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Case report

Metastatic community acquired *Klebsiella pneumonia* infection, secondary to skin and soft tissue infection: A case report

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ABSTRACT

Klebsiella pneumonia is known to cause hospital-acquired infections, primarily in immunocompromised patients. Recently, a distinct syndrome of community-acquired invasive *Klebsiella pneumonia* infection has been observed, mainly in the Southeast Asian population. This syndrome is associated with disseminated infection and the formation of multiple organ abscesses. Affected organs include the liver, the meninges, the brain, the eyes, and rarely the skin and soft tissue. Most of the affected patients suffer from diabetes mellitus. We present a case of invasive community-acquired *Klebsiella pneumonia* infection with the skin as the primary source. The patient was found to have multiple abscesses involving the skin, the liver, the right lung, and the brain. Cultures from the wound, the liver abscess, and the blood all revealed *Klebsiella pneumonia*. The liver abscess was drained, and the patient received a prolonged course of antibiotics based on the sensitivity. One month later, the patient achieved full recovery. Our report highlights the emerging syndrome of invasive community-acquired *Klebsiella pneumonia* infection and the need for timely diagnosis and treatment to achieve favorable outcomes.

Introduction

Klebsiella pneumonia (k. pneumonia) is a gram-negative aerobic rod-like bacterium. It was isolated in the late nineteenth century by Edwin Klebs, who identified it as a human pathogen. It is usually associated with hospital-acquired infections in immunocompromised patients. Less commonly, it can be community-acquired and causes pneumonia and urinary tract infections. Human beings are the main reservoir for *k. pneumonia* with a variable carrier state in the community, ranging from 5–38 % in stool samples and 1–6 % in the nasopharynx [1]. However, Klebsiella species are rarely carried on the skin [1].

Impaired host defenses (e.g., diabetes mellitus, malignancy, chronic obstructive pulmonary disease, alcoholism, renal failure, and steroid therapy) are well-known risk factors for K. pneumonia infections [2]. Also, certain virulence factors (e.g., K1, K2 serotypes) are associated with more severe infections [3]. Although *K. pneumonia*-related infections occur worldwide, a unique form of community-acquired invasive *K. pneumonia* infection was identified in East Asia, particularly Taiwan. This syndrome is associated with multiple organ abscesses that might involve the liver, eyes, central nervous system, lungs, and skin [4].

The most common pathogens to cause cellulitis are *beta-hemolytic Streptococci* and *Staphylococcus auris*. However, *K. pneumonia* has been reported as a causative agent, more commonly in Taiwan, with one series reporting it as the most common underlying pathogen for cellulitis [5]. *K. pneumonia*-associated skin and soft tissue infections are associated with a wide range of severity that might cause life-threatening necrotizing fasciitis with bacteremia and distant abscesses [6]. To the best of our knowledge, this is the second case of metastatic community-acquired *K. pneumonia* infection to be reported in the state of Qatar [7] and the first to identify the skin as the primary source.

Case presentation

A 49-year-old Bangladeshi male, who has been diagnosed with type 2 DM for 10 years. However, he was not adherent to his medications with the latest HbA1c of > 12 %, presented to our general hospital with a complaint of swelling on the back of the right shoulder for 15 days that

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was associated with fever and generalized weakness. A review of other systems was unremarkable at the time of admission. On general exam, he appeared sick; he was alert and oriented, and his vital signs included a pulse rate of 91 beats per minute, BP of 83/57 mmHg, respiratory rate of 22, SpO2 of 97 % on 3 liters oxygen by nasal cannula, and temperature of 38.5 *C orally. His skin revealed an ulcer with purulent discharge on the back of the right shoulder (Fig. 1). Further physical exam revealed no significant signs.

Investigations were reviewed and showed elevated inflammatory markers, C-Reactive Protein of 306 mg/L (normal range 0–5 mg/L), procalcitonin of > 100 ng/ml (normal range <0.5 ng/L), blood leukocyte of 12300/ μL (normal range 4000 −10000 / μL), neutrophil count of 10200/ μL (normal range 2000 – 7000 / μL), and platelet count of 125000/μL (normal range 150–410/μL). The patient suffered from an acute kidney injury and sepsis-related cardiomyopathy as evidenced by a creatinine of 123 μmol/L (normal range 62–106 μmol /L) and troponin-T of 468 ng/L (normal range 3–15 ng/L). Serial ECGs did not show signs of acute ischemia. AST and ALT were mildly elevated, and serum glucose was very high at 40 mmol/L (normal range 3.3 −5.5 mmol/L). However, the Beta-Hydroxybutyrate level was normal (0.18 mmol/L). ABG showed mixed respiratory alkalosis and metabolic acidosis with an elevated lactic acid level of 3.8 mmol/L (normal range 0.5–2.2 mmol/L). (Table 1).

The patient was admitted to the Medical Intensive Care Unit as a case of sepsis secondary to skin infection. Two sets of peripheral blood cultures and wound cultures were taken. We commenced him on empirical piperacillin/ tazobactam and clindamycin for the skin infection that is complicated by septic shock. He received intravenous fluids and an insulin infusion to control his blood sugar. Soft tissue ultrasonography of the wound did not show any sizable collection. The surgical team performed a debridement of the wound for source control. However, there were no signs of necrotizing fasciitis.

On the second day of admission, the patient became confused, and signs of meningism and left-third cranial nerve palsy were observed.

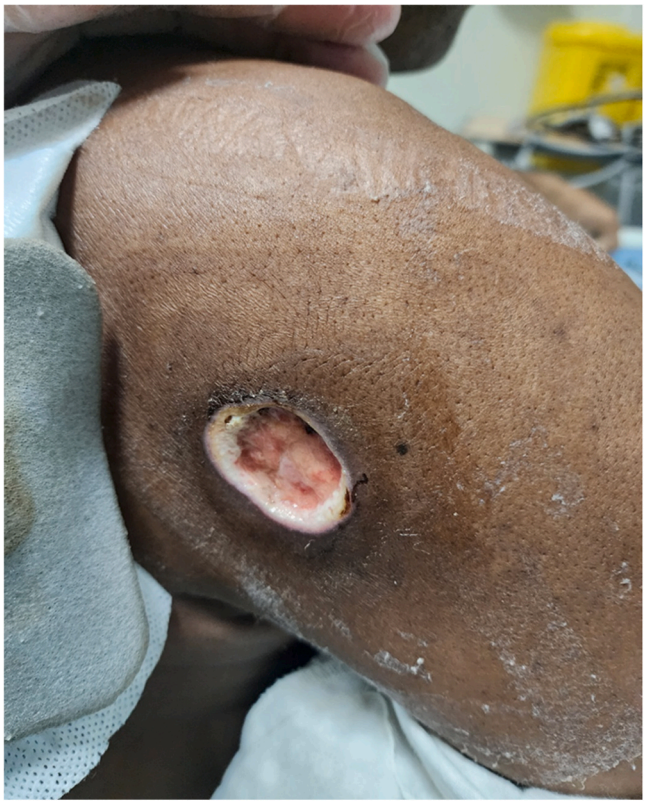

Fig. 1. Ulcer at the back of the right shoulder with purulent discharge.

Table 1
Summary of laboratory investigations at presentation.

Laboratory test	Presentation	Normal range
Hemoglobin (g/dL)	11.1	13 −17
White Blood Cell count (*10^3/μL)	12.3	4 −10
Absolute Neutrophil Count (*10^3/μL)	10.2	2 −7
Platelet (*10^3/μL)	125	150 −400
Urea (mmol/L)	10.8	2.5 −7.8
Creatinine (μmol/L)	123	62 −106
Bilirubin (μmol/L)	17	0 −21
ALT (U/L)	69	0 −41
AST (U/L)	80	0 −40
Alkaline phosphatase (U/L)	134	40 −129
C-reactive protein (mg/L)	306.9	0 −5
Procalcitonin (ng/ml)	> 100	< 0.5
Glucose (mmol/L)	40	3.3 −5.5
B-hydroxybutyrate (mmol/L)	0.18	0.03 −0.3
HbA1C %	> 12 %	< 5.7 %
Lactate (mmol/L)	3.8	< 2
Pro-BNP (pg/ml)	4392	< 125
Troponin T (ng/L)	468	3 −15

Contrasted brain CT was unremarkable, and the patient underwent a lumbar puncture. CSF analysis was consistent with bacterial meningitis and revealed a WBC of 10,850/ μL (normal range < 5/ μL) with 85 % neutrophils, a protein level of 1.85 g/L (normal range 0.15 – 0.45 g/L), and glucose level of 0.86 mmol/L (normal range 2.2 −3.9 mmol/L). CSF gram stain showed gram-negative bacilli. (Table 2). After reviewing the CSF analysis result, piperacillin-tazobactam was replaced by meropenem at a meningeal dose. Later, we deescalated the antibiotics to ceftriaxone based on antibiotic sensitivity in cultures. (Table 3).

Two ill-defined hypoechoic liver focal lesions were identified by Abdomen ultrasound, which was done to evaluate elevated liver enzymes. (1,5 *1.1 cm and 1.4 * 1.4 cm) (Fig. 2). Other investigations included transthoracic echocardiography, which showed mild global left ventricular hypokinesia with EF of 43 %; there were no valve vegetations. Contrasted CT abdomen confirmed the presence of multiple liver abscesses (the largest was 3 *2.8 *2.6 cm) (Fig. 3). Two small lung nodules in the right lung upper lobe (one with cavitation) alongside bilateral minimal pleural effusion were observed in the high-resolution chest CT (Fig. 4). Later, the blood, the wound, and the tissue cultures all grew *K. pneumonia*. No growth was observed in the CSF. Examination by the ophthalmologist ruled out endophthalmitis.

By the eighth day of admission, the patient's level of consciousness improved, and he became oriented. MRI brain with IV gadolinium showed multifocal lesions in supra and infratentorial parenchyma, consistent with multiple brain microabscesses (Fig. 5). A transesophageal echocardiogram ruled out any valvular vegetations. The largest liver abscess was drained on the tenth day of admission under the guidance of ultrasound because the patient continued to suffer from fever, inflammatory markers were trending up, and repeat blood cultures showed the persistence of *K. pneumonia* bacteremia. The culture of the drained liver abscess grew *K. pneumonia* as well.

On day 14, the patient was transferred to the medical ward to complete IV antibiotics. By day 24, the patient had completely recovered, and a repeat brain MRI showed the resolution of microabscesses

Table 2
Cerebrospinal fluid analysis on day 2 of admission.

CSF analysis	Result	Normal range
Total nucleated cells (cell/μL)	10856	< 5
RBC (cell/μL)	1011	0.0
Neutrophils%	85	0 −6
Lymphocytes%	11	40 −80
Monocytes%	4	15 −45
Protein (gm/L)	1.85	0.15 −0.45
Glucose (mmol/L)	0.86	2.22 −3.89
Gram stain	Scanty gram-negative bacilli	

Table 3
Blood culture antibiotic-sensitivity result.

Drug	MIC interpretation
Amoxicillin / clavulanate	Sensitive
Ampicillin	Resistant
Ceftriaxone	Sensitive
cefuroxime	Sensitive
Gentamicin	Sensitive
Trimethoprim/ sulfamethoxazole	sensitive

(Fig. 5). also, echocardiogram showed recovery of cardiac function with normalization of ejection fraction. The patient was discharged on day 30 with a plan for an additional four weeks of IV ceftriaxone in an outpatient IV room. Further follow-up could not be made as the patient had traveled to his home country.

Discussion

We described a case of community-acquired *K. pneumonia* skin and soft tissue infection complicated by bacteremia, metastatic infection, and multiple organ abscess formation. The rarity of such a syndrome in Qatar challenged our diagnostic approach. However, with early identification of *K. pneumonia* and the complications commonly associated with this infection, alongside source control and the use of proper IV antibiotics, the patient recovered with no long-term sequelae.

Metastatic *K. pneumonia* infection is usually associated with community-acquired primary liver abscess syndrome (KLA), defined as an abscess in the liver with a normal hepatobiliary tract. The above have

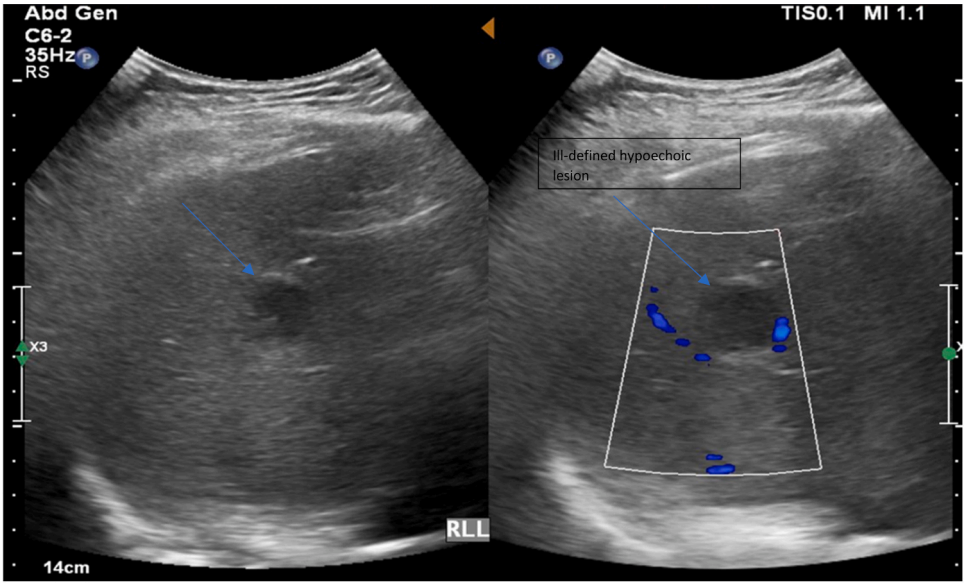

Fig. 2. Liver ultrasound showing an ill-defined hypoechoic focal lesion in the right liver lobe.

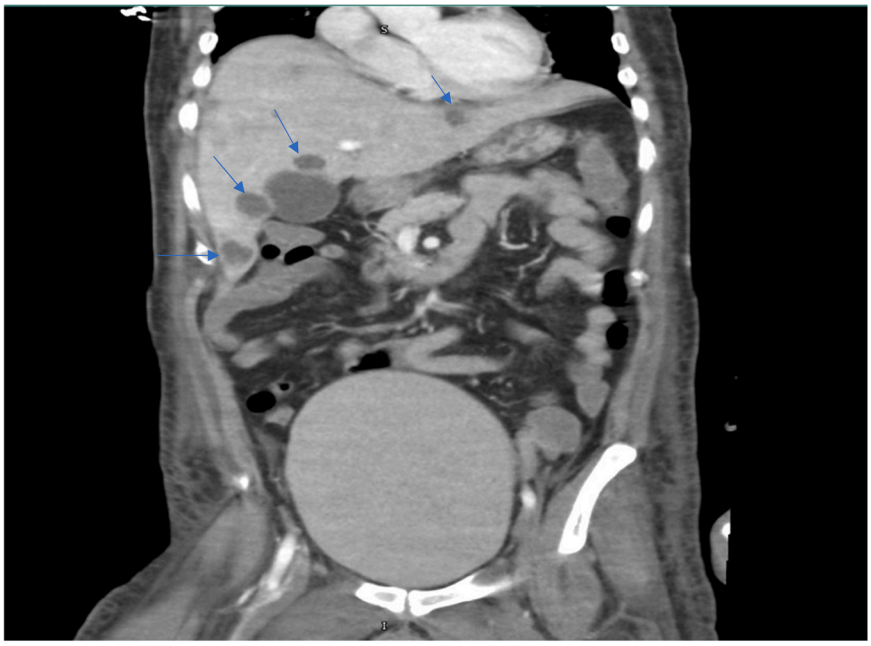

Fig. 3. abdomen CT with intravenous contrast showing multiple hypodense lesions in both lobes of the liver. Some are showing irregularity, conglomeration and ring enhancement.

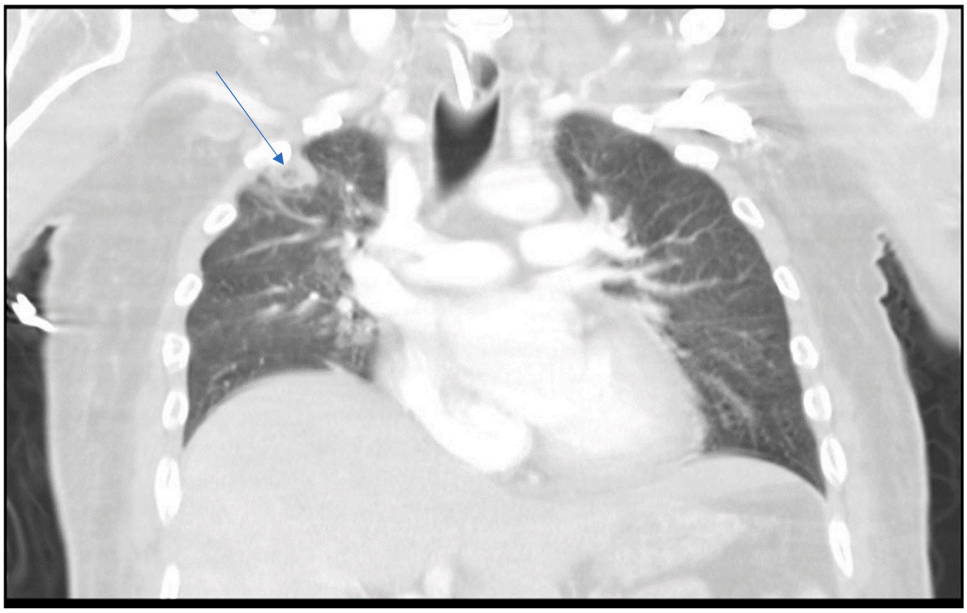

Fig. 4. Chest CT with IV contrast showing right upper lobe lesion with central cavitation.

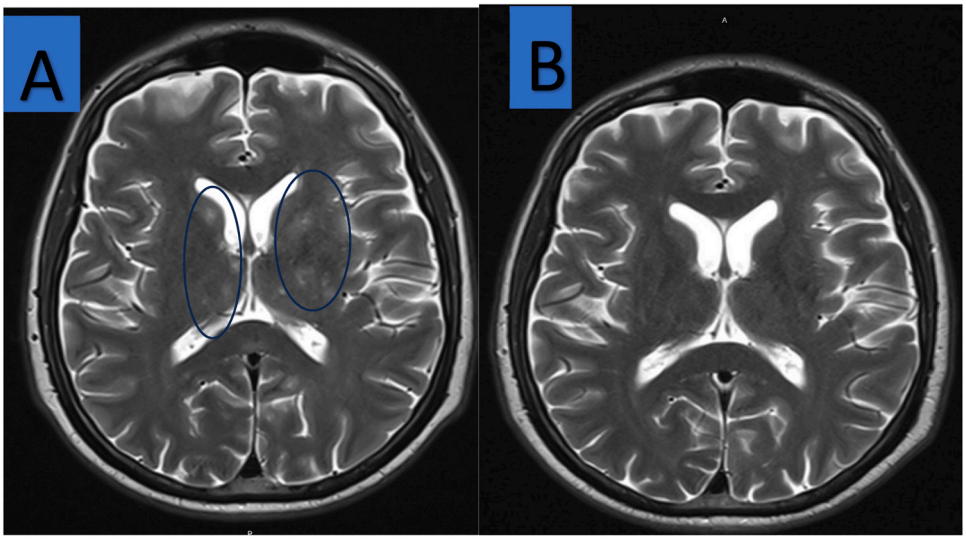

Fig. 5. MRI brain with intravenous gadolinium showing asymmetrical multifocal T2 hyperintense foci. Some are showing ring enhancement (A). Repeat MRI brain with IV gadolinium on day 24 showing resolution of microabscesses(B).

mostly been reported in Taiwan [8]. In a series from Twain, metastatic infection was reported in 12 % of the patients with KLA, most commonly manifested as endophthalmitis and meningitis. [9]. On the other hand, Community-acquired primary *K. pneumonia* skin and soft tissue infection is rare but was associated with a higher prevalence of bacteremia and distant abscess formation. In a small series of 15 cases of monomicrobial *K. pneumoniae* necrotizing fasciitis from Taiwan, concomitant bacteremia (80 %) and distant abscesses (27 %) were common [6]. Community-acquired KLA has also been reported in other Asian countries [10] and Asian patients living in other countries. [11], [12].

Diabetes mellitus is a known risk factor for KLA [13]. However, conflicting data have been reported regarding the association of DM with metastatic infection [4,14]. Until now, no human genes have been identified to predispose to KLA and metastatic *K. pneumonia* infection, but there is a possibility that genetic factors may play a role as the syndrome is observed mainly in Asian patients. Our patient is a Bangladeshi male who had a poorly controlled DM and a very high glucose level at presentation.

Several virulence factors have been described in community-acquired *k. pneumonia* infection. K1 and K2 capsular serotypes and hypermucoviscosity phenotypes were observed more in community-acquired *K. pneumonia* infections, particularly in Taiwan [4]. The K1 serotype has been identified as a risk factor for metastatic infection [4]. Other virulent factors associated with metastatic infection are the presence of magA gene [15] (mucoid-associated gene A) or rmpA [16] (regulator of mucoid phenotype) genes that are related to the hpermucovescosity phenotype. Further testing for the virulence factors was unavailable in our institution, which is a limitation of this case report. It might be essential to consider adding the test of virulent strains to the routine testing of invasive *K. pneumonia* infection.

Prolonged antibiotic course (4–6 weeks) and abscess drainage are the main treatments for invasive *K. pneumonia* infection. Although ESBL and carbapenemases-producing strains are emerging worldwide, hypervirulent strains that cause community-acquired disseminated infections are

generally susceptible to cephalosporins [17,18]. Furthermore, the possibility of CNS abscess formation should be investigated to determine the proper antibiotics (with good CNS penetration), the appropriate dose, and the duration of the treatment. Repeat imaging should be done to assess the response to therapy and the resolution of abscesses.

Primary *K. pneumonia* skin infection is a rare condition. However, it is commonly associated with bacteremia and distant abscess formation. Early identification and management of these serious complications are crucial to decrease the morbidity and mortality related to this infection. Additionally, it might be important to consider routine testing for virulence factors once *K. pneumonia* has been identified in cultures.

Ethical considerations

This work was conducted in accordance with the Declaration of Helsinki (1964). This case report was approved by the Institutional Review Board at Hamad Medical Corporation, Doha, Qatar (reference number MRC-04–24-351).

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CRediT authorship contribution statement

Mohammad Badawi Numan: Writing – review & editing, Writing – original draft, Supervision, Project administration, Data curation. **Joyal Mathew:** Data curation. **Wasfy Hamad:** Supervision. **Mohammad Abuhmaira:** Writing – review & editing. **Hassan Werah:** Data curation. **Almokhtar Khamkham:** Writing – original draft.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests Mohammad Badawi Numan reports administrative support was provided by Hamad Medical Corporation. Mohammad Badawi Numan reports financial support was provided by Qatar National Library. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Author contributions

All authors were involved in data collection, literature search, and manuscript writing. All authors read and approved the final manuscript.

Ethical approval

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Consent statement

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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Not applicable.

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