Case report

The patient was an 83-year-old female with a past medical history of paroxysmal atrial fibrillation and hypertension. She presented to the hospital with a 5 day history of shaking chills, fevers, a non-productive cough and haemoptysis. She denied any diarrhoea, abdominal pain, sick contacts, recent travel, recent hospitalization or antibiotic usage. She went to an outside hospital initially and was discharged home from their accident and emergency department with a presumed viral upper respiratory infection. She did not receive antibiotics and a chest X-ray was not performed. Her symptoms did not improve so she returned the next day to our accident and emergency department. Physical examination revealed a SpO2 of 98 % in moderate respiratory distress with accessory muscle use. Auscultation revealed crackles with decreased bibasilar breath sounds. Initial laboratory tests were: white blood cell count, 7.1 x 10^3 μl⁻¹; haemoglobin, 11.2 g dl⁻¹; platelets, 165 x 10^3 μl⁻¹; segmented neutrophils, 72 %; and band cells, 5.0 %. Her blood urea nitrogen was 54 mg dl⁻¹, creatinine 2.84 mg dl⁻¹ and glucose 148 mg dl⁻¹ with normal liver function tests. Analysis of urine was negative for infection. The N-terminal Pro-BNP was 13 300 pg ml⁻¹.

A chest X-ray showed a right lower lobe infiltrate consistent with pneumonia. Two sets of blood culture were obtained. Due to multiple antibiotic allergies (penicillin caused hives, trimethoprim–sulfamethoxazole caused a rash and hives, moxifloxacin caused an unknown reaction), she was started on azithromycin and clindamycin empirically and admitted to the hospital. Her initial CURB-65 score was 3, with a calculated mortality rate of 17 %.

On hospital day 1, two blood cultures grew Gram-negative bacilli. Intravenous aztreonam was initiated. Both urinary streptococcal and legionella antigen tests were negative. On hospital day 2, the patient’s respiratory condition worsened and she was transferred to the intensive care unit. She was noted to have a temperature of 38.7 °C. Azithromycin and clindamycin were discontinued after blood cultures revealed Y. enterocolitica sensitive to amikacin, aztreonam, ceftazidime, ceftriaxone, ciprofloxacin, doripenem, gentamicin, levofloxacin, meropenem, piperacillin–tazobactam, tobramycin and trimethoprim–sulfamethoxazole. An echocardiogram was completed which showed moderate mitral regurgitation without any vegetations. A computed tomography scan of the abdomen and pelvis with oral and intravenous contrast showed no acute colonic changes. Her condition improved and she was transferred back to the regular hospital ward on day 3. She was sent to an extended care facility on hospital day 8 for rehabilitation and to complete a 14 day course of intravenous aztreonam.

Discussion

Y. enterocolitica is a pleomorphic Gram-negative bacillus that belongs to the family Enterobacteriaceae. It is a lactose non-fermenter and can be differentiated from other enteric pathogens by its biochemical profile. Over 60 serotypes and six biotypes have been identified (Sihvonen et al., 2012). Our laboratory did not identify the serotype of the patient’s...
Y. enterocolitica is widely distributed in the environment and has been isolated from fresh water, contaminated foods and wild and domestic animals. It is commonly found in the gastrointestinal tract of pigs, and transmission to humans can occur through eating undercooked pork. While healthy adults and children are susceptible to infection, immunocompromised patients have a higher risk for septicaemia and severe illness. Identified risk factors include advanced age, diabetes mellitus, malignancy, immunosuppressive therapy, chronic liver disease, alcoholism and iron overload from blood transfusions (Adamkiewicz et al., 1998). We hypothesize that our patient most likely acquired the infection from contaminated food, although she had no specific recollection of the event. Y. enterocolitica produces β-lactamases which confer resistance to penicillin, ampicillin and first-generation cephalosporins. Treatment options include quinolones, aminoglycosides, tetracyclines and third-generation cephalosporins.

Y. enterocolitica rarely causes pneumonia. French investigators noted that only 15 cases had been reported up to the time of their study (Nicolas et al., 2005). Like our patient, theirs was elderly (a 70-year-old man), immunocompetent and did not have gastrointestinal illness. Since that report, another case has been identified (Girszyn et al., 2007). The patient was a 75-year-old man with diabetes mellitus who recovered after treatment with ofloxacin. Additional cases are detailed in Table S1, available in JMM Online.

In conclusion, pneumonia from Y. enterocolitica is very rare, especially in immunocompetent patients. Antibiotics for treating community-acquired pneumonia (CAP) such as fluoroquinolones and third-generation cephalosporins also treat this organism. However, antibiotic allergies are common (as in our case) and can present challenges for appropriate therapy. For this reason, we suggest that clinicians treat patients with severe CAP empirically, using antibiotics that have Gram-negative coverage until the underlying pathogen has been identified.

References


