**Case Report**

**Klebsiella pneumoniae** necrotizing fasciitis in a Latin American male

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Necrotizing fasciitis, caused by *Klebsiella pneumoniae*, is a rare and life-threatening bacterial infection. Most documented cases have been reported from Asia, particularly associated with diabetes mellitus. The prevalence of this infection in the USA is rare, especially among persons of non-Asian descent and those without travel to Asia. We report a case of disseminated necrotizing fasciitis, caused by *K. pneumoniae*, in a Latin American male with diabetes mellitus. Given our review of the literature, this is the only case report, to our knowledge, of a Latin American patient with *Klebsiella* necrotizing fasciitis in the USA. This case may reflect the geographical spread and emergence of *K. pneumoniae* infection in the USA. Clinicians need to be aware of the possible relationship between this organism and necrotizing fasciitis in persons of Latin American descent with diabetes mellitus.

**Introduction**

*Klebsiella pneumoniae* is a common human bacterial pathogen that can cause urinary tract infections, pneumonia and bacteremia. It is also recognized as causing primary liver abscesses and necrotizing fasciitis among patients in eastern Asia (Liu et al., 2005). Necrotizing fasciitis is a rare and life-threatening bacterial infection involving the deeper layers of skin and subcutaneous tissues. This type of infection is strongly associated with diabetes mellitus and dissemination to other organ systems (Wong et al., 2004). *K. pneumoniae* strains with the K1 and K2 capsular serotypes as well as the hypermucoviscosity phenotypes are highly associated with complicated, invasive disease (Yu et al., 2007, 2008). The occurrence of this infection in the USA is rare. Recently, a few cases of necrotizing fasciitis caused by this organism have been reported in the USA, especially among persons of Asian descent or those who have travelled to Asia (Kelesidis & Tsiodras, 2009; Gunnarsson et al., 2009; Kohler et al., 2007). We report a case of disseminated, monomicrobial necrotizing fasciitis caused by *K. pneumoniae* in a Latin American male with diabetes mellitus.

**Case report**

A 57-year-old Latin American male sought medical attention at our emergency department with a 7-day history of jaundice, right arm pain and swelling, and lower extremity oedema. He saw his primary care physician 3 weeks prior to admission with fever, nausea, vomiting and diarrhoea, and was diagnosed with acute viral gastroenteritis with a resolution of symptoms in a few days. Further history revealed recurrence of fevers with chills and sweats, cough with sputum production and increasing right arm pain and swelling with an erythematous rash. The patient did not seek medical attention, and used over-the-counter medications for fever, arm pain and rash. Past medical history was significant for untreated diabetes mellitus type 2 and hypertension. He had recently returned from a 1-month family vacation in Guadalajara, Mexico, without any prior travel to Asia. Our patient denied current usage of tobacco, alcohol and intravenous drugs.

Initial vital signs in the emergency department were temperature of 97.8 °F, heart rate of 115 min⁻¹, respiratory rate of 38 min⁻¹, oxygen saturation of 92 %, and systolic/diastolic blood pressure of 100/63 mm Hg. Examination revealed an obese, jaundiced, diaphoretic Latin American male in respiratory distress. The patient also had tachycardia, tachypnoea, jaundice, scleral icterus, right upper quadrant tenderness with palpation and 2+ lower extremity oedema to the knees. Further physical examination revealed severe right upper arm pain with passive movements of the shoulder and elbow joints. There was moderate erythema, extending from the antecubital fossa of the elbow to the axilla of the right upper arm. In addition, the arm was oedematous with positive signs of crepitus by palpation and auscultation.
Significant laboratory studies revealed a white blood cell count of 14.3 (4.3–11.3) cells ml\(^{-1}\), sodium of 121 (136–145) mEq l\(^{-1}\), creatinine of 4.1 (0.6–1.3) mg dl\(^{-1}\), random glucose of 349 (76–106) mg dl\(^{-1}\), total bilirubin of 10.1 (0.2–1.0) mg dl\(^{-1}\), alkaline phosphatase of 750 (50–136) IU l\(^{-1}\), alanine aminotransferase of 59 (12–78) IU l\(^{-1}\), lactate of 2.3 (0.4–2.0) mg dl\(^{-1}\), prothrombin time of 18.1 (11.0–13.4) s, international normalized ratio of 1.7 (0.9–1.2) and partial thromboplastin time of 43.4 (23.3–33.5) s. Urinalysis revealed high levels of bilirubin and trace ketones, a low level of leukocyte esterase, a negative nitrite test and a large number of white blood cells. Blood, sputum and urine cultures were collected in the emergency department before antibiotic administration. Significant radiological studies revealed bilateral lower lobe infiltrates on a portable chest radiograph, 3 cm hepatic fluid collection on the abdominal ultrasound, and diffuse subcutaneous emphysema and fascial oedema of the right upper arm, axilla, subscapular region and lateral chest wall on a computed tomography scan of the right upper extremity without intravenous contrast (Fig. 1).

Empiric antibiotic treatment with linezolid 600 mg intravenously every 12 h, metronidazole 500 mg intravenously every 6 h and a renal dosage of piperclillin–tazobactam 2.25 g intravenously were initiated. General surgery was consulted and the patient was quickly taken to the operating room for wide debridement of infected tissue. On surgical exploration, multiple tracking abscesses and subcutaneous necrosis involving the deep layers of fasciae were seen. The patient’s postoperative course was complicated by septic shock requiring multiple vasopressor medications, acute renal failure requiring haemodialysis, acute liver failure, disseminated intravascular coagulation and prolonged mechanical respiratory intubation and ventilation. The patient could not undergo ultrasound or computed-tomography-guided drainage of the hepatic fluid collection due to the above complications. The patient made a full recovery after 35 days in the intensive care unit and was transferred to the internal medicine ward for physical therapy and rehabilitation. All cultures of blood, wound, sputum and urine revealed 
\textit{K. pneumoniae} with similar antimicrobial susceptibility profiles. The isolates were resistant to ampicillin (MIC $\geq$32) and sensitive to cefazolin (MIC $\leq$4), cefoxitin (MIC $\leq$4), ceftazidime (MIC $\leq$1), ceftriaxone (MIC $\leq$1), cefepime (MIC $\leq$1), ertapenem (MIC $\leq$0.5), imipenem (MIC $\leq$1), gentamicin and tobramycin (MIC $\leq$1), and trimethoprim–sulfamethoxazole (MIC $\leq$20). Antibiotic treatment was changed to cefazolin 1 g intravenously every 12 h for a total of 6 weeks.

**Discussion**

Necrotizing fasciitis in the USA is a rare disease. After careful review of the literature, we believe that this is the only case report of a Latin American patient with monomicrobial, disseminated, community-acquired \textit{K. pneumoniae} necrotizing fasciitis in the USA. The only two published reports of necrotizing fasciitis in the USA were in persons of Asian descent or persons who had travelled to Asia (Gunnarsson \textit{et al.}, 2009; Kohler \textit{et al.}, 2007). Our medical facility, located in southern California, provides medical care to an increasing Latin American population. Over the past year, our facility has witnessed six additional cases of \textit{K. pneumoniae} infection in Latin American patients with liver abscesses, bacteraemia and soft tissue wound infections.

**Fig. 1.** Horizontal and frontal views on computed tomography without intravenous contrast of the right upper extremity that demonstrate diffuse subcutaneous emphysema shown by arrows, and underlying muscular oedema of the right upper arm with extension into the lateral chest wall fascia and muscles.
This serious infection can also be acquired in the hospital. There was a case of necrotizing fasciitis caused by K. pneumoniae in the USA during radiation therapy in a hospitalized patient (Kelesidis & Tsiodras, 2009). The case is unique because the patient was Native American and had never travelled outside the USA. Diabetes mellitus and his radiation therapy for cancer were risk factors for infection. Medical professionals need to be aware that radiation therapy in combination with diabetes mellitus and malignancy can result in the development of nosocomially acquired K. pneumoniae necrotizing fasciitis.

Migration and travel between Asia, Latin America and the USA may be possible contributors to the rising infection rates in the USA. K. pneumoniae strains with the K1 and K2 capsular serotypes and hypermucoviscosity phenotypes are highly associated with complicated, invasive disease in Asia (Yu et al., 2007, 2008). These virulent strains from Asia may be spread by colonization through migrating immigrants and travellers to Latin America and the USA. Historically, thousands of Chinese immigrants have travelled to Mexico. Currently, two large Chinese immigrant communities exist in Mexico City and Mexicali (Camacho, 2009). We believe that our patient may have been colonized with this organism from his recent visit to Mexico.

The incidence of disease is likely to increase, particularly in patients of Asian and Latin American descent. Medical professionals should be aware of the various presentations of K. pneumoniae infection as well as the associated risk factors such as diabetes mellitus, chronic liver disease, malignancy and travel to Asia and Latin America. Diabetes mellitus and his recent travel to Mexico may have predisposed our patient to this serious infection. Particularly, in patients with necrotizing fasciitis, clinicians should be vigilant in screening for disseminated infection. This case report illustrates that K. pneumoniae can disseminate to multiple organ systems such as the blood, liver, kidneys and lungs. We believe that our patient obtained necrotizing fasciitis from direct haematological seeding from the hepatic abscess. The fact that our patient had wide dissemination of the organism in the blood, lungs and urinary tract system supports a primary liver abscess, which is a common initial presentation in prior case reports.

In addition, the rising incidence of infection caused by invasive K. pneumoniae isolates requires better national and international surveillance of infection as well as the identification of highly virulent strains with the K1 and K2 capsular serotypes and hypermucoviscosity phenotypes. Given the wide extent of infection in our patient, we believe that he carried a highly virulent strain. A prerequisite for the surveillance of these invasive and life-threatening K. pneumoniae strains requires a more widespread testing capability of the capsular serotypes as well as the mucoid phenotypes. Typing of K. pneumoniae is not readily available at local laboratories and can be obtained by referral to reference laboratories. We stress the importance of local laboratories and clinicians using reference laboratories for further characterization of K. pneumoniae strains in the foreseeable future. We also recommend preserving bacterial strains that have caused serious infections for later use in detailed identification studies. With better awareness, early diagnostic and surgical interventions, and with appropriate empiric antibiotics, clinicians can make a substantial difference in increasing the survival rate of patients with this emerging invasive infection.

References


