Abstract Patients with severe acute respiratory syndrome (SARS) may present with extra-pulmonary symptoms. We report a 16-year-old adolescent with SARS who presented with diarrhoea. Treatment directed against SARS was prompted by an epidemiological link and the clinical picture as the disease evolved. This atypical presentation posed a diagnostic challenge for physicians. Conclusion: Proper disposal of patient excreta is important to prevent the spread of severe acute respiratory syndrome.

Keywords Atypical presentation · Diarrhoea · Infection control · SARS-associated coronavirus · Severe acute respiratory syndrome

Abbreviations RT-PCR: reverse transcriptase polymerase chain reaction · SARS: severe acute respiratory syndrome · SARS-CoV: severe acute respiratory syndrome-associated coronavirus

Introduction

Severe acute respiratory syndrome (SARS) is a highly contagious disease. Early identification is important not only in clinical management but also institution of proper infection control measures will reduce the chance of spreading the disease. Patients may not present with the classical presentation as defined by the WHO and the CDC. This case report describes an adolescent with SARS whose main presenting symptoms were gastrointestinal instead of respiratory in origin. In particular, the feature of watery diarrhoea was most prominent.

Case report

A 16-year-old adolescent was admitted to Princess Margaret Hospital, Hong Kong with high swinging fever up to 39.9°C for 5 days. He developed profuse diarrhoea with passage of greenish, watery, loose stools 1 day before admission. He also complained of dry cough, myalgia and general malaise. There was no running nose, chills, rigor, headache or shortness of breath. He resided in a housing estate (Amoy Garden) where a massive outbreak of SARS had occurred. There was no travel history or history of contact with SARS patients. General examination showed a febrile lethargic child with mild dehydration (ca. 5%). Examination of the abdomen showed mild epigastric tenderness but no organomegaly and bowel sounds were normal. There was no respiratory distress. Examination of the other systems was unremarkable. A chest X-ray film revealed right upper zone infiltrates (Fig. 1a). He was administered intravenous cefotaxime and oral clarithromycin to cover the usual organisms associated with community acquired pneumonia. Isolation in a negative pressure room was instituted in view of the epidemiological link to a SARS outbreak.

His fever persisted after admission and a maximum temperature of 40°C was recorded on day 6 from onset of illness. He refused oral intake as he vomited after every meal. A pulse-temperature deficit resembling that of typhoid fever was noted on the observation chart (Fig. 2). On day 6 from onset of fever, intravenous ceftriaxone, the empiric treatment of choice for typhoid fever in our locality, was administered in place of cefotaxime. Ribavirin was commenced for suspected SARS in view of the lack of clinical response to the initial antibiotic regimen. His general condition did not improve and he continued to run a high swinging fever with malaise, epigastric pain, and persistent vomiting.
and diarrhoea. On day 8, intravenous hydrocortisone was instituted because of radiographic progression of the right upper lobe consolidation (Fig. 1b).

Fever subsided 1 day after the initiation of hydrocortisone therapy and his general condition as well as the gastrointestinal symptoms improved rapidly. Radiological resolution was more gradual. Steroids were continued in the form of oral prednisolone after 4 days of intravenous hydrocortisone therapy and tailed off over 10 days. Ribavirin was given for a total of 10 days. Ceftriaxone and clarithromycin were discontinued after 7 days. He was kept under isolation and discharged on day 21 after onset of fever.

The initial total white cell count was 4.4×10^9/l (reference range 4.0–10.0×10^9/l) with absolute neutrophil and lymphocyte counts of 3.2×10^9/l (reference range 2.0–7.0×10^9/l) and 0.9×10^9/l (reference range 1.0–3.0×10^9/l) respectively. The lowest lymphocyte count was 0.6×10^9/l which occurred on day 13. The initial platelet count was 132×10^9/l (reference range 150–600×10^9/l). Reactive thrombocytosis up to 635×10^9/l was noted on day 21. Haemoglobin level and the clotting profile were normal. C-reactive protein, erythrocyte sedimentation rate, creatine kinase and lactate dehydrogenase levels were not elevated. Alanine aminotransferase level was elevated to 472 IU/l (reference range 1–40 IU/l) on day 13. Total bilirubin was elevated up to 37 μmol/l (reference range 4–20 μmol/l) on day 15.

No bacteria were recovered from stool and blood cultures. Widal serology was negative. Viral culture and reverse transcriptase polymerase chain reaction (RT-PCR) assay targeting severe acute respiratory syndrome-associated coronavirus (SARS-CoV) were performed on nasopharyngeal aspirates, stool and urine samples. RT-

Fig. 1  a Admission chest X-ray film 5 days from onset of disease showing right upper lobe consolidation.  b Chest X-ray film showing progression of right upper lobe consolidation on day 8

Fig. 2 Observation chart showing the pulse-temperature deficit from day 5 to day 7 and the temperature response to steroid treatment
PCRs were positive in nasopharyngeal aspirates collected on days 5 and 12. RT-PCRs was also positive in stool samples collected on days 8, 15, 19, 21 and 22. Viral culture was negative. Cold agglutinin titre was not raised and anti-mycoplasma IgM was negative. Paired acute and convalescent serum samples showed a rise in SARS-CoV titre (< 25 on day 2, 25 on day 32, and 400 on day 118). No increase in antibody titres to influenza A and B, parainfluenza virus types 1, 2 and 3, adenovirus, respiratory syncytial virus, *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* were demonstrated in convalescent serum samples.

**Discussion**

SARS was reported worldwide following an outbreak of atypical pneumonia, which occurred in Guangdong Province, People’s Republic of China in mid-November 2002 [14]. A novel coronavirus was shown to be the cause of the SARS epidemic [4, 5, 9]. Several case series were published documenting the clinical features and the severity of the disease [1, 3, 5, 10, 11]. The clinical case definition for SARS were fever, lower respiratory symptoms of cough, difficulty in breathing or shortness of breath, and radiological evidence of pneumonia as defined by the WHO and the CDC [12,13]. Our case report describes an adolescent with confirmed SARS whose main presenting symptoms were gastrointestinal instead of respiratory in origin. In particular, the feature of watery diarrhoea was most prominent. The implication of this atypical presentation is that the patient may escape clinical diagnosis with consequent influence on both treatment and infection control considerations.

In our patient, the main presenting features were persistent fever, vomiting, diarrhoea with dehydration and radiological evidence of pneumonia. The initial differential diagnosis included community acquired pneumonia with acute gastroenteritis and typhoid fever. SARS would not have been suspected early if the notable epidemiological link was absent. The lack of clinical response to empirical broad-spectrum antibiotic therapy and rehydration was an additional clue leading to suspicion of the diagnosis.

This case illustrates that SARS can present with prominent extra-pulmonary manifestations and may mimic gastrointestinal infections. The importance of searching for an epidemiological link and closely monitoring the patient for clinical progression as the disease unfolds cannot be overemphasised.

In reported series, the frequency of diarrhoea at presentation varies, from 9.5%, 20.3%, 20.5% to 73% [1, 6, 7, 8] in patients suffering from SARS. According to the report of the investigation into the outbreak of SARS at Amoy Gardens, Kowloon Bay, Hong Kong, diarrhoea was present in 66% of the 321 infected residents during the course of illness [2]. Although the diarrhoea was generally self-limiting, patients with diarrhoea were more likely to require ventilatory support and intensive care [5,7]. Biopsy of the small and large intestine in adult SARS patients have shown normal histology despite the findings of coronavirus particles on the surface of the gut mucosa when examined under electron microscopy [7]. No evidence of inflammatory enterocolitis was demonstrated. The diarrhoea caused by SARS-CoV is most probably secretory in origin.

SARS-CoV was identified by RT-PCR in the stool sample of our patient up to day 22 despite a negative viral culture. The likelihood of persistent excretion of infectious virus particles in the stools of convalescent patients remains to be determined as demonstration of live viruses by viral culture is limited by the relatively low sensitivity of the current method. Although there are no data concerning infectivity of patient urine or stool, proper handling and disposal of the patients’ excreta is an important infection control measure. Likewise, sanitation facilities, especially the sewage disposal system, need to be properly designed not only for hospitals, but for the community as a whole in the face of a possible threat of re-emergence of SARS.

This case illustrates an atypical presentation of SARS with prominent watery diarrhoea early in the illness. This knowledge is not only helpful in the early diagnosis and appropriate treatment of the patient but should lead to early hospitalisation and proper isolation to disrupt further transmission in the community as well as in the healthcare setting.

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