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SM Journal of Pediatrics

Case Report

Intraperitoneal Abscess in a Preterm Infant: a Case Report

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Article Information

Received date: Dec 06, 2016
Accepted date: Jan 14, 2017
Published date: Jan 19, 2017

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Keywords Abscess; Neonate; Peritonitis

Abstract

Developments in empiric antibiotic therapy in recent decade shave led to a decrease in idiopathic primary peritonitis in neonates. Furthermore, intraperitoneal abscesses are extremely rare in neonates. We report a case of an intraperitoneal abscess without an apparent cause, such as necrotizing enterocolitis or surgery, in a preterm infant. The main contribution of our report is a reminder that intra-abdominal abscesses, although rare, can and do occur in neonates, and that diagnosing them can be difficult.

Introduction

Intraperitoneal abscesses in neonates are extremely rare, with a few published reports in term neonates [1]. But none in preterm neonates. Intra-abdominal abscesses (including sub-phrenic, hepatic, intraperitoneal, and retroperitoneal) can occur as complications of peritonitis, or secondary to necrotizing enterocolitis, surgery, or trauma [2]. Until the 1980s, reports of neonatal idiopathic primary peritonitis were published [3-7]. However, with the early use of empiric antibiotics in recent decades, neonatal peritonitis has become rare; antibiotics often cure peritonitis arising from hematogenous bacterial dissemination.

We report a case of an intraperitoneal abscess in a preterm female neonate, with unusual Computed Tomography (CT) findings. We present the case as a reminder that intraperitoneal abscesses in neonates, although rare, can and do occur.

Case Report

A preterm female was born via emergency cesarean section at 31 (+0) weeks' gestational age (birth weight, 1,172 g). Her 41-year-old mother was diagnosed with severe pregnancy-induced hypertension at 20 weeks' gestation; this was the indication for the cesarean section. The child's Apgar scores were 9/10 and 9/10 at 1 and 5 minutes of life, respectively. Maternal vaginal smear was negative for Streptococcus group B at 31 weeks' gestation.

Apart from mild chest wall retraction with breathing, the neonate's physical examination on admission revealed no abnormalities. Initial laboratory results were unremarkable: complete blood cell count was normal, C-Reactive Protein (CRP) was normal (<0.3mg/dL), and serum immunoglobulin levels were normal (immunoglobulin G, 742 mg/dL; immunoglobulin A, 11 mg/dL; and immunoglobulin M, 8 mg/dL).

A Peripherally Inserted Central Catheter (PICC) was used for intravenous nutrition; nasal Continuous Positive Airway Pressure (CPAP) was initiated soon after neonatal intensive care unit admission. The patient's general condition remained good. On day eight, nasal CPAP was discontinued and PICC was removed.

At 35 days of age, despite a normal physical examination, laboratory investigations revealed an elevated White Blood Cell (WBC) count (30,800/mm^3; 28% lymphocytes; 78% neutrophils) but no left shift, and CRP level of 3.1 mg/dL. A presumptive diagnosis of bacterial infection was made; empiric intravenous antibiotic therapy was initiated with ampicillin (50 mg/kg/dose every 8 hours) and amikacin (5 mg/kg/dose every 12 hours). By 39 days of age, WBC count had increased to 39,400/mm^3 and CRP was elevated (5.6 mg/dL). However, blood culture did not reveal any pathogenic bacteria; her general condition remained good. We thus discontinued antibiotic therapy at 40 days of age. The CRP level at 43 days of age had spontaneously decreased, but was still slightly higher than normal (<0.3 mg/dL) at 0.4 mg/dL. We wondered, therefore, whether the recent increase in inflammatory markers had actually been due to an infection. The patient remained afebrile and displayed no signs of intestinal obstruction, but, at 51 days of age, she developed increased work of breathing. Repeat laboratory investigation revealed an elevated WBC count (40,700 mm^3) and CRP

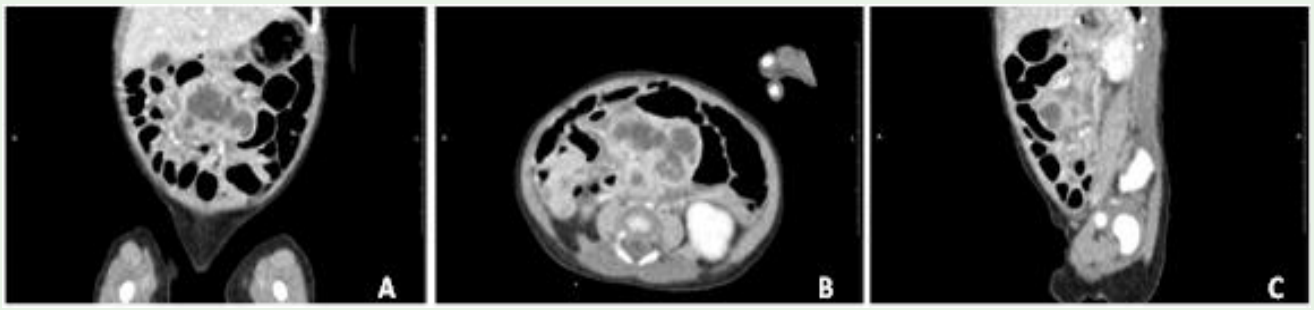
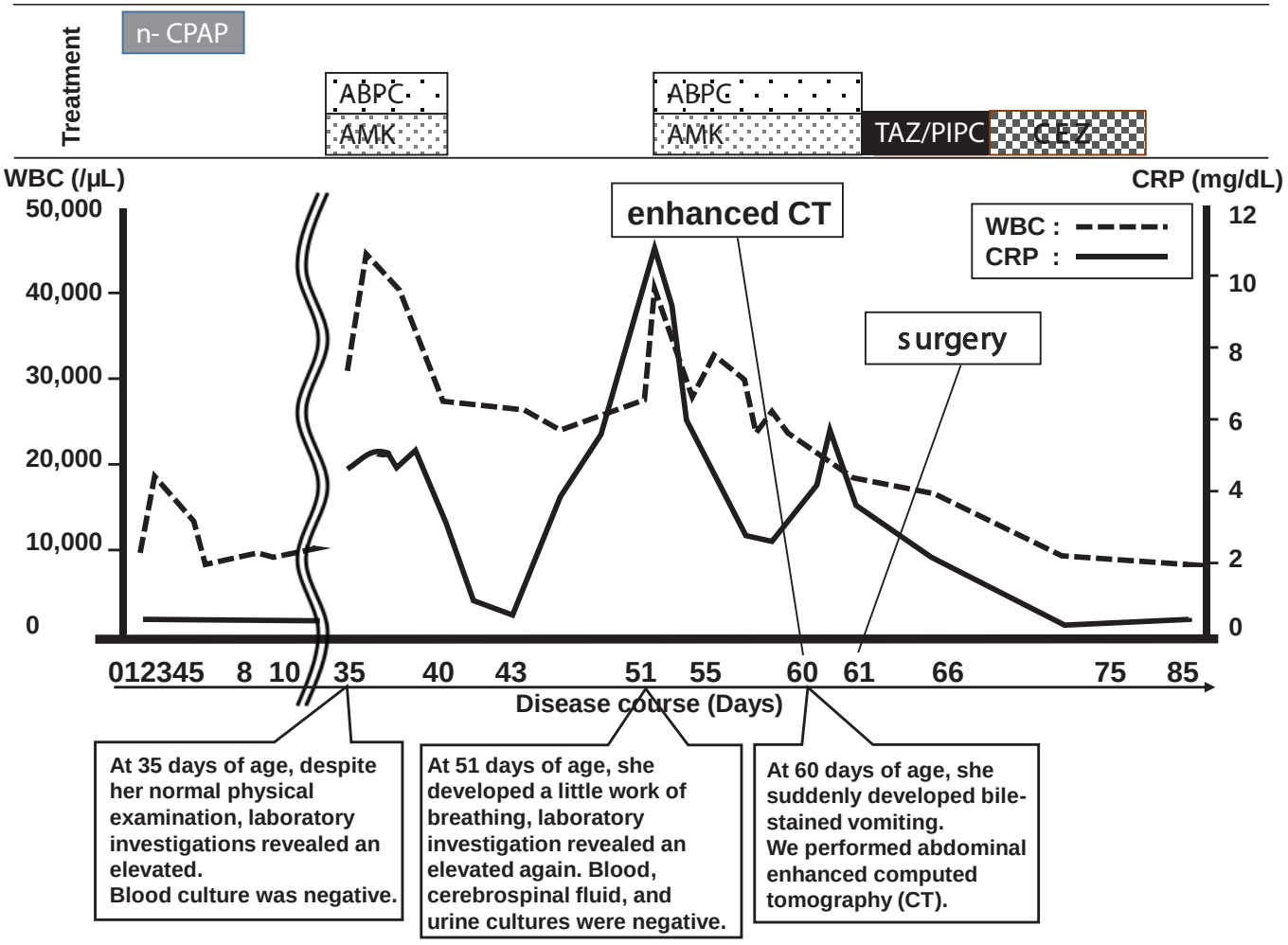


Figure1: A) Coronal view abdominal enhanced computed tomography image demonstrating a 3.5×3.0×2.8 cm septated cystic mass with an irregularly enhancing thickened wall, in the central abdomen. B) Axial view. C) Sagittal view.

level (10.5 mg/dL). A second empiric course of intravenous ampicillin (50 mg/kg/dose every 8 hours) and amikacin (5 mg/kg/dose every 12 hours) was initiated. Blood, cerebrospinal fluid, and urine cultures were negative. The CRP level decreased to 3.5 mg/dL. However, at 60 days of age, she suddenly developed bile-stained vomiting. We performed abdominal enhanced Computed Tomography (CT), which demonstrated a 3.5×3.0×2.8 cm septated cystic mass with an irregularly enhancing thickened wall in the central abdomen, suggestive of an intraperitoneal abscess (Figure 1). At 61 days of age, she underwent surgery for bowel obstruction due to the abscess. At surgery, a well-defined, round, capsular abscess was removed from the jejunal peritoneum; a5-cm segment of associated jejunum was resected; and an end-to-end anastomosis was performed. There was minimal localized peritonitis (with resultant ileus), but no generalized

Table 1: Timelinen-CPAP: nasal continuous positive airway pressure, ABPC: ampicillin, AMK: amikacin, CEZ: cefazoline, CT: computer tomography, TAZ/PIPC: tazobactam/piperacillin.



peritonitis. Histology demonstrated inflammatory cell infiltration from the serosal to the mucosal layer of the jejunum. These surgical and histological findings confirmed that there were no underlying disorders, such as localized intestinal perforation, necrotizing enterocolitis, or intestinal duplication.

Tazobactam/piperacillin (100 mg/kg/dose intravenously every 8 hours) was administered for five days post-surgery. The abscess fluid subsequently cultured Methicillin-Sensitive Staphylococcus Aureus (MSSA); hence, antibiotic therapy was de-escalated to cefazolin (50 mg/kg/dose intravenously every 8 hours) for ten days, (Table 1).

Post operative follow-up, by means of clinical review and imaging studies, revealed no abnormality at 3 months of age. At her outpatient follow-up visit at 6 months of age, the patient was well, and her physical examination was normal.

Discussion

Abscesses can occur in any solid tissue. The Gastrointestinal (GI) tract consists of four layers of specialized tissue: the mucosa, submucosa, muscular layer, and serosa, from the lumen outwards. The serosa-outermost layer of the GI tract-consists of several connective tissue layers. In this neonate, the intraperitoneal abscess had developed within this narrow connective tissue region.

Delayed diagnosis and treatment of an intraperitoneal abscess can result in increased mortality [2]. In this case, the diagnosis was difficult to establish in the early phase, for the following reasons: First, the patient had no underlying cause, such as peritonitis, necrotizing enterocolitis, surgery, or trauma [2]. Idiopathic primary peritonitis in neonates is caused mostly by sepsis, but can be associated with omphalitis [1, 3-7]. However, the patient's blood culture was negative and she did not have omphalitis. Furthermore, she did not have necrotizing enterocolitis or intestinal duplication.

Second, she displayed no symptoms until bile-stained vomiting started at 60 days of age. The increased work of breathing on day 51 may have been caused by the abscess. However, the overlap between signs of neonatal peritonitis and sepsis made it difficult to differentiate between the two based on the clinical findings alone.

Third, we did not suspect an intraperitoneal abscess. Although Ultra Sonography (US) of solid organs (e.g., brain, liver, kidney) was performed to screen for a suspected abscess because of laboratory data suggesting chronic inflammation, solid tissue of the small intestine was not considered. Furthermore, intestinal gas may have masked intra-abdominal pathology on ultrasound. Other intra-abdominal abscesses in neonates have been reported [2], but an intraperitoneal abscess as in this case is considered extremely rare.

In terms of microbiology, Brook reported that the Bacteroides fragilis group, Peptostreptococcus, and Escherichia coli were the predominant pathogens in a case series of 36 children with intra-abdominal abscesses [8]. Conversely, gram-positive organisms are considered to be predominant among neonates with idiopathic primary peritonitis [9]. S.aureus has been reported as the most common etiology of abscesses seeding from a distant site, while abscesses originating in the GI tract are usually due to bowel commensals [10]. Thus, MSSA cultured from the abscess in this patient suggested that the abscess seeded from a distant site and did not originate in the GI tract. This result is consistent with the histologic findings.

Although the blood culture was negative in our patient, blood cultures have been reported to be positive in only around 50% of neonates with liver abscesses [2]. The diagnosis of an intraperitoneal abscess can be missed because the clinical, routine laboratory and blood culture findings are nonspecific. An enhanced abdominal CT should be performed if an abscess is suspected.

A limitation of this report is that the original focus of infection could not be identified. We assumed that this was an idiopathic intraperitoneal abscess. However, we speculated that the PICC or an injury from a heel prick blood test might have been the route of entry of the original infection, and that the preterm neonate's immature immune system allowed hematogenous spread, leading to abscess formation.

Conclusion

In conclusion, to our knowledge, this is the first report of an intraperitoneal abscess in a preterm infant. We should recognize that abscesses can occur in any solid tissue, including that of the small intestine, and may require CT for detection.

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