

Status epilepticus as the presenting manifestation of disseminated *Klebsiella pneumoniae* meningoencephalitis: A case report

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ABSTRACT

Central nervous system involvement in disseminated *Klebsiella pneumoniae* infection is uncommon and, when present, tends to cause severe neurological damage. We describe a previously healthy young individual who presented with status epilepticus requiring mechanical ventilation. Magnetic resonance imaging of the brain showed diffuse meningoencephalitis, and electroencephalogram showed diffuse background slowing with generalized suppression. On workup, poorly controlled, previously undiagnosed diabetes was found, along with two separate infective foci – a liver abscess and a perianal wound – both of which grew *K. pneumoniae* with identical antimicrobial susceptibility profiles, confirming hematogenous dissemination. The patient made a full recovery following antibiotics, source control, and intensive care support. This case illustrates a rare neurological presentation of invasive *Klebsiella* infection, with evidence of true dissemination, and argues for a thorough workup whenever status epilepticus appears without any obvious cause.

Key words: Diabetes mellitus, *Klebsiella pneumoniae*, Liver abscess, Meningoencephalitis, Status epilepticus

Status epilepticus is a neurological emergency with a wide range of causes – structural, metabolic, autoimmune, and infectious [1]. *Klebsiella pneumoniae* is a well-documented cause of invasive disease, particularly the pyogenic liver abscess syndrome, but it rarely affects the central nervous system (CNS) [2]. *Klebsiella* meningitis and meningoencephalitis remain infrequent clinical findings, particularly in patients who appear otherwise immunocompetent at presentation [3]. Status epilepticus as the initial presenting feature of disseminated *Klebsiella* infection is rare, with only a few cases documented in the literature [4].

We report this case to draw attention to this unusual presentation and to emphasize why we should actively screen for occult systemic infection when managing patients with unprovoked seizures or status epilepticus.

CASE REPORT

A previously healthy young Indian man without any comorbidities was transferred to our center on mechanical ventilatory support in view of new-onset status epilepticus. His seizures were controlled with levetiracetam and lacosamide, followed by maintenance therapy.

On physical examination, he had neck rigidity, pointing toward meningeal irritation. Initial blood work revealed anemia, thrombocytopenia, and leukocytosis. His metabolic panel showed severe hyperglycemia with a hemoglobin A1c of 12%, revealing previously undiagnosed diabetes mellitus.

Due to the clinical picture of meningoencephalitis with uncontrolled diabetes, empiric broad-spectrum antibiotics were started (ceftriaxone and vancomycin). Lumbar puncture was briefly delayed due to low platelet count but was safely performed on day 2 following a platelet transfusion. Cerebrospinal fluid (CSF) analysis showed a cell count of 105 cells/ μ L (85% polymorphonuclear leukocytes), elevated protein of 143 mg/dL, and glucose of 123 mg/dL against a random blood glucose of 348 mg/dL. Adenosine deaminase levels were normal, and a tuberculosis polymerase chain reaction was negative. CSF cultures and diagnostic stains, including Gram, India ink, and Ziehl–Neelsen stains, were negative, as were blood cultures sent concurrently. The CSF profile strongly suggested pyogenic meningitis; antibiotics were therefore escalated to intravenous meropenem for its CNS penetration, together with vancomycin, alongside a basal-bolus insulin regimen to control his blood sugars.

Contrast-enhanced brain magnetic resonance imaging (MRI) demonstrated diffuse leptomeningeal enhancement

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(Fig. 1). Bedside electroencephalogram (EEG) monitoring showed diffuse background slowing with generalized background suppression and no epileptiform discharges, in keeping with a diffuse encephalopathic process. Given the systemic inflammatory response, a workup to identify the underlying primary source of infection was undertaken. An abdominal ultrasound revealed an ill-defined hypoechoic lesion in the liver measuring 65×47×49 mm, with an approximate volume of 95 mL, containing internal liquefaction. This confirmed a pyogenic liver abscess (Fig. 2). After obtaining informed consent, percutaneous drainage was performed using a pigtail catheter.

The aspirated fluid was thick and purulent (Fig. 3). Cultures of this aspirate grew *K. pneumoniae*, sensitive to first-line antimicrobials (Table 1). Once *K. pneumoniae* was confirmed as the sole causative organism, vancomycin was discontinued, and intravenous tigecycline was added for its penetration at the infective foci, whereas meropenem was continued for CNS coverage. A subsequent examination revealed a perianal sinus opening at the 6 o'clock position, approximately 1 cm from the anal verge, with minimal discharge and no induration; as there was no discrete collection requiring surgical drainage, a wound swab was taken and sent for culture and sensitivity. This grew *K. pneumoniae* with an antimicrobial susceptibility profile identical to that of the liver abscess (Table 1), confirming hematogenous dissemination from a single source.

The patient was managed with intravenous antibiotics, antiepileptics, intensive care support, and glycemic control. As his neurological status stabilized, he was successfully extubated following a weaning trial. His sensorium improved gradually, and he made a full recovery to a Glasgow Coma Scale of 15. He was discharged home with the pigtail catheter in place, which was removed on an outpatient basis 7 days later.

DISCUSSION

Invasive *K. pneumoniae* infection has been on the rise across Asia, especially among diabetic patients. While the pathogen can affect almost any organ, it characteristically presents as a liver abscess with a high tendency for hematogenous seeding to distant sites [2,5]. A recent systematic review of 53 cases of hypervirulent *K. pneumoniae* liver abscess reported CNS involvement, most commonly brain abscess or meningitis, in 15% of patients [6]. Neurological spread is nonetheless rare relative to the overall burden of invasive disease, usually manifesting as a localized brain abscess or meningitis [3,7]. Diffuse meningoencephalitis presenting as status epilepticus, as seen in our patient, is a distinctly rare presentation [3,4].

The diagnosis of meningoencephalitis in our patient was supported by a combination of findings rather than any single test: Diffuse leptomeningeal enhancement on contrast-enhanced MRI, a CSF profile consistent with

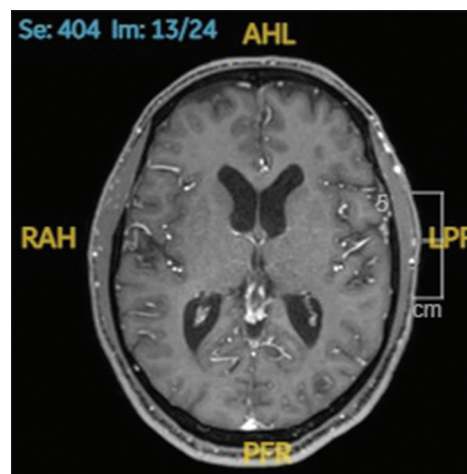


Figure 1: T1 post-contrast magnetic resonance imaging brain (axial) showing diffuse leptomeningeal enhancement

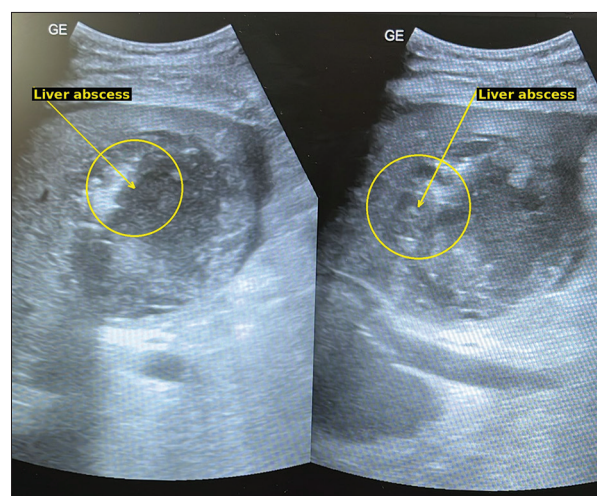


Figure 2: Abdominal ultrasound showing liver abscess (circled, with arrow) in two imaging planes

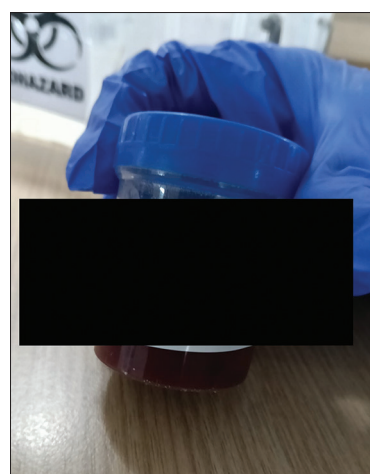


Figure 3: Gross appearance of the purulent liver abscess aspirate (specimen label redacted to protect patient identity)

pyogenic meningitis, and bedside EEG showing diffuse background slowing with generalized suppression, indicating widespread cortical dysfunction. This combination of imaging, CSF, and electrophysiological findings is consistent with meningoencephalitis in the appropriate clinical context.

A recently reported case described a diabetic patient whose seizures progressed to refractory status requiring

Table 1: Antimicrobial susceptibility (MIC) of *Klebsiella pneumoniae* isolated from the liver abscess aspirate and the perianal wound swab

Antibiotic	Liver abscess isolate MIC (µg/mL)	Interpretation	Perianal swab isolate MIC (µg/mL)	Interpretation
Amikacin	≤1	S	≤1	S
Amoxicillin/Clavulanic acid	≤2	S	≤2	S
Cefepime	≤0.12	S	≤0.12	S
Cefoperazone+Sulbactam	≤8	S	≤8	S
Ceftriaxone	≤0.25	S	≤0.25	S
Cefuroxime	2	S	2	S
Ciprofloxacin	≤0.06	S	≤0.06	S
Colistin	≤0.5	I	≤0.5	I
Ertapenem	≤0.12	S	≤0.12	S
Imipenem	≤0.25	S	≤0.25	S
Meropenem	≤0.25	S	≤0.25	S
Piperacillin+Tazobactam	≤4	S	≤4	S

S: Sensitive, I: Intermediate, MIC: Minimum inhibitory concentration

intubation, in the setting of *Klebsiella* meningitis and a liver abscess [8]. Our case differs from this in two important respects: Neuroimaging here documented diffuse leptomeningeal enhancement, and dissemination was confirmed by culturing the identical organism, with matching antimicrobial susceptibility profiles, from two separate extra-neural sites, i.e., a liver abscess and a perianal wound. This distinction provides evidence of a genuinely disseminated, hematogenously spread infection rather than a localized process with secondary meningeal seeding.

Our case stands out for several other reasons. First, the patient's primary presentation was status epilepticus, a presentation whose differentials include viral encephalitis, autoimmune conditions, or toxic-metabolic derangements rather than an occult bacterial infection [1]. Second, the neuroimaging was atypical, showing diffuse meningoencephalitis rather than the focal, space-occupying parenchymal abscesses more typically seen with *Klebsiella* CNS disease [3]. Third, because the patient had no prior history, this fulminant presentation served as the unmasking event for his severe, underlying metabolic disease [2,7]. Finally, isolating the identical pathogen, with matching susceptibility profiles, from two separate, deep-tissue extra-neural sites provides clear microbiological evidence of an aggressive, disseminated syndrome, reflecting the evolving spectrum of neurological infections increasingly reported in India [4,9,10].

Recognizing this atypical presentation early allowed the treating team to quickly control seizures, start targeted antibiotics, achieve source control at both foci, and correct the patient's metabolic profile, which likely contributed to his favorable outcome.

CONCLUSION

Disseminated *K. pneumoniae* infection should therefore be kept on the differential diagnosis for patients presenting with acute meningoencephalitis and status epilepticus,

particularly when concurrent, uncontrolled diabetes is seen. Prompt systemic screening and aggressive source control remain vital to ensure a favorable outcome.

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CONTRIBUTORS DETAILS

Harsh Bhardwaj (HB) was responsible for the concept and design of the report, clinical management of the patient, acquisition and interpretation of data, drafting and critical revision of the manuscript, and final approval of the version to be published. Nirja Pandey (NP) and Dinesh Sareen (DS) contributed to clinical management of the patient and critical revision of the manuscript for important intellectual content, and approved the final version to be published. HB is the guarantor of this work and takes responsibility for the integrity of the content.

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