Acute Pancreatitis Associated with *Mycoplasma pneumoniae*: A Case Report of Missed Diagnosis

Abdulla Al-Abassi

Department of Surgery, Saqr Hospital, Ras Al-Khaimah, United Arab Emirates

Key Words

Acute pancreatitis · Extrapulmonary manifestations ·
Mycoplasma pneumoniae

Abstract

Objective: *Mycoplasma pneumoniae* is responsible for approximately 20% of community-acquired pneumonia. A wide variety of extrapulmonary manifestations related to *M. pneumoniae* infection can occur. The diagnosis of *M. pneumoniae* and its association with acute pancreatitis is briefly reviewed. **Clinical Presentation:** We describe the case of a 9-year-old boy with clinical features of acute abdomen. **Intervention:** A perforated appendix was suspected and the patient was accordingly operated on. The clinical, radiological and serological findings that led to the proper diagnosis are described. **Conclusion:** The case is presented to help increasing the awareness of *M. pneumoniae* infection as a potential cause of acute pancreatitis in children.

Copyright © 2002 S. Karger AG, Basel

Introduction

Mycoplasma pneumoniae is an important and treatable cause of community-acquired atypical pneumonia in children. It accounts for as many as 20% of all cases of community-acquired pneumonia in the general population [1, 2]. The clinical course of pneumonia due to *M. pneumoniae* is typically mild and self-limited [3]. There are, however, several case reports of acute pancreatitis and other severe complications following this illness with considerable morbidity and mortality [1–5]. In some of these cases respiratory manifestations may be absent [2].

To our knowledge, only a few proven cases of acute pancreatitis in association with *M. pneumoniae* have been reported [6].

Case Report

A previously healthy 9-year-old boy presented to his primary care physician with mild fever, dry cough, anorexia, mild central abdominal pain and occasional vomiting in relation to food. He was treated for upper respiratory tract infection with appropriate doses of Augmentin (amoxicillin; clavulanate potassium) and paracetamol as required. On the 4th day of his illness, abdominal pain was persis-

KARGER

Fax +41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

© 2002 S. Karger AG, Basel
1011–7571/02/0112–0112\$18.50/0

Accessible online at:
www.karger.com/journals/mpp

Dr. Abdulla Ahmed Al-Abassi
PO Box 3405, Ras Al-Khaimah Branch
Ras Al-Khaimah (United Arab Emirates)
Tel. +971 7 2361157, Fax +971 7 2222760
E-Mail alabassi@yahoo.com

Fig. 1. Acute pancreatitis associated with *M. pneumoniae* infection. Enhanced computed tomography of the abdomen with oral and intravenous contrast. A Pancreatic enlargement with irregular contour and areas of decreased attenuation (phlegmon), notably at its head. B Extrapancreatic fluid collection (phlegmon) with prominent thickening of the root of the mesentery and adjacent parts of jejunum and ileum.

tent, severe, and more prominent on the right side of the abdomen. He was referred to our surgical department to exclude appendicitis. On admission, he was mildly dehydrated, and his temperature was 38.8°C, pulse rate was 128 beats/min, and blood pressure was 110/70 mm Hg. Few discrete submandibular and upper cervical lymph nodes were palpable. His abdomen was not distended; there was ten-

derness and guarding all over the abdomen, maximally on the right side. Mild rebound tenderness was also present on percussion. His bowel sounds were absent. The findings on general physical examination were otherwise normal. Complete blood count revealed hemoglobin of 16.2 g/dl, a white cell count of $11.14 \times 10^3/\mu\text{l}$, and a hematocrit of 47.9%. Serum creatinin, albumin, bilirubin, and liver

enzymes were normal. The chest X-ray was normal, and abdominal X-ray film showed multiple air-fluid levels (suggestive of paralytic ileus). Abdominal ultrasonography revealed a free intraperitoneal fluid collection in the upper peritoneal recesses, iliac fossae, and pelvis. The pancreas could not be visualized because of small bowel gasses. Other intra-abdominal solid organs were normal.

Provisional diagnosis of acute abdomen was made, the most probable cause of which was thought to be a perforated appendix. The patient was operated on under general anesthesia through an incision centered at the right McBurney point. This corresponds to a point at the junction of the middle and outer thirds of a line joining the umbilicus to the right anterior superior iliac spine.

Approximately 400 ml of serous fluid was aspirated, a sample of which was taken for biochemical analysis and culture. The appendix looked normal. Few enlarged terminal ileal lymph nodes were noted. The appendix and a terminal ileal mesenteric lymph node were excised and sent for histopathological examination.

Piperacillin, 1 g intravenously, was started with induction of anesthesia and continued postoperatively 6-hourly for 5 days.

Acute pancreatitis was suspected at operation on the basis of clear ascitic fluid in the background of normal liver and kidney function tests. This was confirmed by a high peritoneal fluid amylase of 4,410 units/l. After surgery, serum amylase was 525 units/l, and lipid profile was normal. Computed tomography of the abdomen showed pancreatic enlargement with areas of decreased attenuation (phlegmon), notably at its head (fig. 1A). Extrapancreatic fluid collection was also noted. The root of the mesentery, the proximal jejunal and ileal loops were prominently thickened (fig. 1B). The overall picture was that of severe acute pancreatitis (CT grading III–IV) [7].

Histopathological examination revealed a normal appendix and features of reactive tissue hyperplasia with sinus histiocytosis of the excised mesenteric lymph node. Direct culture of a peritoneal fluid sample did not grow bacteria.

Follow-up computed tomography of the abdomen 6 weeks later showed marked improvement with regard to phlegmon collection, with no evidence of pseudocyst formation.

Serological screening revealed nonsignificant titers for influenza A and B, adenovirus, Q-fever and chlamydia antibodies. Measles and mumps IgM-specific antibodies were not detectable. *M. pneumoniae* antibodies were detected by complement fixation test. The antibody titer was 1:80 (acute infection is suspected if the titer is >1:20). Repeated serological test with particle agglutination test for *M. pneumoniae* 6 weeks later revealed a fourfold increase in antibody titer indicating an acute *M. pneumoniae* infection.

Postoperatively, the patient showed sustained improvement. He was discharged on the 12th postoperative day. He had no further symptoms or complications on regular follow-up over the subsequent 6 months.

Discussion

Acute pancreatitis is a relatively uncommon disease in children. In contrast to adults in whom 80% of cases of acute pancreatitis are associated with alcoholism and biliary disease, the etiology in children is diverse. Lerner et al. [8] summarized the etiological causes of acute pancreatitis in children as: idiopathic 22%, trauma 20%, infec-

tion 15%, biliary tract disease 14%, drugs 13%, miscellaneous 11% and congenital anomalies 5%.

M. pneumoniae has never been identified in pancreatic specimens, and the association of *M. pneumoniae* with acute pancreatitis is based on isolated case reports and serological surveys of patients with acute pancreatitis [6]. This is partly because *M. pneumoniae* are very tiny organisms (<350 µm in length) and are not visible at light microscopy. Unlike other *Mycoplasma* species, they grow more slowly and take 1–3 weeks before colonies can be detected [2]. Besides, there is deficient knowledge of the epidemiology and extrapulmonary manifestations of their infection, so that the diagnosis often is not considered [9]. A definite diagnosis of pancreatitis is based on evidence of pancreatitis at surgery or radiological evidence of pancreatitis by ultrasound and/or computed tomography, while a probable diagnosis of pancreatitis is based on characteristic symptoms and a threefold increase in amylase. A definite diagnosis of the etiological agent is based on identifying the organism in pancreatic tissue obtained by open or percutaneous biopsy, either by staining or culture, while probable evidence of the infection is determined by detection of the etiological agent in blood or pancreatic fluid and/or serological diagnosis based on a fourfold rise in antibody titer [6]. Based on this classification, our case proved to be a definite diagnosis of acute pancreatitis and a probable evidence of *Mycoplasma* infection being the etiological agent. Taking into consideration that other possible causes of acute pancreatitis have been excluded, our case clearly demonstrates the strong association between *M. pneumoniae* infection and acute pancreatitis.

The exact mechanism by which *pneumoniae* induce pancreatitis is not well established. Possible mechanisms include ascension of *Mycoplasma* organisms up the pancreatic duct from small bowel or biliary tree, seeding via the blood or lymphatic system, organ-specific toxin production, and autoimmune response to *Mycoplasma* infection [1, 6].

Appropriate antibiotic therapy for *M. pneumoniae* infection, usually erythromycin, clarithromycin, azithromycin or tetracycline, can help prevent spread of the infection to contacts, and limit the course of the respiratory disease in the patient [2]. In most cases, extrapulmonary manifestations are preceded by respiratory symptoms, and treatment of pulmonary disease seems to have little effect on subsequent nonrespiratory disease [2].

Conclusion

The laboratory results and clinical findings for our patient show that he had a recent *M. pneumoniae* infection and a concurrent episode of acute pancreatitis. No coincidental causes of acute pancreatitis were evident. Viral infections most commonly associated with acute pancreatitis have not been detected on serological screening. The findings in this case suggest that *M. pneumoniae* infection induced acute pancreatitis. We therefore believe that *M. pneumoniae* infection should be considered

among the possible causes of acute pancreatitis. It may also be considered as a rare possible cause of acute abdomen in children.

Acknowledgment

The author gratefully appreciates the cooperation of Dr. Mohamed Emad, MD, Head of Radiology Department, Dr. Farook Awad, MD, Director of Laboratories Department and Ms. Deirdre McEnery, AIMLT, Senior Charge Technician in Serology Laboratory, Saqr Hospital, Ras Al-Khaimah, United Arab Emirates.

References

- 1 Berger RP, Wadovsky RM: Rhabdomyolysis associated with infection by *Mycoplasma pneumoniae*: A case report. *Pediatrics* 2000;105:433–436.
- 2 John SD, Ramanathan J, Swischuk LE: Spectrum of clinical and radiographic findings in paediatric *Mycoplasma pneumoniae*. *Radiographics* 2000;21:121–131.
- 3 Kim CK, Chung CY, Kim JS, Kim WS, Park Y, Koh YY: Late abnormal findings on high-resolution computed tomography after *Mycoplasma pneumoniae*. *Pediatrics* 2000;105:372–378.
- 4 Dionisio D, Valassina M, Mata S, Rossetti R, Vivarelli A, Esperti FC, Benvenuti M, Catalani C, Uberti M: Encephalitis caused directly by *Mycoplasma pneumoniae*. *Scand J Infect Dis* 1999;31:506–509.
- 5 Perez C, Gurtubay F, Martinez-Ibanez F, Quesada P, Maravi E: More on polymyositis with *Mycoplasma pneumoniae* infection. *Scand J Rheumatol* 1999;28:125.
- 6 Parenti DM, Steinberg W, Kang P: Infectious causes of acute pancreatitis. *Pancreas* 1996;13:356–371.
- 7 Balthazar EJ, Robinson DL, Megibow AJ, Ranson JH: Acute pancreatitis: Value of CT in establishing prognosis. *Radiology* 1990;174:331–336.
- 8 Lerner A, Branski D, Lebenthal E: Pancreatic diseases in children. *Pediatr Clin North Am* 1996;43:125–156.
- 9 Baum SG: *Mycoplasma pneumoniae* and atypical pneumonia; in Mandell GL (ed): *Principles and Practice of Infectious Diseases*. Edinburgh, Churchill Livingstone, 2000, pp 2018–2025.