Meconium Peritonitis

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Abstract

Meconium peritonitis is an unusual and often fatal form of neonatal peritonitis characterized by intraperitoneal calcification, numerous fibrosis with or without pseudocyst formation due to antenatal extravasation of meconium. This report is a retrospective study of 32 pediatric patients who were treated at the Surgical Department of the Children's Hospital from 1987 to 1996. The purposes of this study emphasize clinical manifestations, radiological findings, operative procedures and results of treatment. Twenty seven were neonates and five were older infants. The most common clinical presentation was abdominal distension at birth. The abdominal X-rays showed abnormal calcification and mass lesion in the peritoneal cavity in 71.9 and 46.9 per cent respectively. Only one patient was not treated surgically because he had no evidence of gut obstruction and inflammation. Thirty-one patients were operated on. At laparotomy, all of them had numerous inflammatory adhesion bands and matted bowel loops. Giant pseudocysts and intestinal perforations were noted in 64.5 and 54.8 per cent respectively. The obvious causes of meconium peritonitis were ileal atresia in 4, jejunal atresia in 3 and appendiceal perforation in 1. In the other 23 patients, no apparent cause of perforation was noted.

Only lysis of the adhesion with or without drainage was done in 9 patients and one of these died. Partial resection of pseudocysts and exterioization of the perforated bowel were done in 10 patients and 2 of these babies died. Primary anastomosis after resection of the perforated bowel was done in 12 patients and 5 of these cases died. The overall survival rate was 75 per cent.

Our data from this study suggested that partial resection of the pseudocyst and temporary enterostomy should be done in cases with bowel perforation and severe meconium contamination. Early diagnosis, proper operative procedure and meticulous postoperative care offer the best opportunity for survival of patients with meconium peritonitis.

Key word : Meconium Peritonitis

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Meconium peritonitis (MP) is defined as aseptic, chemical and foreign body peritonitis which is produced by extravasation of meconium from the fetal gastrointestinal tract into the peritoneal cavity during the intrauterine life[1]. In many of the cases, the intestinal perforation spontaneously closed with plastic exudate and fibrous tissue by the time of birth. In some cases, the perforation will occasionally remain open with massive pneumoperitoneum and ascites which may be superimposed by bacterial peritonitis. It is desirable to differentiate the MP from post-natal intestinal perforation by the evidence of extensive fibrous adhesion, calcium deposition with or without pseudocyst formation.

Morgagni was the first clinician to report the MP in 1761 and the first report of successful operation was in 1943 by Agerty[2]. Even though this disease was successfully treated by surgery long time ago, the mortality rate has remained high up to the present. Because of this, we decided to study our experience of MP at the Children's Hospital.

MATERIAL AND METHOD

Medical records of the patients who were treated in the Department of Surgery at the Children's Hospital for MP during the period of January 1987 to December 1996 were retrospectively reviewed. Thirty two patients were available for the study. Their records were collected and analysed regarding the clinical presentations, radiological findings, operative procedures and the results of treatment. The babies having postnatal intestinal perforations were excluded from this study. The clinical data were tested for the difference of statistical significance by paired *t*-test.

RESULTS

General incidence

The age of the patients at diagnosis ranged from one day to four months (average 16.8 days). Twenty seven neonates (84.5%) developed symptoms within the first month of life, 25 were present at birth and 2 after one week. Four infants had mild abdominal distension and clinical manifestations of intestinal obstruction became obvious after one month of life. One patient was incidentally found to have intraabdominal calcifications during the investigation of gastroesophageal reflux (GER).

Of the 32 patients, 20 were male and 12 were female. The male to female ratio was 1.7 : 1. Five neonates were born at Rajavithi Hospital, where 164,853 neonates were born during this period. The incidence of MP at Rajavithi Hospital was therefore about 1 in 33,000 live births.

The birth weight ranged from 1,630-4,600 g. Twenty two (68.75%) weighed more than 2,500 g, while 10 (31.25%) were premature babies.

Clinical presentation

Abdominal distension at birth was the most common presentation of this disease. Most of them failed to pass meconium and developed progressive abdominal distension (Fig. 1). Twenty four neonates (75%) had bilious vomiting within 24 hours after birth. Birth asphyxia was noted in 13 cases (40.6%) and 5 of them were resuscitated by endotracheal intubation and respiratory support. Eight patients had ascites. An ill-defined mass was palpable in 6 cases. Two boys had marked scrotal swelling and one girl had meconium from the umbilicus (Table 1).

Radiological findings

Abdominal X-rays demonstrated intra-abdominal calcification in about 72 per cent of all the cases (Fig. 2). Abdominal mass lesion, evidence of gut obstruction and ground glass appearance (Fig. 3) were noted in 46.9, 37.5 and 18.8 per

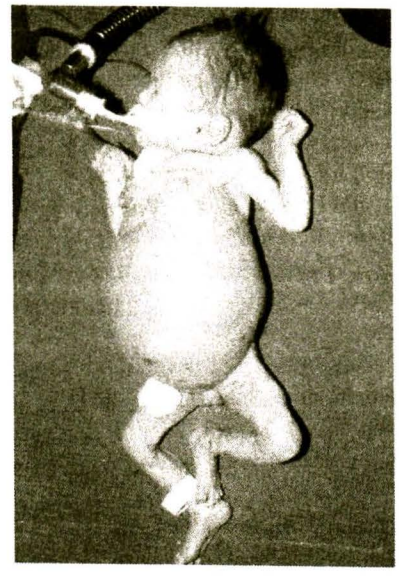

Fig. 1. A neonate with MP has marked abdominal distension at birth.

Table 1. Clinical presentation in 32 patients.

Clinical presentation	Number of patients	Per cent
Abdominal distension	28	87.5
Bilious vomiting	24	75
Birth asphyxia	13	40.6
Ascites	8	25
Jaundice	7	21.9
Abdominal mass	6	18.8
Scrotal swelling	2	6.3
Umbilical fecal fistula	1	3.1

Table 2. Radiological findings.

Radiological findings	Number of patients	Per cent
Intraperitoneal calcification	23	71.9
Abdominal mass	15	46.9
Intestinal obstruction	12	37.5
Ascites	8	25.0
Ground glass appearance	6	18.8
Pneumoperitoneum	5	15.6

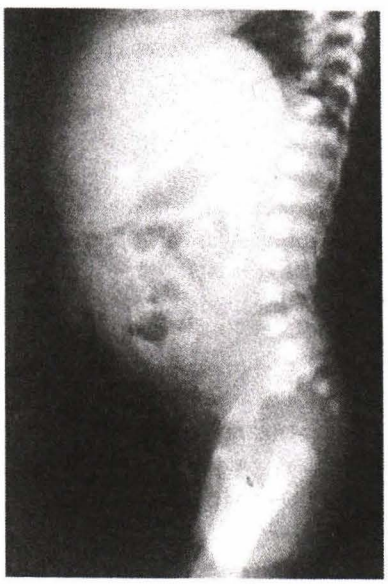

Fig. 2. Abdominal film shows intraperitoneal calcification.

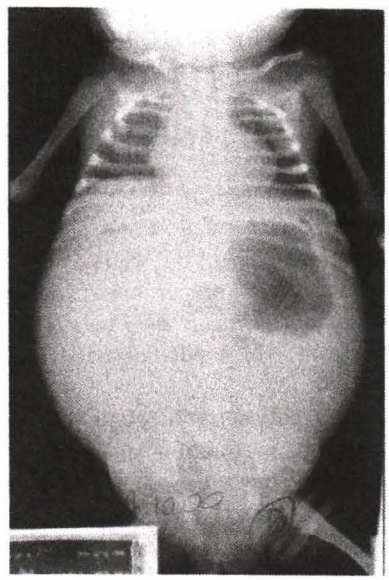

Fig. 3. Ground glass appearance.

cent respectively (Table 2). An intrathoracic calcification was surprisingly found in one neonate who had associated congenital diaphragmatic hernia (CDH) with meconium peritonitis (Fig. 4).

Associated anomalies

Seven patients had associated intestinal atresia, ileal in 4 and jejunal in 3. One baby was referred with the main problem of CDH and we diagnosed MP by chest X-rays and operative finding of calcifications at the surface of the jejunum. Meckel's diverticulum, ileal duplication, GER, hydrops fetalis and cryptorchidism were noted in one case each.

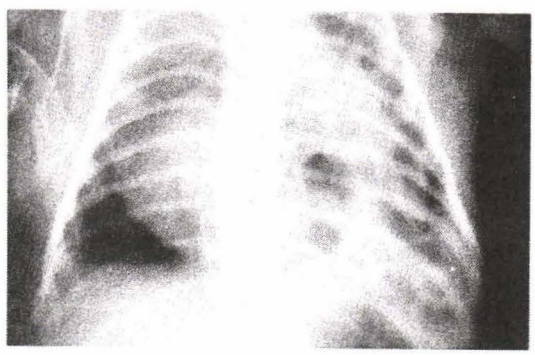

Fig. 4. A chest film of one baby with CDH reveals intrathoracic calcifcation.

Treatment

One boy who was incidentally found with GER was not treated surgically because he had no evidence of intraperitoneal inflammation or gut obstruction.

Thirty one patients underwent an exploratory laparotomy. Operative findings showed numerous inflammatory adhesion bands with calcifications and matted bowel loops in all of them. Giant pseudocysts were noted in 20 neonates (64.5%). Eighteen patients (54.8) had evidence of intestinal perforations. The points of perforation were noted in the terminal ileum, mid small bowel and jejunum in 12,5 and 1 case respectively. Only 8 cases had obvious causes of MP, ileal atresia in 4, jejunal atresia in 3 and appendiceal perforation in 1. In the other 23 cases, no apparent cause of bowel perforation was noted.

Nine patients had only numerous fibrosis without pseudocyst formation and no apparent intestinal perforation. Therefore, lysis of the adhesion was performed with intraperitoneal drainage in 5 cases and without drainage in 4 cases. One of the nine died on the third postoperative day due to septicemia.

In the patients with giant pseudocyst including intestinal perforation and bowel necrosis, partial resection of the pseudocyst, evacuation of meconium and exteriorization of the perforated bowel were done in 10 cases and 2 of these babies unfortunately died. One died in the immediate postoperative period because of necrotizing enterocolitis (NEC) and jejunal perforation . Nine who survived from the critical status had closure of the enterostomy within 3 months after the first operation and one infant died due to anastomotic leakage and septicemia.

Primary anastomosis after resection of the pseudocysts and the perforated bowel was performed in the remaining 12 patients and 5 of these cases died (Table 3). The causes of death in this group were the combination of anastomotic leakage, intraperitoneal abscess and septicemia.

Twenty four patients survived. The overall survival rate was 75 per cent. Table 3 summarizes the correlation of prognosis and other factors. The patients who were operated on during the infancy period without evidence of bowel perforation and had only lysis of adhesion or pseudocyst resection and enterostomy of the perforated bowel had a better chance of survival ($p<0.05$).

A follow-up of more than 6 months was available in all the survivors. The infant who was not surgically treated had normal growth and development. The vomiting of GER case spontaneously disappeared. Twenty three patients who were treated by operation recovered satisfactorily from laparotomy with excellent weight gain. None of them developed intestinal obstruction due to adhesions during the follow-up.

Table 3. Correlation of the factors that influence the prognosis.

Clinical data	No. of patients (cases)	Survival (per cent)	Survival rate
1. Age of onset of symtoms			
1.1 Neonate	27	19	70.4
1.2 Infant	5	5	100
2. Pathological findings			
2.1 Pseudocyst formation	20	15	75.0
Absence of pseudocyst	12	9	75.0
2.2 Bowel perforation	18	11	61.1
No evidence of perforation	14	13	92.9
3. Operative procedures			
3.1 Only lysis of adhesion	9	8	88.9
3.2 Resection of pseudocyst and perforated bowel + enterostomy	10	8	80.0
3.3 Resection of pseudocyst and perforated bowel + primary anastomosis	12	7	58.3

DISCUSSION

The incidence of MP is approximately 1 in 35,000 live births in the USA[2] and 1 in 20,000-40,000 live births in the Netherlands[3]. Our experience of 32 patients at the Children's Hospital of Thailand was about 1 in 33,000 live births and similar to that in Western countries.

In 1953, Bendel and Michel defined MP as a non-bacterial, chemical and foreign body peritonitis occurring during intrauterine life. It was believed to develop early in the fourth intrauterine month when the meconium had reached the ileocecal valve[1,2]. Since then, the etiology and pathophysiology of MP has been better understood. Lorimer and Ellis classified this entity into 3 pathological subtypes as fibroadhesive, cystic and generalized MP[4] but Martin classified it into 4 subtypes of adhesive, cystic, ascitic and infected MP[5]. There is frequently a significant overlap of these groups. The manifestations of MP depend on the timing of the antenatal perforation of the gastrointestinal tract and whether or not the perforation spontaneously seals[6].

The most common cause of MP that was reported from Western countries is meconium ileus [2,5,7-10]. In our study, in contrast, jejunoileal atresia is the most common cause and meconium ileus has not been found in Thailand. Intrauterine appendiceal perforation was surprisingly noted in one baby from this study. More than half of the cases often had no etiology[10]. Early prenatal perforation created an aseptic peritonitis with numerous fibrous adhesions and sometimes the perforation closed spontaneously[7]. Cystic MP is usually associated with a persistent postnatal communication between the intestine and the pseudocyst.

The sterile meconium creates marked chemical and foreign body reaction with rapid calcification within 12 hours[10,11]. Neuhauser considered the radiological finding of intraabdominal calcification in neonates to be pathognomonic of MP[12]. From other reports, intraabdominal calcification was found in approximately 60-75 per cent of all MP[9,13] while in our study this was present in about 72 per cent. However, Miller et al suggested that intraabdominal calcification was not always MP[14]. It may be intraluminal calcification which is found in patients with high typed imperforate anus and rectourinary fistula, total colonic aganglionosis, esophageal atresia and intestinal atresia[15-18]. They suggested differentiating MP from intraluminal calcification by serial supine and upright abdominal films for detection of calcification movement with position changes. Scrotal calcification was noted in some cases which was suggested to be pathognomonic of MP[19-22]. The apparent intrathoracic calcification in the case of CDH and MP, intrapelvic calcification and calcified buttock mass were present in many reports[23-26]. Some reported that leaking meconium drained as a persistent discharge from the vagina by passing through the fimbria, follopian tube and uterus[27]. This was not found in our patients. One of our cases had meconium discharge from the umbilicus. Hydrops fetalis was also found to be associated with MP[28].

MP most oftenly presents with the clinical manifestation of intestinal obstruction. Abdominal distension, bilious vomiting and birth asphyxia were the three common presentations in this study. More than 80 per cent of the cases developed the symptomatology shortly after birth. However, a late presentation of intestinal obstruction was found in 5 of 32 our patients and this was similar to the report of Dayalan[29].

The management of MP is either surgical or non surgical. The patients with asymptomatic MP do not require surgery. Almost all the cases of MP are operated on. The indications for laparotomy are usually intestinal obstruction, peritonitis and pseudocyst formation. Early diagnosis and prompt surgical intervention offer the best chance for survival. If surgery is delayed, intestinal flora which colonizes postnatally will convert an aseptic form to infected and septic peritonitis. The types of operative performance in each instance depend upon the pathological findings and the judgement of the surgeons. Lysis of adhesions only is suitable for patients with adhesive MP without atresia or perforation. Careskey[6] and Moore[30] suggested that the surgical procedure for giant cystic type was meconium evacuation, meticulous decortication of pseudocyst and temporary exteriorization of the perforated bowel. On the other hand, Tanaka[31] documented a minimal surgical procedure by tube drainage under ultrasonography in very ill neonates and surgical intervention was electively performed after the patients were improved by lysis of adhesion, resection of non-viable intestine and primary anastomosis. In our experience, the patients who underwent bowel resection with temporary enterostomy had a better survival rate than those who had

bowel resection and primary anastomasis ($p<0.05$). The patients with bowel resection and primary anastomosis had a higher risk of anastomotic leakage, progressive bowel necrosis and septicemia, especially the sick neonates. Peritoneal drainage is controversial. Whether to drain or not to drain does not have any significance on complication or survival(6). At the end of the operative procedure, the peritoneal cavity should be meticulously lavaged with warm saline and aspirated to remove particulate foreign debris. Postoperative ventilation support, parenteral hyperalimentation administration and high efficacy antibiotics are the best procedure for management of MP.

SUMMARY

A retrospective study of 32 pediatric patients who were treated for MP at the Children's Hospital from 1987 to 1996 indicated that the incidence of MP occurred in about 1 in 33,000 live births at Rajavithi Hospital. It is not so rare, therefore, it should be suspected in neonates with abdominal distension, respiratory distress and bilious vomiting. Almost all of the patients need surgical intervention for lysis of adhesion, pseudocyst resection, exteriorization of perforated bowels with or without peritoneal drainage.

Available information suggested that appropriate operative procedures should be done suitably for the patient's condition. Radical pseudocyst decortication, aggressive lysis of adhesion and primary bowel anastomosis are not recommended in neonates in poor condition because of the high risk of complications and high mortality rate. Four major factors that influence a good prognosis are late onset of clinical presentation, absence of intestinal perforation, proper operative procedure and well recognized post operative care.

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REFERENCE

1. Bendel WL Jr, Michel ML Jr. Meconium peritonitis. Surgery 1953; 34:321-33.
2. Cerise EJ, Whitehead W. Meconium peritonitis. Am Surg 1969; 35:389-92.
3. Tibboel D, Molenaar JC. Meconium peritonitis. A retrospective prognostic analysis. Z Kinderchir 1984; 39:25-8.
4. Lorimer WS Jr, Ellis DG. Meconium peritonitis. Surgery 1966; 60: 470-5.
5. Martin LW. Meconium peritonitis. In : Ravitch MM, Welch KJ, Benson CD, et al (eds). Pediatric Surgery, 3th ed. Chicago : Medical Publishers, 1979 : 952-5.
6. Careskey JM, Grosfeld JL, Weber TR, et al. Giant cystic meconium peritonitis (GCMP): improved management based on clinical and laboratory observations. J Pediatr Surg 1982; 17:482-9.
7. Ein SH. Meconium ileus. In : Freemann NV, Burge DM, Griffiths M, et al (eds). Surgery of the Newborn. Edinburgh, London : Chirchill Livingstone, 1994:139-57.
8. Fox PF, Potts WJ. Meconium ileus and meconium peritonitis. Arch Surg 1957; 74:733-5.
9. Smith B, Clatworthy HW. Meconium peritonitis : prognostic significance. Pediatrics 1961; 27:967-70.
10. Santulli TV. Meconium ileus. In : Holder TM, Ashcraft KW (eds). Pediatric Surgery,1 st ed. Philadelphia : Saunders, 1980: 356-73.
11. Heetderks DR Jr, Verbrugge GP. Healed meconium peritonitis presenting as a scrotal mass in an infant. J Pediatr Surg 1969; 4: 363-5.
12. Neuhauser EBD. The roentgen diagnosis of fetal meconium peritonitis. AJR 1944; 51: 421-4.
13. White RB. Meconium peritonitis, a surgical emergency. J Pediatr 1956; 48: 793-8.
14. Miller JP, Smith SD, Newman B, et al. Neonatal abdominal calcification : is it always meconium peritonitis ? J Pediatr Surg 1988; 23 : 555-6.
15. Berdon WE, Baker DH, Wigger HJ, et al. Calcified intraluminal meconium in newborn males with imperforate anus. AJR 1975; 125: 449-55.
16. Bager J, Bar-Marr JA. Intraluminal intestinal calcifications in a newborn with atresia of the esophagus and imperforate anus. Clin Pediatr 1980; 19: 770-2.
17. Fletcher BD, Yulish BS. Intraluminal calcifications in the small bowel of newborn infants with total colonic aganglionis. Radiology 1978; 126: 451-5.
18. Martin CE, Leonidas JL, Armoury RA. Multiple gastrointestinal atresias with intraluminal calci-

fications and cystic dilatation of bile ducts. A newly recognized entity resembling a "string of pearls". Pediatrics 1976; 57 : 268-71.
19. Olnick HM, Hatcher MB. Meconium peritonitis. JAMA 1953; 152: 582-4.
20. Boix-Ochoa J. Meconium peritonitis. J Pediatr Surg 1968; 3: 715-22.
21. Gunn LC, Ghionzoli OG, Gasdner HG. Healed meconium peritonitis presenting as a reducible scrotal mass. J Pediatr 1978; 92: 847.
22. Dehmer LP, Scott D, Stocker JT. Meconium peritonitis. A clinicopathologic study of four cases with a review of the literature. Hum Pathol 1986; 17: 807-12.
23. Kimball D, Smith W. Meconium peritonitis with thoracic extension. Am J Radiol 1985; 144: 113-4.
24. Christopher TD, Effmann EL, Filston H. Meconium peritonitis and pleuritis : a clue to perforation of intestine and incarcerated Bochdalek Hermia in a neonate.J Pediatr Surg 1990; 25: 558-9.
25. Mitsudo SM, Boley SJ, Rosenzweig MJ, et al. Extraperitoneal pelvic meconium extravasation in a newborn infant. J Pediatr 1983; 103: 598-600.
26. West DK, Touloukian RJ. Meconium pseudocyst presenting as a buttock mass. J Pediatr Surg 1984; 23: 864-5.
27. Gauderer WL, Darrell LC. Meconium per vagina secondary to ileal atresia and meconium peritonitis. Pediatr Surg Int 1992; 7: 64-6.
28. Hasegawa T, Imura K, Kobayashi T, et al. Giant cystic meconium peritonitis with hydrops fetalis. Pediatr Surg Int 1993; 8: 433-5.
29. Dayalan N, Ramakrishnan MS. Meconium peritonitis. J Pediatr Surg 1974; 9: 243-4.
30. Moore TC. Giant cystic meconium peritonitis. Ann Surg 1963; 157: 566-72.
31. Tanaka K, Hashizume K, Kawarasaki H, et al. Elective surgery for cystic meconium peritonitis : report of two cases. J Pediatr Surg 1993; 28: 960-1.

เยื่อบุช่องท้องอักเสบจากขี้เทา

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Meconium Peritonitis เป็นความผิดปกติที่พบได้ไม่บ่อยนักและเป็นสาเหตุที่ทำให้ทารกเสียชีวิตได้มาก เป็นภาวะที่มีการทะลุของระบบทางเดินอาหารทำให้มีขี้เทาไหลออกไปอยู่ในช่องท้องตั้งแต่ทารกอยู่ในครรภ์มารดา เกิดการอักเสบชนิดที่ไม่มีการติดเชื้อ ทำให้มีเยื่อพังผืดเกิดขึ้นเป็นจำนวนมาก ร่วมกับมี calcification และอาจจะมี pseudocyst หรือไม่มีก็ได้ รายงานนี้เป็นการศึกษาย้อนหลังผู้ป่วยเด็กทารก 32 รายที่ได้รับการรักษาในกลุ่มงานศัลยกรรม โรงพยาบาลเด็ก ตั้งแต่ปี พ.ศ. 2530-2539 มีวัตถุประสงค์เพื่อศึกษาถึงลักษณะทางคลินิก ภาพรังสี วิธีการผ่าตัดและผลของการรักษา ผู้ป่วย 27 ราย เป็นเด็กอายุต่ำกว่า 1 เดือนและ 5 รายมีอายุมากกว่า 1 เดือน ลักษณะทางคลินิกที่พบบ่อยที่สุดคืออาการท้องอืดตั้งแต่แรกเกิด ภาพรังสีของช่องท้องพบ calcificaiton และก้อนในท้องประมาณร้อยละ 72 และร้อยละ 47 ตามลำดับ มีผู้ป่วยเพียง 1 รายที่รักษาโดยการไม่ผ่าตัดเพราะไม่มีอาการทั้งลำไส้อุดตันและการอักเสบภายในช่องท้อง

ผู้ป่วย 31 รายรักษาโดยการผ่าตัด พบว่าทุกรายมีเยื่อพังผืดเป็นจำนวนมากเกิดขึ้นภายในช่องท้องพร้อมกับมีลำไส้ติดกันคล้ายเป็นก้อน พบว่ามี pseudocyst และการทะลุของลำไส้ยังปรากฏอยู่ประมาณร้อยละ 65 และร้อยละ 55 ตามลำดับ สาเหตุของการทะลุที่พบเด่นชัดมีเพียง 8 รายคือจากไอเลียมตัน 4 ราย เจจูนัมตัน 3 ราย และไส้ติ่งแตกตั้งแต่อยู่ในครรภ์มารดา 1 ราย ส่วนอีก 23 รายไม่ทราบสาเหตุที่แน่ชัด

ผู้ป่วย 9 รายรักษาโดยการตัดและเลาะแยกเยื่อพังผืดออกจากลำไส้เพียงอย่างเดียว และมีทารกที่เสียชีวิตจากการรักษาโดยวิธีนี้เพียง 1 ราย รักษาโดยการตัด pseudocyst บางส่วนออก ร่วมกับนำลำไส้ส่วนที่ทะลุเปิดออกชั่วคราวที่ผนังหน้าท้อง 10 รายและมีเด็กเสียชีวิต 2 ราย ผู้ป่วยที่เหลืออีก 12 รายรักษาโดยการตัด pseudocyst ออกบางส่วนและนำลำไส้ที่ทะลุหรือลำไส้ที่ตัดทิ้งมาเย็บต่อเข้าหากันในครั้งเดียวกันและมีผู้ป่วยเสียชีวิต 5 ราย ผู้ป่วยทั้ง 32 รายรักษาหายเป็นปกติ 24 ราย คิดเป็นอัตราผู้รอดชีวิตร้อยละ 75

ข้อมูลนี้ได้รับจากการศึกษาครั้งนี้พบว่าทารกที่มีการทะลุของลำไส้ตั้งแต่อยู่ในครรภ์มารดาและมีอาการหนักควรได้รับการรักษาโดยการตัด pseudocyst ออกเพียงบางส่วนร่วมกับนำลำไส้ที่ยังมีการทะลุมาเปิดออกชั่วคราวที่ผนังหน้าท้องน่าจะให้ผลดีกว่าการเย็บต่อลำไส้ทันที การวินิจฉัยผู้ป่วยให้ได้โดยเร็วที่สุดร่วมกับการตัดด้วยวิธีที่เหมาะสมและการดูแลเอาใจใส่อย่างดีภายหลังผ่าตัดจะทำให้ผู้ป่วยมีโอกาสรอดมากขึ้น

คำสำคัญ : เยื่อบุช่องท้องอักเสบจากขี้เทา

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