Pneumococcal Peritonitis in Previously Healthy Adults: Case Report and Review

Carolyn Hemsley and Susannah J. Eykyn

From the Division of Infection, St. Thomas’ Hospital, London, United Kingdom

We report a case of primary pneumococcal peritonitis in a young woman with no predisposing features, and we review 26 other cases of pneumococcal peritonitis in previously healthy adults. This disease is very rare in adults without cirrhosis, ascites, nephrotic syndrome, autoimmune disease, or immunocompromise. It occurs almost exclusively in women, and in most of these patients, there is a probable genital tract source of the organism. A few cases are associated with acute appendicitis. Although pneumococcal peritonitis was usually fatal in the preantibiotic era, with antibiotic therapy and surgical intervention the outcome for patients with this infection is excellent.

Pneumococcal peritonitis is a well-recognized infection in children [1–5] but is rare in adults, in whom it is usually associated with cirrhosis [6–8] or nephrotic syndrome [9]. There are scattered reports of pneumococcal peritonitis in patients undergoing continuous ambulatory peritoneal dialysis [10, 11], those who have undergone bone marrow transplantation [12], and those with rheumatoid arthritis [13] or systemic lupus erythematosus [14]. In addition, pneumococcal peritonitis is sometimes associated with genital tract infections in women [15]. We describe a young woman with primary pneumococcal peritonitis who had no underlying disease, and we review the literature on this infection.

Case Report

A 36-year-old woman was admitted to the hospital for evaluation of upper abdominal pain of 3 days’ duration and diarrhea and vomiting of 2 days’ duration. She had been previously healthy and denied any preceding respiratory or gynecologic symptoms. She was mid–menstrual cycle and did not have a tampon or an intrauterine contraceptive device (IUCD) in situ. She was married, denied having any new sexual partners, and had no risk factors for HIV infection. On physical examination she was obviously unwell, with a temperature of 39°C, pulse rate of 120, and a normal blood pressure. There was generalized abdominal tenderness. Findings on a pelvic examination were unremarkable. Her peripheral WBC count was 7.6 × 10^9/L with a normal differential, and the C-reactive protein level was 273. Blood for cultures was drawn, and she was treated with intravenous cefuroxime and metronidazole. Despite antibiotic therapy and supportive measures, she remained toxic and febrile. An ultrasonogram showed free fluid in the abdomen and, in view of suspected peritonitis, a laparotomy was performed and revealed diffuse peritonitis but no visceral perforation. Her appendix, uterus, ovaries, and fallopian tubes were macroscopically normal. Loops of small bowel were covered with a thick creamy exudate that on gram-stained smear contained gram-positive diplococci; the next day, culture of this exudate yielded a heavy pure growth of a mucoid Streptococcus pneumoniae (serotype 1). Pneumococci were also isolated from both bottles of the single blood culture. She was transferred to the intensive care unit for ventilatory and inotropic support. Her antibiotic therapy was changed to intravenous amoxycillin, 500 mg t.i.d. for 10 days. No high vaginal swab was obtained before treatment was begun.

She initially responded well to the postoperative treatment but was difficult to wean from the ventilator because of poor respiratory muscle function and inability to clear secretions. An attempt at extubation failed, and a tracheostomy was formed. She developed bulbar dysfunction with recurrent aspiration of solids and fluids. Investigations showed no evidence of myasthenia gravis, multiple sclerosis, or any apparent cause for her myopathy. She had no clinical evidence of a connective tissue disorder, but this was not further investigated. Her condition improved over a period of several weeks with supportive treatment, physiotherapy, and speech therapy, and the tracheostomy was closed. She was discharged from the hospital after 7 weeks and has remained well.

Literature Review

Spontaneous primary pneumococcal peritonitis in children has been recognized for almost 100 years [1, 5]. Its peak incidence is between the ages of 5 and 7 years, and it is more common in girls than boys (ratio, 4:1) [4]. Patients present with a short history of abdominal pain, high fever, diarrhea, and vomiting [2–5, 16]. At operation, there is a classically thick purulent exudate covering the bowel, from which S. pneumoniae is isolated. Although this infection is rarely seen today, at the beginning of the century it was reported to account for 8%–10% of abdominal emergencies in children [4, 5]. The association between pneumococcal peritonitis and nephrotic syndrome in children was first reported in 1940 [9].

Received 24 November 1997; revised 9 March 1998.
Reprints or correspondence: Professor Susannah J. Eykyn, Division of Infection, St. Thomas’ Hospital, Lambeth Palace Road, London, SE1 7EH, United Kingdom.

Clinical Infectious Diseases 1998;27:376–9
© 1998 by the Infectious Diseases Society of America. All rights reserved.
1058–4838/98/2702–0020$03.00
Table 1. Clinical findings and diagnostic criteria for patients with pneumococcal peritonitis.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (y)/sex</th>
<th>Blood culture (serotype)</th>
<th>Peritoneal fluid culture (serotype)</th>
<th>Other sites of infection</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 [18]</td>
<td>76/F</td>
<td>+</td>
<td>+</td>
<td>...</td>
<td>Ruptured ovarian cyst</td>
</tr>
<tr>
<td>2 [19]</td>
<td>35/F</td>
<td>-</td>
<td>-</td>
<td>IUCD</td>
<td>Pelvic inflammatory disease</td>
</tr>
<tr>
<td>3 [19]</td>
<td>29/F</td>
<td>-</td>
<td>+</td>
<td>Cervix</td>
<td>Postpartum (12 d)</td>
</tr>
<tr>
<td>4 [20]</td>
<td>33/F</td>
<td>-</td>
<td>+</td>
<td>...</td>
<td>Pelvic inflammatory disease, IUCD</td>
</tr>
<tr>
<td>5 [21]</td>
<td>28/F</td>
<td>ND</td>
<td>+</td>
<td>...</td>
<td>Pelvic inflammatory disease</td>
</tr>
<tr>
<td>6 [22]</td>
<td>21/F</td>
<td>ND</td>
<td>+</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>7 [23]</td>
<td>30/M</td>
<td>ND</td>
<td>ND</td>
<td>Appendix (19)</td>
<td>Appendicitis</td>
</tr>
<tr>
<td>8 [23]</td>
<td>31/F</td>
<td>ND</td>
<td>ND</td>
<td>Appendix (23)</td>
<td>Appendicitis</td>
</tr>
<tr>
<td>9 [24]</td>
<td>20/F</td>
<td>+</td>
<td>+</td>
<td>...</td>
<td>Pelvic inflammatory disease</td>
</tr>
<tr>
<td>10 [25]</td>
<td>38/F</td>
<td>+</td>
<td>+</td>
<td>...</td>
<td>Pelvic inflammatory disease, IUCD</td>
</tr>
<tr>
<td>11 [26]</td>
<td>31/F</td>
<td>+ (1)</td>
<td>+</td>
<td>Cervix, endometrium</td>
<td>Pelvic inflammatory disease</td>
</tr>
<tr>
<td>12 [27]</td>
<td>32/F</td>
<td>+</td>
<td>ND</td>
<td>...</td>
<td>IUCD</td>
</tr>
<tr>
<td>13 [27]</td>
<td>46/F</td>
<td>+</td>
<td>ND</td>
<td>...</td>
<td>IUCD</td>
</tr>
<tr>
<td>14 [27]</td>
<td>46/F</td>
<td>+</td>
<td>+</td>
<td>...</td>
<td>IUCD</td>
</tr>
<tr>
<td>15 [28]</td>
<td>23/F</td>
<td>ND</td>
<td>+ (3)</td>
<td>...</td>
<td>Appendicitis</td>
</tr>
<tr>
<td>16 [28]</td>
<td>78/M</td>
<td>ND</td>
<td>+ (6)</td>
<td>...</td>
<td>Appendicitis</td>
</tr>
<tr>
<td>17 [28]</td>
<td>80/F</td>
<td>ND</td>
<td>+</td>
<td>...</td>
<td>Appendicitis</td>
</tr>
<tr>
<td>18 [29]</td>
<td>32/F</td>
<td>+</td>
<td>+ (3)</td>
<td>Vagina</td>
<td>IUCD</td>
</tr>
<tr>
<td>19 [30]</td>
<td>26/F</td>
<td>ND</td>
<td>+</td>
<td>...</td>
<td>Pelvic inflammatory disease, postpartum (3 mo)</td>
</tr>
<tr>
<td>20 [31]</td>
<td>36/F</td>
<td>ND</td>
<td>+</td>
<td>...</td>
<td>Retained placenta 5 mo previously</td>
</tr>
<tr>
<td>21 [31]</td>
<td>38/F</td>
<td>-</td>
<td>+</td>
<td>...</td>
<td>IUCD</td>
</tr>
<tr>
<td>22 [32]</td>
<td>27/F</td>
<td>+</td>
<td>+</td>
<td>...</td>
<td>IUCD</td>
</tr>
<tr>
<td>23 [33]</td>
<td>16/F</td>
<td>ND</td>
<td>+ (1)</td>
<td>...</td>
<td>Appendicitis</td>
</tr>
<tr>
<td>24 [34]</td>
<td>25/F</td>
<td>+</td>
<td>+</td>
<td>...</td>
<td>IUCD</td>
</tr>
<tr>
<td>25 [35]</td>
<td>42/F</td>
<td>+</td>
<td>-</td>
<td>Vagina, throat</td>
<td>Uterine abscess</td>
</tr>
<tr>
<td>26 [36]</td>
<td>27/F</td>
<td>+</td>
<td>-</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>27 [PR]</td>
<td>36/F</td>
<td>+ (1)</td>
<td>+ (1)</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

NOTE. IUCD = intrauterine contraceptive device (in situ); ND = not done; PR = present report; + = positive; – = negative.

Pneumococcal peritonitis in adults was first described in cirrhotic patients [6–8, 17], and it is uncommon in adults without underlying cirrhosis, nephrotic syndrome, or immunocompromise. A MEDLINE search of the literature from 1966–1997 was undertaken to review such cases. For the purpose of the review, pneumococcal peritonitis was defined as peritonitis in which pneumococci were isolated from the blood or cultures of intraabdominal fluid, and peritonitis was defined as the presence of clinical signs and evidence of intraabdominal fluid or pus. Children <16 years of age and patients with known cirrhosis, ascites, nephrotic syndrome, autoimmune disease, or immunocompromise were excluded.

Table 1 gives the details of 26 previously reported cases of pneumococcal peritonitis in previously healthy adults [18–36] and of our own case. These cases confirm that the infection occurs predominantly in women (93% [25 of 27 patients]), and in most of these cases (68% [17 of 25]), the genital tract was the primary source of the infection. Genital tract involvement included pelvic inflammatory disease, vaginal discharge, an IUCD in situ, or recent childbirth. S. pneumoniae was isolated from genital tract specimens in only 20% (5 of 25) of women. In 16% of cases (4 of 25), vaginal discharge was a presenting feature. Infection occurred predominantly in women of menstrual age (median age, 32 years; range, 16–80 years).

S. pneumoniae was isolated from the blood in all but four cases in which blood cultures were performed (13 [76%] of 17). There was no reference to blood cultures in the remaining cases (10 of 27) and, therefore, this figure may be an underestimate of the frequency of bacteremia.

In six cases (22%), histologically proven appendicitis was present and S. pneumoniae was cultured from the appendix or appendiceal mass [23, 28, 33]. Only three patients (11%) had a history of respiratory symptoms or radiological evidence of pneumonia, either on admission or within 48 hours of admission [18, 31, 36].

The most common presenting features were abdominal pain (89% [24 of 27 patients]), fever (70% [19 of 27]), and diarrhea and vomiting (52% [14 of 27]), suggesting visceral perforation or appendicitis. In two-thirds of the patients (63% [17 of 27]), an obvious primary source was found at operation: an inflamed appendix or edematous fallopian tubes associated with widespread peritonitis. In the other one-third (37% [10 of 27]), there was no obvious intraabdominal source. In almost all patients, loops of bowel were covered in thick, creamy pus (as much
as 2 L in some cases). *S. pneumoniae* was cultured in pure growth from the peritoneal exudate in 25 cases; however, for two of the patients with appendicitis, the organism was isolated in mixed culture with anaerobes and other gut flora.

All patients received antibiotics on admission, and all but three had undergone an operative procedure (22, laparotomy; 2, laparoscopy). Of the three patients who did not undergo surgery, one responded to treatment with intravenous chloramphenicol alone [27]; one responded to treatment with penicillin, metronidazole, and gentamicin, with removal of an IUCD [27]; and the other had pus removed at culdocentesis and then responded to an antibiotic regimen of penicillin, gentamicin, and metronidazole, then changed to cefotaxime, and finally to chloramphenicol and erythromycin [33]. Of the patients who were treated surgically, 15 underwent laparoscopy or laparotomy in the first 24 hours after admission, and the remaining nine underwent surgery ≤1 week later because of failure to respond to conservative management alone. The initial choice of antibiotics varied, but on isolation of the organism most patients were treated with intravenous penicillin. The mean duration of antibiotic treatment was 11 days (range, 7–18 days). Two patients died: an elderly woman with a ruptured ovarian cyst, and a woman who presented with septic shock and acute renal failure [18, 27].

**Discussion**

The pathogenesis of pneumococcal peritonitis remains controversial. Pneumococci may gain entry to the peritoneal cavity and cause disease via the genital tract, the gastrointestinal tract, or by hematogenous spread from the respiratory tract. Pathogenesis may differ in adults and children. Our review suggests that in adults, the disease affects females predominantly and that the genital tract is the most common source of *S. pneumoniae*. This parallels the early findings for children [1–5, 16]. McCartney and Fraser [5] reported that only 12 cases among 56 children had occurred in boys. Similarly, data on children (1956–1970) show a ratio of females to males of 4:1 [3]. *S. pneumoniae* is occasionally found as a vaginal commensal and can presumably then cause ascending infection [15], with factors such as an the presence of an IUCD or recent delivery predisposing to this [19, 20, 25, 27, 29, 31, 32, 34].

The gastrointestinal tract has also been thought to be a source of pneumococci. It is postulated that infection is secondary to transmigration of the organism through the intestinal wall into the peritoneal cavity. There is also clearly an association between acute appendicitis and pneumococcal peritonitis [23, 28, 33]. It seems unlikely that transmigration of organisms from within the bowel lumen would occur without a preexisting bowel lesion. When there is an obvious intraabdominal source such as appendicitis, such a case could be correctly termed secondary pneumococcal peritonitis.

Pneumococcal peritonitis could also arise as a result of bacteremia secondary to infection elsewhere, specifically the respiratory tract, but there were only three reported cases with respiratory involvement [18, 31, 36], which provides scant support to the theory that pneumococcal pneumonia, bacteremia, or secondary peritoneal seeding give rise to pneumococcal peritonitis, at least in adults. However, cases of secondary hematogenous seeding from primary respiratory infections have been reported among children [3, 5]. Bacteremia is a consistent finding in cases of pneumococcal peritonitis in both children and adults [3, 10, 13, 18, 25–27, 29, 32, 34–36], but bacteremia may follow the peritoneal infection rather than precede it. It has been shown that bacteria can be recovered from the blood within minutes of an intraperitoneal injection in rabbits [37].

The management of pneumococcal peritonitis involves timely surgical intervention and treatment with antibiotics. Analysis of peritoneal fluid obtained via paracentesis may be helpful for distinguishing primary from secondary peritonitis, but this procedure cannot always be relied upon. When pneumococcal peritonitis occurs secondary to an intraabdominal lesion, a gram stain of the peritoneal fluid may show both gram-negative and gram-positive bacteria, and culture likewise may yield mixed growth [28], whereas in cases of primary pneumococcal peritonitis, both a gram stain and a culture show a single organism. Mixed organisms are not always found in cases of pneumococcal peritonitis secondary to appendicitis. In three reports, the organism was grown in pure culture [23, 28, 33]. There may be a role for diagnostic or therapeutic laparoscopy in the management of pneumococcal peritonitis [19, 21, 26], but it appears that in only a few cases is surgical intervention completely avoidable. The clinical features are nonspecific and the diagnosis is made at operation.

Antibiotic-resistant strains of *S. pneumoniae* have been identified worldwide, and the prevalence of these resistant strains is as high as 57% in some countries, although in the United States, Canada, and the United Kingdom, the prevalence of these strains remains low (<10%) [38]. In areas where the prevalence of resistant pneumococci is high, cefotaxime or ceftriaxone is the empirical therapy of choice [39, 40]. Susceptibility testing should be done to determine definitive antimicrobial therapy. For patients infected with penicillin-susceptible organisms, penicillin remains the preferred treatment.

In conclusion, pneumococcal peritonitis in adults without ascites or nephrotic syndrome is rare but is similar to that described in children since the beginning of the century. This infection is much more common in females and usually arises from the genital tract. Surgery plays a significant role in management, and with proper treatment, the outcome is excellent.

**References**