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Disseminated Klebsiella Pneumoniae Infection Presenting as Septic Shock, Hyperosmolar Hy-perglycemic Syndrome, Endogenous Endophthalmitis, and Multiorgan Failure: A Case Report and Literature Review

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Abstract **Case Report**

Hypervirulent Klebsiella pneumoniae infection is increasingly recognized as a cause of invasive syndrome characterized by bacteremia, liver abscess, metastatic infection, and rapid progression to septic shock, particularly among patients with diabetes mellitus. We report a 60-year-old man with poorly controlled diabetes mellitus who presented with hyperosmolar hyperglycemic syndrome with ketoacidosis, septic shock, acute respiratory failure, acute kidney injury requiring continuous renal replacement therapy, and disseminated Klebsiella pneumoniae infection involving the liver, lungs, urinary tract, bloodstream, pleural space, and bilateral eyes. Ophthalmologic examination demonstrated endogenous endophthalmitis with subretinal abscesses and Roth spots requiring intravitreal antibiotic injection. Despite broad-spectrum antibiotics, mechanical ventilation, vasopressor support, pleural drainage, and continuous venovenous hemofiltration, the patient developed refractory multiorgan failure and profound neurologic dysfunction. This case highlights the fulminant presentation of invasive K. pneumoniae syndrome in diabetic patients and underscores the importance of early recognition of metastatic complications, especially endogenous endophthalmitis, in critically ill patients with Gram-negative bacteremia.

Keywords: Klebsiella pneumoniae, Septic shock, Diabetes mellitus, Hyperosmolar hyperglycemic syndrome, Endogenous endophthalmitis.

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I. INTRODUCTION

Klebsiella pneumoniae has emerged as a major pathogen causing community-acquired invasive infections, particularly in East Asia but increasingly worldwide. Hypervirulent strains are associated with pyogenic liver abscesses and hematogenous dissemination to distant organs including the lungs, central nervous system, and eyes [1-3]. Diabetes mellitus is among the strongest predisposing conditions for invasive disease and metastatic infection [4,5]. Endogenous endophthalmitis caused by K. pneumoniae is associated with poor visual and systemic outcomes despite prompt intervention [6].

Septic shock accompanied by hyperosmolar hyperglycemic syndrome (HHS) and diabetic ketoacidosis (DKA) carries high mortality because of severe metabolic derangement and multiorgan dysfunction [7]. In critically ill patients, pulmonary complications, acute kidney injury, and disseminated intravascular inflammatory responses may rapidly progress despite guideline-directed care [8,9]. We report a case of invasive K. pneumoniae syndrome presenting with severe septic shock, hyperglycemic crisis, bilateral endogenous endophthalmitis, and refractory multiorgan failure.

II. CASE REPORT

A 60-year-old man with a history of type 2 diabetes mellitus, hypertension, and previously treated pulmonary tuberculosis presented to the emergency department after progressive generalized weakness and altered mental status for 2 to 3 days following an upper respiratory tract infection. According to his family, he developed poor appetite, worsening fatigue, and inability to ambulate independently. On the day of admission, he was found collapsed in the bathroom with dyspnea and

decreased responsiveness. There was no reported fever, vomiting, or diarrhea.

On arrival, the patient appeared critically ill. His Glasgow Coma Scale score was E2V1M4. Vital signs revealed a temperature of 35.4°C, blood pressure of 81/57 mm Hg, heart rate of 170 beats per minute, respiratory rate of 26 breaths per minute, and oxygen saturation requiring emergent ventilatory support. Physical examination demonstrated shallow tachypnea, cold extremities, weak peripheral pulses, and signs of poor perfusion. Initial laboratory evaluation demonstrated severe metabolic derangement, including serum glucose 797 mg/dL, serum osmolality 368 mOsm/kg, blood ketone level 5.0 mmol/L, arterial pH 7.255, bicarbonate 9.9 mmol/L, lactate 10.5 mmol/L, creatinine 2.84 mg/dL, blood urea nitrogen 81 mg/dL, sodium 148.5 mmol/L, potassium 2.80 mmol/L, platelet count 72 × 10^3/μL, and C-reactive protein 345.6 mg/L. Urinalysis demonstrated glucosuria, ketonuria, pyuria, bacteriuria, and positive nitrite testing. Chest radiography revealed bilateral pulmonary infiltrates and pleural effusions (Fig. 1). Computed tomography of the abdomen demonstrated multiple hypodense liver lesions with rim enhancement suggestive of liver abscesses or infected hepatic cysts (Fig. 2). Brain computed tomography revealed cerebral atrophy and intracranial atherosclerotic changes without acute hemorrhage.

The patient underwent aggressive fluid resuscitation, insulin infusion, electrolyte replacement, broad-spectrum antimicrobial therapy with piperacillin-tazobactam, vasopressor support with norepinephrine, and endotracheal intubation with mechanical ventilation. Continuous venovenous hemofiltration (CVVH) was initiated because of oliguric renal failure and severe metabolic acidosis. Blood cultures, urine cultures, and endotracheal aspirate cultures subsequently grew Klebsiella pneumoniae. Antimicrobial therapy was adjusted to cefoperazone-sulbactam and levofloxacin according to infectious disease consultation. During intensive care unit hospitalization, the patient remained profoundly encephalopathic. Neurologic examination revealed absent pupillary light reflexes, no spontaneous movement, and quadriplegia-like flaccidity. Electroencephalography was planned but delayed because of ongoing CVVH therapy. On hospital day 3, ophthalmologic consultation was obtained because of suspected ocular involvement. Examination demonstrated bilateral endogenous endophthalmitis with subretinal abscesses, diffuse retinal hemorrhages, and Roth spots. Intravitreal vancomycin and ceftazidime injections were administered bilaterally. The respiratory course was complicated by progressive pulmonary edema and bilateral pleural effusions. Serial chest radiographs showed worsening mixed alveolar-interstitial infiltrates, cardiomegaly, right upper lobe atelectasis, and bilateral pleural fluid accumulation. A right-sided pigtail catheter was inserted for drainage of exudative pleural effusion, yielding large volumes of yellow pleural fluid.

Despite treatment, the patient developed refractory vasopressor-dependent shock, persistent anuria, generalized edema, peripheral cyanosis, spontaneous bullae formation, thrombocytopenia, and worsening tissue hypoperfusion. Mechanical ventilation requirements increased to FiO2 70% to 100% with positive end-expiratory pressure support. After multidisciplinary discussion involving critical care, infectious disease, nephrology, ophthalmology, and palliative care teams, the family elected transition to comfort-focused care because of irreversible multiorgan failure and poor neurologic prognosis. Vasopressor escalation and further invasive monitoring were withheld, and hospice shared care was initiated.

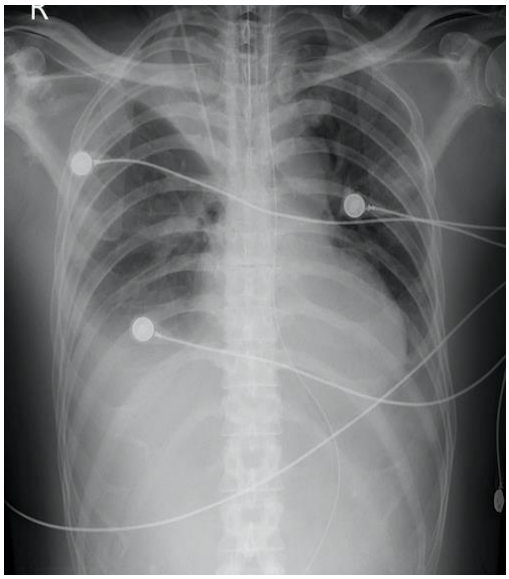


Figure 1: Chest radiography showed bilateral pulmonary infiltrates and pleural effusions especially right side

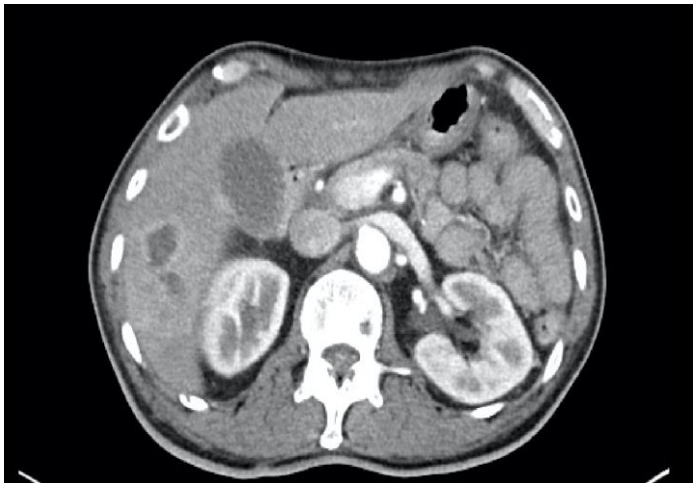


Figure 2: Based on the results of the abdominal computed tomography (CT) scan, there were multiple hypo-dense liver lesions with rim enhancement suggestive of liver abscesses or infected hepatic cysts

III. DISCUSSION

This case illustrates classic invasive K. pneumoniae syndrome with metastatic dissemination in a patient with diabetes mellitus. Since first described in Taiwan, invasive K. pneumoniae liver abscess syndrome has become increasingly recognized worldwide [1,2]. Hypervirulent strains frequently affect patients with diabetes and may disseminate hematogenously to the eyes, lungs, central nervous system, and soft tissues [3-5].

The patient exhibited several hallmark manifestations of invasive syndrome, including bacteremia, liver abscesses, pneumonia, urinary tract infection, pleural involvement, and endogenous endophthalmitis. Diabetes mellitus likely contributed to impaired neutrophil phagocytosis and increased susceptibility to invasive disease [10]. Hyperglycemia additionally promotes bacterial virulence and immune dysfunction [11]. Endogenous endophthalmitis is among the most devastating complications of K. pneumoniae bacteremia. Visual outcomes are generally poor because diagnosis often occurs late in critically ill patients [6,12]. Bilateral disease, as seen in this patient, is associated with particularly severe systemic infection and high mortality [13]. Subretinal abscesses and Roth spots suggested extensive hematogenous retinal seeding. The coexistence of DKA and HHS reflected profound metabolic stress from severe infection. Combined DKA-HHS presentations are associated with higher mortality than isolated diabetic emergencies [7]. Septic shock further exacerbated tissue hypoperfusion, acute kidney injury, and lactic acidosis. Respiratory failure in this patient was multifactorial, involving pneumonia, pulmonary edema, pleural effusions, and systemic inflammatory lung injury.

Prior studies in the American Journal of Respiratory and Critical Care Medicine have emphasized the prognostic implications of septic shock–associated respiratory failure and multiorgan dysfunction [8,14]. The need for escalating ventilator support and persistent vasopressor dependency indicated severe circulatory collapse. Acute kidney injury requiring continuous renal replacement therapy is common in severe septic shock and independently predicts mortality [15]. Persistent anuria despite CVVH suggested irreversible renal hypoperfusion and systemic microvascular dysfunction. The patient additionally developed severe peripheral cyanosis, generalized edema, and spontaneous skin bullae, indicating advanced circulatory failure. Current Surviving Sepsis Campaign guidelines emphasize early antimicrobial administration, hemodynamic resuscitation, and source control [9]. However, mortality remains high in invasive K. pneumoniae syndrome complicated by multiorgan failure. Early recognition of metastatic infection is essential, particularly in diabetic patients presenting with liver abscess and Gram-negative bacteremia.

This case also highlights the importance of integrating palliative care in refractory critical illness. Despite maximal supportive therapy, the patient's neurologic and hemodynamic deterioration indicated an extremely poor prognosis. Shared decision-making allowed transition toward comfort-focused treatment aligned with family wishes.

IV. CONCLUSION

Invasive Klebsiella pneumoniae syndrome should be suspected in diabetic patients presenting with septic shock and liver abscesses, particularly when metastatic complications develop. Endogenous endophthalmitis represents a vision-threatening and life-threatening manifestation requiring urgent ophthalmologic evaluation. This case demonstrates the fulminant progression of disseminated K. pneumoniae infection complicated by hyperosmolar hyperglycemic crisis, respiratory failure, renal failure, and refractory septic shock despite comprehensive intensive care management.

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