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Prenatal diagnosis and postnatal management of meconium peritonitis

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Abstract

Background: Meconium peritonitis (MP) is a rare prenatal condition that leads to substantial neonatal morbidity and mortality.
Patients and methods: All patients between 1998 and 2006 referred for prenatal diagnosis were retrospectively analyzed for diagnosis of MP. Prenatal ultrasound findings were compared with postnatal etiology, intraoperative findings, and postnatal outcome of the patients.
Results: Antenatal MP was diagnosed in 14 fetuses between 18 and 38 weeks' gestation. The prenatal diagnosis of MP was confirmed by clinical and radiological findings in 8 (62%) of 13 infants born alive. All patients were delivered preterm between 33 and 36 weeks' gestation by cesarean section. Urgent neonatal surgery for treatment of bowel obstruction was required in all eight infants. Prenatal ultrasound diagnosis of bowel dilatation was the only variable found to be associated with the need for subsequent surgical intervention ($P = 0.02$).
Conclusions: Clinical outcome of MP diagnosed antenatally can be either mild or severe form. The underlying cause of severe MP is heterogeneous and neonatal surgery was always required.

Keywords: Meconium peritonitis; neonatal surgery; neonates; prenatal diagnosis.

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Introduction

Meconium peritonitis (MP) is defined by sterile chemical peritonitis due to prenatal small bowel perforation *in utero* [3, 9]. Meconium leakage into the abdominal cavity induces an inflammatory response by stimulating peritoneal macrophages followed by intestinal obstruction in several cases [14].

The estimated incidence of MP is 1:30,000 [9, 16]. MP leads to substantial perinatal morbidity and mortality as high as 80% in a third world healthcare setting [1, 3, 15]. Early recognition of the underlying etiology, pathophysiology as well as a specific perinatal management is prerequisite for optimizing postnatal outcome [5]. Continuing progress in prenatal diagnostic procedures and postnatal intensive care decrease mortality rates below 10% in some series [3, 19] and represent the current situation in Germany but were unsupported by previously published data.

The typical prenatal and postnatal ultrasound findings include abdominal calcification, ascites, polyhydramnion, meconium pseudocyst, echogenic masses and bowel dilatation or bowel obstruction. Due to the heterogeneous ultrasound findings clear definition of postnatal treatment options and prognosis is difficult [3, 14, 15].

The aim of our study was to determine how prenatal diagnosis of MP is associated with perinatal management and outcome, particularly with the prediction of neonatal surgery.

Methods

Medical records of 34,962 pregnant women referred for antenatal examination at the Department of Prenatal Medicine, and 3022 patients admitted between January 1998 and December 2006 to the tertiary NICU of the Department of Neonatology (both at the University Hospital of Bonn) were retrospectively screened for suspected or proven MP. Study was performed in accordance with the guidelines of the Bonn University Ethics Committee.

The diagnosis of MP was suspected prenatally based on the following ultrasound findings: intraabdominal calcifications, meconium pseudocyst, ascites, hyperechogenic bowel, or bowel dilatation. The prenatal and postnatal findings were compared with respect to etiology, the operative findings and the postnatal outcome.

Patients were scored according to the prenatal findings as previously described by Zangheri [19]. Briefly, score 0 describes isolated calcification, score 1A calcification associated with ascites, score 1B calcification associated with a pseudocyst, score

Table 1 Perinatal clinical data in patients with antenatal diagnosis of meconium peritonitis.

Case number	Prenatal ultrasound findings	Scoring [19]	Operative findings	Outcome	Etiology/ underlying disease
1	Intraabdominal calcification, polyhydramnion	0	–	Pregnancy was terminated because of severe associated fetal anomalies	No autopsy
2	Intraabdominal calcification, mild ascites, hyperechogenic bowel	1A	–	Well and alive	Unknown
3	Intraabdominal calcification, mild ascites	1A	–	Well and alive	Unknown
4	Intraabdominal calcification	0	–	Well and alive	Unknown
5	Intraabdominal calcification, polyhydramnion	0	–	Well and alive	Unknown
6	Intraabdominal calcification, mild ascites, hyperechogenic bowel	1A	–	Well and alive	Unknown
7	Intraabdominal calcification, ascites, hyperechogenic bowel, bowel dilatation, polyhydramnion	2	Meconium ileus with perforation of coecum, microcolon	Died (age of 1 year)	Anatomic cause/ hemochromatosis
8	Intraabdominal calcification, ascites, meconium pseudocyst	2	Perforation in transition from jejunum to ileum, fibrin coated intestine with thick intestinal wall, meconium pseudocyst, non-used-gut	Well and alive	Cholestasis of unknown origin
9	Intraabdominal calcification, ascites, meconium pseudocyst, hydrops fetalis	2	Perforation of distal ileum, meconium peritonitis, meconium pseudocyst, microcolon	Well and alive	Unknown
10	Intraabdominal calcification, ascites, bowel dilatation, hydrops fetalis	2	Malrotation, perforation of colon transversum, meconium peritonitis	Well and alive	Anatomic cause
11	Intraabdominal calcification, meconium pseudocyst, bowel dilatation	2	Malrotation with atresia of the proximal and middle part of jejunum as well as atresia of ileum	Well and alive	Anatomic cause
12	Intraabdominal calcification, ascites, polyhydramnion, bowel dilatation	2	Ileum atresia, intraabdominal calcification, meconium peritonitis	Well and alive	Anatomic cause
13	Intraabdominal calcification, meconium pseudocyst, severe bowel dilatation	2	Meconium peritonitis due to volvolus and perforation of the jejunum and secondary jejunum atresia	Well and alive	Anatomic cause/ cystic fibrosis
14	Intraabdominal calcification, ascites, meconium pseudocyst, bowel obstruction and dilatation	3	Meconium pseudocyst, jejunum atresia, meconium peritonitis	Failure to thrive, liver failure	Anatomic cause/ biliary atresia

Scoring [19]: 0 = isolated calcification, 1A = calcification associated with ascites, 1B = calcification associated with pseudocyst, 1C = calcification with bowel dilatation, 2 = calcification with two associated findings, 3 = all feature present.

1C calcification with bowel dilatation, score 2 calcification with two associated findings; in score 3 all feature are present.

Postnatal diagnosis included clinical examination, abdominal ultrasound examination and/or X-ray, histopathology and screening for cystic fibrosis by screening for the most common gene mutations and sweat chloride test and screening for congenital infections including herpes simplex virus, cytomegalovirus, parvovirus B19 and toxoplasmosis.

Statistical analysis was performed by SPSS 10.0 software package for Windows (SPSS Inc., Chicago, Illinois, USA). Prenatal ultrasound findings and the postnatal need for neonatal surgery were compared by using Fisher's exact test for dichotomous variables. $P < 0.05$ was considered statistically significant.

Results

Prenatal diagnosis of MP was suspected in 14 fetuses (10 males and 4 (29%) females) between 18 and 38 weeks' gestation.

Termination of pregnancy was performed in one patient with complex congenital malformations including Dandy-Walker-malformation, intraabdominal calcifications polyhydramnion at 26 weeks of gestation (Table 1). In 5 (38%) of the remaining 13 fetuses the initial prenatal ultrasound findings subsequently disappeared during gestation. The antenatal ultrasound findings of this group of patients were intraabdominal calcifications (5/5), moderate ascites (3/5), polyhydramnion (1/5) and hyperechogenic bowel (2/5). These patients were all delivered without any complication with a median gestational age of 40 weeks (range 37–41) and with a median birth weight of 2970 g (range 2640–3400 g). The abdominal ultrasound carried out in the postnatal period, physical examination of the abdomen and the further course in these patients were without pathological findings, and surgical interventions were not necessary. Screening for congenital infections and cystic fibrosis were negative in all five patients.

In 8 (62%) of the fetuses with suspected intrauterine MP pathological sonographic findings were detectable throughout pregnancy. The predominant ultrasound findings were intraabdominal calcification (4/8), ascites (6/8), meconium pseudocyst (5/8), bowel dilatation (6/8), hydrops fetalis (1/8), and polyhydramnion (1/8) (Tables 1 and 2). All these infants were born preterm by cesarean section at a median gestational age of 35 weeks (range 33–36) and with a median birth weight of 2480 g (range 1660–3730 g). All neonates required surgical intervention within 48 h because of ileus but no patient died of perioperative complications. The operative findings are described in detail in Table 1. MP was associated with cases of cystic fibrosis, biliary atresia, neonatal hemochromatosis and cholestasis of unknown origin. The outcome of the patients in need of surgery was very variable. Six of the eight patients with MP and neonatal surgery had an uneventful course on follow-up. The patient with hemochromatosis died at the age of one year and the patient with biliary atresia underwent a Kasai portoenterostomy with severe hepatic cirrhosis.

The comparison of the patients requiring surgical intervention (Table 2) demonstrates that prenatal diagnosis of fetal bowel dilatation was the only variable significantly indicator of postnatal surgical intervention ($P = 0.02$). Furthermore, the ultrasound finding of meconium pseudocyst was common in infants requiring surgery ($P = 0.07$) and never seen in patients who did not require neonatal surgery (Table 2). All other prenatal ultrasound findings (intraabdominal calcification, ascites, hyperechogenic bowel, hydrops fetalis, polyhydramnion) were found in both groups of patients (Table 2). According to the prenatal scoring system described by Zangheri [19], six patients had score 0 ($n = 3$) or 1A ($n = 3$) with no need for surgery in all five live born infants. All other patients with score 2 ($n = 7$) or 3 ($n = 3$) needed neonatal surgical intervention (Table 1).

Discussion

MP is a result of intestinal perforation and leakage of meconium into the abdominal cavity *in utero* [3, 9, 14]. On the basis of sonographic findings we could identify 14 fetuses with MP. Despite the sonographic findings, surgical interventions were only required in 60% of the 13 live born neonates. Our study demonstrates that prenatal ultrasound reveals variable findings but that the "typical" findings do not exist simultaneously [7, 10].

Table 2 Comparison of the prenatal ultrasound findings and postnatal need for neonatal surgery.

Prenatal ultrasound findings	Patients without neonatal surgery ($n = 5$)	Patients with neonatal surgery ($n = 8$)	Fisher's exact test P-value*
Intraabdominal calcification	5/5	8/8	1.00
Ascites	3/5	6/8	1.00
Meconium pseudocyst	0/5	5/8	0.07
Bowel dilatation	0/5	6/8	0.02
Hyperechogenic bowel	2/5	0/8	0.13
Hydrops fetalis	0/5	1/8	1.00
Polyhydramnion	1/5	1/8	1.00

*$P < 0.05$ was considered statistically significant.

The etiology of MP is variable [2, 8, 15]. The predominant findings in our study were anomalies of the gastrointestinal tract such as atresia [15]. Perforation mainly in the small intestine could be detected in all neonates who underwent surgery and three had an anatomic cause. The incidence of cystic fibrosis in infants with MP was previously reported to vary between 8% and 40% [6]. In our series, diagnosis of cystic fibrosis was confirmed in one case only. Studies also found a correlation of MP with viral infections such as cytomegalovirus or parvovirus B19 [17, 18], but none were found in the current population.

Other diseases associated with MP and are heterogeneous. Cholestasis due to biliary atresia or familial progressive intrahepatic cholestasis are rare [4, 11, 12]. However, we identified two additional cases with this association (Table 2). A new association was found in one of our patients who suffered from hemochromatosis.

Recently, Zangheri [19] described a scoring system and found the lowest risk (0%) for neonatal surgery in cases with isolated calcifications (score 0). In all other patients with score 1–3, the probability was increased to $>50\%$ [19]. We scored our patients according to the Zangheri and found no need for surgery in patients with score 0 as well but also in patients with score 1A (calcification associated with ascites). All our patients with scores >1 were associated with the need for surgical intervention, compared with 80–100% described by Zangheri [19]. For these infants (score >1), Zangheri's scoring system perfectly predicted the need for surgery. Our statistical analysis also revealed that only antenatal bowel dilatation was a statistically significant prognostic factor for the need for neonatal surgery, suggesting that prediction of patients who will require surgery should be made very carefully [19]. Other classification systems [6, 13] poorly correlated with the need for neonatal surgery and outcome.

We conclude that patients with a score >1 have a high probability for urgent neonatal surgery and therefore, should be transferred *in utero* to a tertiary center with available neonatal surgery.

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