, and surgical management outcomes of meconium peritonitis.

Results. According to R. Payne et al. [32], in 1983 the incidence of MP in the United States was 1 per 35,000 live births. By 1990, German authors reported an increase to 1 per 10,000. Since some cases complete their course intrauterinely without postnatal symptoms, the true incidence is likely higher. In Russia, there are no precise statistics; MP

was officially recognized as a separate nosological entity in ICD-10 (code P78.0) only in 1989.

MP results from intrauterine intestinal perforation, leading to meconium leakage into the peritoneal cavity. Two mechanisms are described:

1. Perforation due to obstruction of an intestinal segment.
2. Perforation without obstruction.

Obstruction may be intraluminal (atresia, meconium plugs, volvulus, stenosis, intussusception, Hirschsprung's disease) or extraluminal (malrotation, compression by embryonic bands, tumors, or internal hernias) [28]. MP also occurs in 7.1–40% of children with cystic fibrosis [41].

Normally, meconium reaches the ileocecal junction by the 16th–17th gestational week and the rectum by the 20th [12,17]. Obstruction causes distension, ischemia, necrosis, and perforation, allowing meconium to spill into the abdominal cavity.

Idiopathic perforations — not associated with obstruction — are believed to result from mesenteric vascular compromise due to teratogens (alcohol, narcotics) or viral infections (parvovirus B19, CMV, hepatitis A) [34–39].

Three main forms are distinguished:

- Fibroadhesive MP: The most common and mild form, with intra-abdominal calcifications visible on prenatal ultrasound or postnatal radiography. Often asymptomatic, but may cause secondary atresia requiring surgery [14,27].
- Cystic MP: Occurs when the fibroplastic reaction is insufficient. Leads to pseudocyst formation — a cavity filled with liquefied meconium surrounded by necrotic bowel loops, sometimes calcified [1,18].
- Generalized MP: Meconium distributed throughout the abdomen, often due to perforation immediately before or during birth.

Cystic and generalized forms have the highest mortality. Early prenatal diagnosis significantly improves outcomes. Typical ultrasound findings include dilated intestinal loops, ascites, polyhydramnios, and calcifications [43].

G. Zangheri et al. [43] proposed a four-level prenatal severity scale based on ultrasound findings, ranging from isolated calcifications (grade 0) to the presence of all signs (grade 3).

Discussion. Prenatal differentiation of MP from non-immune hydrops and other cystic lesions (e.g., ovarian or urachal cysts, duplication cysts, teratomas) is crucial [33]. According to S. Nam et al. [28], prenatal diagnosis is possible in 92.7% of cases. MRI and CT are used only for differential diagnosis [16,36,40].

Delivery should take place in a center with neonatal surgery facilities, with timing determined by a multidisciplinary team. Premature delivery or cesarean section may be indicated if the abdomen is markedly enlarged or pseudocyst rupture risk exists [8,35].

Postnatally, the clinical picture ranges from asymptomatic (fibroadhesive) to severe peritonitis or intestinal obstruction (cystic/generalized). Diagnostic imaging confirms meconium ascites, pseudocysts, or free gas.

Surgical management depends on disease form and extent. For fibroadhesive MP with obstruction, adhesiolysis and resection with anastomosis are performed. For cystic

and generalized forms, emergency surgery within 24 hours is required. Early intervention reduces mortality from sepsis [33].

In extremely severe cases, decompressive laparocentesis can stabilize the newborn before surgery. Peritoneal drainage as an initial stage was first proposed by S. Ein et al. (1977) [20] and later supported by Tanaka et al. (1993) [37]. Some authors have even proposed intrauterine drainage, though its efficacy remains unproven [24].

For cystic MP, surgery includes adhesiolysis, partial cyst wall removal, and resection with intestinal stoma creation. In generalized MP, resection with enterostomy is recommended. Two-stage treatment (initial enterostomy with later reconstruction) provides lower perioperative mortality [9].

Postoperative complications include adhesive obstruction, intra-abdominal abscesses, and anastomotic leakage, while sepsis and short bowel syndrome are the most severe outcomes [12,28]. All patients require careful postoperative monitoring and nutritional support [16].

Conclusion. Meconium peritonitis remains a rare but severe neonatal surgical pathology. Despite advances in prenatal diagnosis and neonatal intensive care, many aspects of its pathogenesis and optimal surgical management remain controversial.

Future studies should focus on early diagnosis, standardized surgical protocols, and long-term follow-up of survivors.

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