CASE REPORT

Primary group A streptococcal septic shock syndrome simulating perforated appendicitis in a previously healthy girl

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SUMMARY

A previously healthy 6-year-old girl that initially presented with presumed viral gastrointestinal infection for a week and later developed catastrophic primary peritonitis and septic shock requiring resuscitation and emergency exploratory laparotomy without any identifiable intra-abdominal cause of the sepsis and the peritoneal exudates grew group A streptococci. Appropriate antibiotic therapy was instituted and she made a complete recovery.

BACKGROUND

Primary peritonitis without an identifiable intra-abdominal source is exceedingly rare in healthy individuals and commonly seen in the cases of nephrotic syndrome, cirrhosis and end-stage liver disease, ascites, immunosuppression, inflamed peritoneum due to pre-existing auto immune and oncological conditions; but, in all cases, it is due to a single organism.¹ Group A streptococcus (GAS) commonly causes upper respiratory tract infection and cutaneous infections. It very rarely causes gastrointestinal infection and almost extremely rarely responsible for primary peritonitis and septic shock syndrome.^{2–8} We wish to report a 6-year-old previously healthy girl presenting with such odd combinations simulating acute appendicitis with perforative peritonitis.

CASE PRESENTATION

A 6-year-old girl was unwell with a 5-day history of vomiting, central abdominal pain, diarrhoea and fever on and off. The vomiting was persistent, non-bilious and did not contain any mucus. Diarrhoea was watery, non-formed, 7–8 stools per day. She did not have any skin rashes, respiratory or genitourinary symptoms. She was anorexic and felt headache on first day. She had no significant previous illnesses.

She was seen in the children's assessment unit by the surgical and medical teams. She looked ill and was flushed on the face with temperature of 36.4 (C, Pulse pulse of 132/min regular, respiratory rate of 28/m and blood pressure of 95/56 mm of Hg and oxygen saturations of 97% on air. Her chest was clear with equal air entry. She had mild peri-umbilical tenderness and the rest of the abdominal examination was unremarkable.

The patient was thought to have viral gastroenteritis with mild dehydration and was sent home with analgesics and rapid oral rehydration advice.

After 3 days, she suddenly worsened with acute severe generalised abdominal pain, large bilious vomiting 25/day, diarrhoea 20/day, complete anorexia, lethargy and disorientation. She was lethargic, listless, pale and grey with a pulse of 143/m, blood pressure of 90/60 mm Hg and respiratory rate of 26/min. Her abdomen was distended, tense, guarded and diffusely tender which was worse on movements. A clinical diagnosis of acute appendicitis with perforation and peritonitis was made.

INVESTIGATIONS

Laboratory investigations showed normal urine dipstick, haemoglobin 13.9 g%, white cell count of $19.3 \times 10^9/l$ neutrophils $16.06 \times 10^9/l$ platelets $323 \times 10^9/l$ and C reactive protein (CRP) 476 mg/l with normal electrolytes, urea and creatine. The culture of a stool sample was negative for *Campylobacter*, *Salmonella*, *Shigella*, *Vibrio* and *Yersinia* organisms. Her chest x-ray was normal. Throat swab, urine, blood and stool cultures were negative.

DIFFERENTIAL DIAGNOSIS

A differential diagnosis of perforative peritonitis and acute abdomen secondary to sepsis was entertained.

TREATMENT

She was resuscitated with fluids and reassessed. She was started on triple antibiotics consisting of cefuroxime, gentamycin and metronidazole for the presumed acute perforative appendicitis and septic shock following perforative peritonitis empirically after taking all investigations including blood cultures. It was an early therapeutic intervention before the exploratory laparotomy based on the presumed perforated acute appendicitis according to hospital policy for antimicrobial therapy for suspected appendicitis with perforation in the absence of available culture sensitivity report.

Surgical intervention in the form of exploratory laparotomy was part of the diagnostic exploration and investigation with a view to therapeutic intervention if there was a surgical cause—the commonest being the missed perforated appendicitis presenting with secondary peritonitis following perforation. The option of leaving appendix in or doing an appendicectomy should appendix be normal at exploration was discussed with parents and they opted for the second option that we should remove the appendix even if it was normal at exploration giving the benefit of anaesthesia and exploration.

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She underwent an examination under anaesthesia which did not reveal any mass. Exploratory laparotomy revealed gross peritoneal soiling of non-offensive pus and erythematous small bowel and appendix. No evidence of visceral inflammation, infarction or perforation was found. Multiple areas of superficial inflammatory exudates throughout the abdominal cavity were found. Delivery and lavage of bowel and appendectomy together with peritoneal lavage were carried out.

OUTCOME AND FOLLOW-UP

She required fluid boluses in the theatre and in the postoperative period. She had temporary renal and hepatic dysfunction in the postoperative period. On the third postoperative day, the microbiology department contacted us and informed that she has grown beta haemolytic GAS from peritoneal exudates sample.

The organism was sensitive to benzyl penicillin, erythromycin, cefuroxime and gentamycin. She was already on the last two antibiotics started preoperatively and the matter was discussed with the microbiology department and the paediatric medical teams, and the consensus was to continue the current antibiotics for 1 week and then give intravenous benzyl penicillin for a couple of days only to see the response and if she remained afebrile on this single antibiotic parentally, she then can be switched over to oral amoxicillin which we did and it worked well. The histology of excised appendix did not show changes of acute appendicitis. At 6-year follow-up, she remained completely asymptomatic and well. Repeat full blood count, x-ray of the chest, immunoglobulin levels, complements levels, total immunoglobulins, IgG subsets, functional antibodies and lymphocyte subsets were all within normal limits.

DISCUSSION

Primary peritonitis in a previously healthy child is extremely rare outside the neonatal period even within a published series of primary or invasive peritonitis.³ In most cases, it is caused by pneumococci and Gram-negative enteric bacilli. GAS causes skin and respiratory tract infections and it is very unusual for it to cause gastrointestinal symptoms and acute abdominal conditions as seen in our case. Only 10 cases of primary peritonitis in healthy adults have been reported and 7 of the 10 cases were in female patients.^{9–11}

GAS is neither a normal bowel flora nor associated with intra-abdominal sepsis. Recent reports indicate a trend towards an increased incidence of the disease. There are various theories put forward to explain the pathogenesis of primary group A streptococcal peritonitis. Principal theories include haematogenous spread from remote source in respiratory tract or skin, ascending route of infection in the female genital tract with asymptomatic vaginal carriers and translocation from gastrointestinal tract and intact bowel.

It is believed that the catastrophic manifestations of the disease are related more to profound inflammatory response by the super antigen production causing extensive lymphocyte activation and release of cytokines than to bacterial invasion or a direct bacterial toxin production. Dynamic interaction with host factors may influence the severity of the disease as well.

Penicillin is bactericidal and most effective during the logarithmic growth phase of bacterial replication. Another antibiotic inhibiting the bacterial protein synthesis and suppressing the

production of superantigens and antiphagocytic peptides, such as clindamycin, works synergistically. Intravenous polyspecific immunoglobulin G has been shown to possess neutralising activity against a variety of streptococcal superantigens and is associated with reduced mortality in some types of invasive GAS infection.¹²

Learning points

- ▶ Primary peritonitis in an otherwise healthy children is rare and group A streptococcus (GAS) is an unusual organism responsible for acute abdomen with toxic shock syndrome
- ▶ Clinicians should be aware of the possibility of the primary GAS peritonitis with gastrointestinal symptoms and septic shock syndrome mimicking acute appendicitis with perforative secondary peritonitis.
- ▶ Peritonitis may have an organism alien to the abdominal cavity in the absence of any symptoms of the normal tissues of invasion by the organism.
- ▶ Our case highlights the diagnostic and treatment dilemmas associated with GAS primary peritonitis-associated complication of toxic shock syndrome. Acute abdomen in children should have initial diagnostic laparoscopy if possible and feasible.

Contributors All authors have contributed to the active management of this patient, collecting data, writing the manuscript and collating the references and criticising constructively.

Competing interests None.

Patient consent Obtained.

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