CASE REPORT

Streptococcus pyogenes primary peritonitis

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SUMMARY

Streptococcus pyogenes, a Gram-positive bacterium, is a rare cause of primary peritonitis. Diagnosed on imaging and with positive growth in blood cultures, a case of primary peritonitis caused by *S. pyogenes* is discussed here, with a brief literature review, and used to discuss several key principles of antibiotic use, including selection of antibiotic, investigations and non-pharmacological management of infection.

BACKGROUND

There are less than 50 case reports of primary *Streptococcus pyogenes* peritonitis and only a handful of literature reviews.^{1–3} We report here the first case documented in the Australian literature.

Similar to several reported cases, the case reported here did not undergo exploration by laparoscopy or laparotomy; however, the patient did receive a paracentesis.¹ This case adds to the existing sparse literature reported owing to its less severe presentation and non-surgical management. Owing to publication bias, this may more closely reflect the actual case definition.

CASE PRESENTATION

An otherwise healthy 46-year-old woman presented to a peripheral hospital with 2 days of diarrhoea, and 1 day of nausea, vomiting and fever. This followed several days of coryzal symptoms with sore throat that self-resolved a day prior.

On the day of admission, she developed generalised abdominal pain, which was worst around the right iliac fossa. On presentation, she had a temperature of 39.8°C, blood pressure of 104/69 mmHg and heart rate of 126 beats per minute. She had a soft and generally tender abdomen. Murphy's sign was negative and she did not have any focal tenderness. Examination of other systems was unremarkable.

INVESTIGATIONS

White blood cells were elevated on serology (15.9; normal 4–11×10⁹/L) as was C reactive protein (CRP; 34 mg/L) and lactate (2.9 mmol/L).

An abdominal CT scan with intravenous contrast demonstrated intra-abdominal stranding consistent with peritonitis.

Lipase, pregnancy test, liver function tests and electrolytes were within normal range. First catch urine gonorrhoea and chlamydia PCR were negative. Urine dipstick did not reveal nitrites. Urine leucocytes were within normal range, and there was no growth on culture.

She received fluid resuscitation and empirical intravenous ampicillin, metronidazole and gentamicin; however, her pain worsened over the next 24 hours, with worsening neutrophilic white cell count (39.4×10⁹/L), and CRP rise (334 mg/L). Blood cultures taken and inoculated in an aerobic and anaerobic culture demonstrated growth of *S. pyogenes* alone in both bottles.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis included appendicitis, ectopic pregnancy and enteritis. *S. pyogenes* peritonitis was diagnosed based on imaging findings and the results of the blood cultures.

CT is considered a reliable modality to exclude causes of secondary peritonitis,¹ and diagnosis based on blood cultures is consistent with most cases of diagnosed primary *S. pyogenes* (eg, 22/32 in a review of case studies); diagnoses have also been made from peritoneal fluid cultures (eg, 26/32), or rarely, vaginal or tissue culture.¹

TREATMENT

Antibiotics were rationalised to benzylpenicillin 2.4 g every 6 hours. Symptoms, haemodynamics and serology improved gradually over the next week.

Recovery was not uncomplicated. Symptomatically, breathlessness and posterior chest pain developed. Small pleural effusions were seen on chest X-ray, and a CT pulmonary angiogram did not demonstrate pulmonary emboli. Effusions were presumed secondary to intra-abdominal inflammation. Serologically, a cholestatic picture, without a rise in bilirubin, developed on liver function tests (Alkaline phosphatase 320, gamma-glutamyl transferase 341, alanine aminotransferase 39, aspartate aminotransferase 37 units/L), presumed secondary to antibiotics. CRP plateaued at about 137 mg/L. The antibiotic cover was broadened to piperacillin and tazobactam every 6 hours. Clindamycin was also added.

A progress abdominal CT scan revealed loculated intrapelvic collections, consistent with ascites. These were drained under ultrasound guidance. 150 mL of clear, slightly yellowish fluid was drained to symptomatic benefit, and sent for culture (polymorphs seen on microscopy; Gram stain and culture were negative). The serum-ascites albumin gap was 6; fluid albumin was 26 g/L (normal 35–52 g/L) and fluid lactate dehydrogenase was 998 units/L, associated with an exudative collection.

Following drainage, pain gradually improved. She remained afebrile. A thrombocytosis, to a maximum of 1162×10⁹/L (140–400×10⁹/L)

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developed, presumed reactive in origin, and the patient was commenced temporarily on aspirin prophylaxis. A repeat ultrasound scan revealed two ongoing intra-abdominal collections, decreasing in size but still measuring 70×96×47 mm (170 mL) and 87×80×44 mm (170 mL).

The patient was changed to two times per day intravenous amoxicillin and clavulanic acid, with an additional dose of amoxicillin added midday for additional streptococcal coverage. The general medical, general surgical and infectious diseases teams were involved in her care.

OUTCOME AND FOLLOW-UP

Abdominal and chest pain resolved, and the patient's blood-work normalised. The patient was stepped down to oral antibiotics and discharged, with instructions to complete a 4-week course of oral amoxicillin and clavulanic acid with supplemental amoxicillin.

The patient made an uneventful recovery and had no further complications at 1 year follow-up.

DISCUSSION

S. pyogenes, also called group A streptococcus, is a Gram-positive coccus most commonly associated with upper respiratory tract infections, cellulitis, acute rheumatic fever and glomerulonephritis.⁴ It is a rare cause of primary peritonitis (also called spontaneous bacterial peritonitis), which is defined as a peritoneal infection without an identified intra-abdominal source.⁵ The most common causes of primary peritonitis are *Escherichia coli*, *Klebsiella pneumoniae* and *Streptococcus pneumoniae*.¹⁻³

The infective source of primary peritonitis is postulated to stem from a haematogenous, lymphatic, bowel translocation or genital tract source in women.⁵ In a literature review of 23 articles, 13 patients had a likely source identified by investigators: genital tract (5 patients), pharyngitis (2 patients), soft tissue infections (1 patient), necrotising fasciitis (2 patients) and aerosol infection from children with group A streptococcal pharyngitis (3 patients).¹ *S. pyogenes* is a coloniser of the upper respiratory and female genital tracts, and the most common sites of infection of *S. pyogenes* are the upper respiratory tract and soft tissue.^{1,4,5}

Based on the history of upper respiratory tract infection, we presume here that the *S. pyogenes* relates to pharyngeal translocation. A source of infection is not identified in more than half of the reported cases,^{1,3} and more occult sources—such as insect bites, translocation through the bowel wall, haematogenous spread to the retroperitoneum with subsequent translocation to the peritoneum, or translocation through the genital tract—have been proposed to explain this.¹

Reviews of reported cases show a wide range of presentations, with and without septic shock and streptococcal septic shock syndrome—8/35 septic shock, 11/35 streptococcal shock syndrome in one analysis, with 2/35 presenting with necrotising fasciitis in one review of published cases.³ It is also possible that publication bias may skew these statistics towards more severe presentations. Few patients present following development of symptoms at apresumed primary site—identified in only 10/32 patients in one study—5/32 vaginal, 2/32 pharyngeal, 1/32 soft tissue infections and 2/32 retroperitoneal necrotising fasciitis.¹

Most cases of primary *S. pyogenes* peritonitis have been reported in women at a 4:1 ratio, with a median age of 32–38 years and a large reported age range^{1,3}; we report here a case of a woman aged 46. Previous peritonitis, cirrhosis, gastrointestinal haemorrhage and being on a protein pump inhibitor are risk factors for primary peritonitis; none of these were reported in this patient.⁴ A number of

cases of peritonitis have been reported in the context of dialysis in Australia.⁶

S. pyogenes carries an M protein on its cell membrane that acts as a virulence factor, conferring resistance to host phagocytosis.^{1,4} Its capsule is composed of hyaluronic acid, non-antigenic owing to its presence in host connective tissue.⁴ Exotoxins produced by *S. pyogenes* can act as superantigens. These bypass antigen-presenting cells and directly activate up to 20% of circulating T cells, causing a massive cytokine release in some patients.⁴ *S. pyogenes* may also cause necrotising fasciitis through the production of host-lytic enzymes, including deoxyribonuclease, haemolysin, proteases and collagenases.¹

Resuscitation with intravenous fluids, and treatment with broad-spectrum empirical antibiotics after the taking of blood cultures initially is a practical approach for the initial management for suspected streptococcal peritonitis.¹ Antibiotics should be directed based on likely microbes as soon as practicable. Antimicrobial sensitivities, tissue and abscess penetration, severity of infection and polymicrobial infections should all be considered at this point.⁷ Benzylpenicillin was initially selected as therapy in this case based on its tissue penetration and organism sensitivities. Clindamycin may also be given in toxic shock syndrome, with both antistreptococcal and antitoxigenic actions.^{1,8} In serious cases of septic shock, intravenous immunoglobulin has also been trialled, although evidence is limited to a small case series.⁸

Drainage of any abscess fluid collection is ideal. Antibiotic penetration and efficacy in abscesses is often poor, and drainage can help reduce bacterial load.⁷ Surgical exploration can also help exclude differential diagnoses such as perforated diverticula or appendicitis.¹ Response to treatment is also important to consider.⁷ *S. pyogenes* is highly sensitive to beta lactam antibiotics, and failure of antibiotics can be associated with the production of beta lactamase from nearby flora, poor tissue penetration and the presence of coinfections.⁹

Learning points

- *Streptococcus pyogenes* is an uncommon cause of primary peritonitis, most commonly seen in middle-aged women, and has a number of virulence factors contributing to its infectivity and presentation.
- Empirical antibiotics should be directed as soon as possible, and the source of an infection should be investigated.
- Antibiotic choice should be directed as possible against identified microbes, and based on sensitivities, tissue penetration and affected organs.

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