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SHORT COMMUNICATION

Unveiling a rare killer: Community-acquired *Escherichia coli* K1 meningitis in an adult patient

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ABSTRACT

Escherichia coli (E. coli)-induced community-acquired meningitis in adults is exceedingly rare, with an average of only one annual case reported worldwide. The majority of *E. coli* meningitis cases in adults are associated with healthcare settings, typically following head injuries or neurosurgical interventions. Nonetheless, spontaneous *E. coli* meningitis in adults can arise in individuals with various comorbid conditions. This case report details the clinical presentation, diagnostic workup, management, and eventual outcome of a 45-year-old male with significant comorbidities who presented with severe respiratory distress, altered mental status, and generalized tonic-clonic seizure. Subsequently, multiplex PCR analysis of cerebrospinal fluid revealed *E. coli* K1. Despite aggressive management, including broad-spectrum antibiotics, the patient deteriorated and succumbed to his illness. This report contributes to the understanding of *E. coli* K1 as a lethal pathogen in adult meningitis, emphasizing the need for vigilance in diagnosing and treating this condition.

Keywords: *Escherichia coli* K1; adult; meningitis; community-acquired; multiplex PCR.

INTRODUCTION

Escherichia coli (E. coli) K1 is a pathogen that is widely recognized in neonatal meningitis; however, its occurrence in adult meningitis is rare and frequently associated with healthcare settings following neurosurgical procedures or head trauma (Mir *et al.*, 2023). The pathogenesis of *E. coli* meningitis in adults can be multifactorial, with risk factors involving patients with significant comorbidities (Bichon *et al*., 2018). This case report presents an unusual case of fatal community-acquired *E. coli* K1 meningitis in an adult, emphasizing the importance of recognizing atypical pathogens in adult patients with meningitis symptoms, particularly in the context of underlying health issues that may predispose them to invasive infections.

CASE REPORT

This case involves a 45-year-old male with a complex medical history, including diabetes mellitus, hypertension, dyslipidemia, obstructive sleep apnea (OSA), bronchial asthma, and obesity. He presented to the Emergency Department (ED) with a three-day history of fever, cough, lethargy, and poor oral intake. Upon further history, there were no episodes of persistent diarrhea, abdominal pain, or other gastrointestinal symptoms. At the time of presentation, the patient exhibited a decreased level of consciousness with a Glasgow Coma Scale (GCS) score of E2V2M5. Vital signs revealed hypertension (BP: 152/88 mmHg), hypoxemia (SpO2: 89% on a high-flow mask), tachycardia (HR: 113 bpm), and fever (T: 39.4°C) and fingertip glucose of 12.9 mmol/L. Lung examination revealed generalized rhonchi with equal air entry in the lungs. A chest X-ray indicated haziness in the right lower zone, suggestive of pneumonia. Arterial blood gas (ABG) analysis demonstrated a type 2 respiratory failure with severe acidosis (pH 7.092), hypoxemia (pO_2 75.2 mmHg), and hypercapnia (pCO_2 91.1 mmHg), bicarbonate level of (27.1 mmol/L) alongside a high lactate level of 2.8 mmol/L.

Initial management included nebulized salbutamol and ipratropium bromide/albuterol sulfate as well as intravenous magnesium sulfate. In the ED, the patient suddenly developed a generalized tonic-clonic seizure which resolved spontaneously after approximately 30 seconds. Due to impending respiratory collapse, the patient was intubated and subsequently admitted to the Intensive Care Unit (ICU). Laboratory investigations revealed a slightly elevated total white blood cell count of 10.1 x 10^9/L with increased monocyte count of 1.88 x10^3/µL, hemoglobin of 17.5 g/dL, and platelet count of 242 x 10^9/L. Renal profile was deranged with elevated urea (9.9 mmol/L), hyponatremia (sodium: 128 mmol/L), hyperkalemia (potassium: 5.3 mmol/L) and high creatinine (246 µmol/L). C-reactive protein (CRP) was markedly elevated at 167 mg/L (Table 1). The initial working diagnosis was suspected meningoencephalitis, acute kidney injury with hyponatremia, and Type II respiratory failure secondary to community-acquired pneumonia (CAP). He received intravenous ceftriaxone, azithromycin, acyclovir, and isotonic electrolyte solution

Table 1. Full blood count and serum biochemistry

Full Blood Count	Value	Unit	Reference Range
Total White Cells	10.1	10^9/L	4.0 -10.0
Neutrophils	6.56	10^3/µL	2.0-7.0
Lymphocytes	1.66	10^3/µL	1.0-3.0
Monocytes	1.88	10^3/µL	0.2-1.0
Eosinophils	0.00	10^3/µL	0.02-0.5
Basophils	0.02	10^3/µL	0.02-0.1
Hemoglobin	17.2	g/L	13-17
Platelet	302	10^6/L	150-400
Serum Biochemistry Test	**Value**	**Unit**	**Reference Range**
Urea	15.1	mmol/L	3.2-8.2
Sodium	128	mmol/L	136-145
Potassium	5.3	mmol/L	3.4-4.5
Creatinine	246	µmol/L	62-115
Total Protein	74	g/L	57-82
Albumin	38	g/L	32-48
Bilirubin	4	µmol/L	3-19
Alanine Transaminase (ALT)	28	U/L	10-49
Alkaline Phosphatase (ALP)	111	U/L	46-116
Aspartate Transaminase (AST)	88	U/L	<34
C-Reactive Protein (CRP)	170.36	mg/L	0-5
Random Glucose Level	10	mmol/L	>11

Table 2. CSF biochemistry

CSF Biochemistry Test	Value	Unit	Reference Range
Protein	0.44	g/L	0.15-0.44
Chloride	118	mmol/L	119-129
Glucose	5.5	mmol/L	
CSF/serum ratio for Glucose	0.55	–	>0.4

Table 3. Microbiology test

Microbiology Test	Result
Aerobic Blood Culture and Sensitivity	No Growth
Anaerobic Blood Culture and Sensitivity	No Growth
CSF Culture and Sensitivity	No Growth
Cryptococcal Antigen Lateral Flow	Negative
India Ink	No encapsulated yeast seen
Mycobacterium Culture and Sensitivity	No Growth
Tracheal Aspirate Culture and Sensitivity	No Growth
Urine Culture and Sensitivity	No Growth
Bronchoalveolar lavage Culture and Sensitivity	No Growth
HIV Ag/Ab, HBsAg, Anti Hep C	Non-reactive

as part of his initial management. Non-enhanced CT scan of the brain demonstrated mild cerebral edema with no hydrocephalus (Figure 1). A lumbar puncture was performed the following day after antibiotic administration, revealing a clear cerebrospinal fluid (CSF). The CSF biochemistry showed a protein level of 0.44 g/L and a CSF/serum glucose ratio of 0.55, which is within normal limits (Table 2). While other laboratory investigations, including cultures, were unremarkable (Table 3), the only positive result was from the multiplex polymerase chain reaction (PCR) assay of the CSF, which detected *E. coli* K1 (Table 4). On day 2 of the ICU stay, the patient remained ventilated with Airway Pressure Release Ventilation (APRV) mode with FiO2 of 0.8. The ABG results showed improvement. The renal profile (RP) also improved following fluid resuscitation and the administration of SLEED (Sodium Lactate Electrolyte and Energy Delivery). Despite intensive management in the ICU, the patient's condition deteriorated. Unfortunately, he succumbed to his illness on day 7 of admission.

Table 4. CSF multiplex PCR

CSF Multiplex PCR	Result
Enterovirus	Not Detected
Herpes Simplex Virus 1	Not Detected
Herpes Simplex Virus 2	Not Detected
Human Parechovirus	Not Detected
Human Herpes Virus 6	Not Detected
Varicella Zoster Virus	Not Detected
Streptococcus pneumoniae	Not Detected
Neisseria meningitides	Not Detected
Streptococcus agalactiae	Not Detected
Listeria monocytogenes	Not Detected
Haemophilus influenzae	Not Detected
***Escherichia coli* K1**	**Detected**
Streptococcus pyogenes	Not Detected
Mycoplasma pneumoniae	Not Detected
Cryptococcus neoformans/gattii	Not Detected

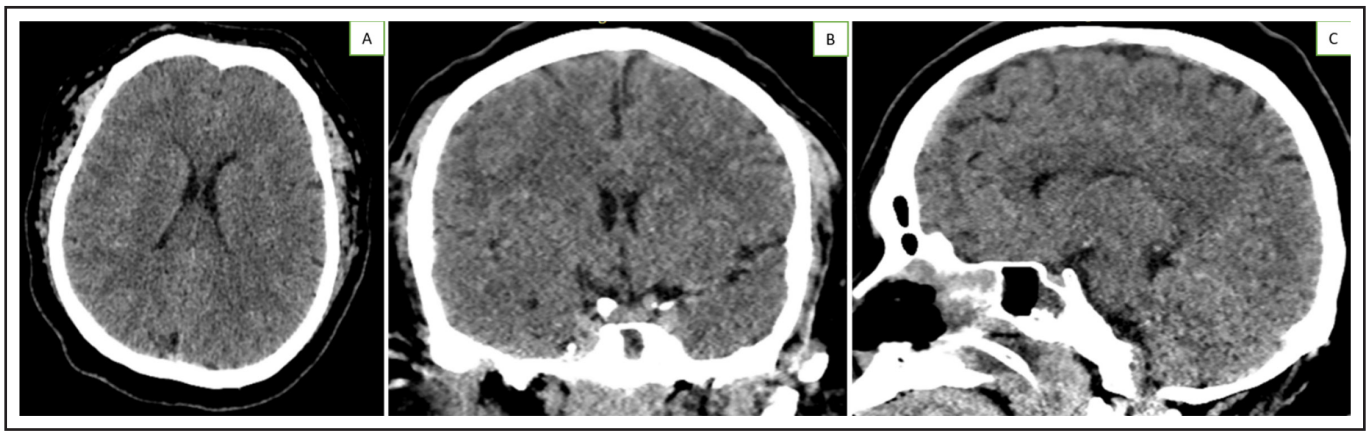

Figure 1. Non-enhanced CT scan of the brain demonstrated mild cerebral edema with no hydrocephalus. [axial (A), coronal (B), and sagittal views (C)].

DISCUSSION

The occurrence of community-acquired *E. coli* meningitis in immunocompetent adults is exceedingly rare, with a documented prevalence of approximately two cases per 100,000 adults. Since 1945, only one case has been reported annually on a global scale. Most cases of *E. coli* meningitis in adults are associated with healthcare settings, particularly following neurosurgical procedures or head injuries. However, spontaneous community-acquired cases can arise in individuals with significant comorbidities, including chronic alcoholism, cirrhosis, human immunodeficiency virus (HIV) infection, chronic obstructive pulmonary disease, the use of immunosuppressive medications, and diabetes mellitus (Kasimahanti *et al.*, 2018). *E. coli* K1 infections in adults are uncommon and typically occur when sterile organs are exposed to the gastrointestinal tract due to trauma or surgical procedures. The initial stages often involve colonization of the gastrointestinal tract, which can lead to bacterial translocation from the small intestine and colon into the bloodstream. The majority of *E. coli* K1 meningitis cases arise from this hematogenous dissemination (Antoine *et al*., 2023).

E. coli is classified into numerous serotypes based on three antigens: somatic (O), capsular (K), and flagellar (H) antigens (Basavaraju & Gunashree, 2022). Specific serotypes, particularly *E. coli* K1, are predominantly associated with neonatal meningitis, accounting for approximately 80% of cases. *E. coli* K1 strains, characterized by their K1 capsular polysaccharide mimic host antigens, serving as a crucial virulence factor. This mimicry allows *E. coli* K1 to evade the immune response, exhibiting antiphagocytic capabilities that facilitate survival in the bloodstream and lead to significant bacteremia (Kim, 2016.; Mir *et al.*, 2023). Once *E. coli* K1 enters the bloodstream, the distinctive virulence mechanisms associated with its K1 capsule enable it to evade lysosomal fusion, allowing the bacteria to traverse and penetrate the blood-brain barrier (BBB) while remaining viable. This capability to cross the BBB as live organisms is a crucial determinant in the pathogenesis of meningitis. (Kim, 2016).

Among the factors associated with *E. coli* meningitis is *Strongyloides stercoralis* infection, particularly in cases of hyperinfection, where larvae migrate from the gastrointestinal tract and may carry *E. coli* into the central nervous system (Gomez *et al.*, 2013). However, the absence of gastrointestinal symptoms and the lack of eosinophilia, which are common features of strongyloidiasis, suggest that this may not be the underlying cause in this patient. He likely developed *E. coli* K1 meningitis through hematogenous dissemination from an undiagnosed bacteremia, potentially secondary to pneumonia. Another possibility for this patient developing *E. coli* meningitis could be gut-derived translocation. Research indicates that individuals with metabolic syndrome frequently exhibit an altered gut microbiome, resulting in a disruption of the equilibrium of beneficial bacteria, which contributes to small bowel pathogenic bacterial overgrowth and increases the permeability of the bowel mucosa leading to subsequent invasion of the bloodstream (Festi *et al.*, 2014; Scheithauer *et al.*, 2020).

In this case, the patient also has underlying OSA and asthma, which notably increased the risk of developing community-acquired pneumonia (CAP). Patients with OSA are at increased risk for pulmonary aspiration during sleep due to intermittent upper airway obstruction. This can lead to the introduction of oropharyngeal or gastrointestinal flora, including *E. coli*, into the lower respiratory tract, resulting in pneumonia (Chiner *et al*., 2016). Although *E. coli* is predominantly linked to gastrointestinal infections, its occurrence in the respiratory tract is infrequent yet well-documented in the medical literature, especially among patients with obstructive sleep apnea, pre-existing lung conditions, or those experiencing aspiration events (Ganipisetti *et al*., 2023). It is plausible that these factors could explain why the patient manifested with both *E. coli* meningitis and CAP.

Mild to moderate hyponatremia (serum sodium levels between 125–135 mmol/L) is commonly observed in cases of bacterial meningitis but may not adequately explain the severity of neurological symptoms (Brouwer *et al*., 2007). Additionally, severe hypercapnia ($PaCO_2$, > 80 mmHg) is likely to contribute to significant neurological impairment (Drechsler & Morris, 2025). Despite interventions being given to correct sodium levels and CO_2, retention, it did not result in significant neurological improvement in this patient. This indicates that while hypercapnia and hyponatremia were contributing factors, they were not the primary causes of the patient's neurological decline. The altered mental status and seizure in this case were likely due to acute bacterial meningitis.

A normal or slightly elevated WBC count does not exclude the diagnosis of bacterial meningitis. Studies have shown that peripheral WBC counts lack sensitivity and specificity in diagnosing bacterial meningitis. In the setting of acute bacterial meningitis, monocytosis is less commonly emphasized compared to neutrophilia. However, it may reflect the body's ongoing immune response to infection (Khalili *et al*., 2015).

Procalcitonin (PCT) testing was not performed in this case. While PCT is a more specific biomarker for bacterial infections, an elevated CRP level can still aid in the diagnosis of bacterial meningitis. The patient's CRP level was significantly elevated at 167 mg/L. Research indicates that CRP levels exceeding 50 mg/L are frequently associated with bacterial meningitis (Hansson *et al*., 1993; Thakur *et al*., 2020).

The diagnosis of bacterial meningitis typically involves analyzing CSF biochemistry. The patient's CSF protein level of 0.44 g/L at the upper limit of the normal range (0.15–0.44 g/L), and a CSF glucose level of 5.5 mmol/L compared to a serum glucose level of 10 mmol/L, resulting in a CSF/serum glucose ratio of 0.55, which is also within normal limits. Typical bacterial meningitis CSF findings are elevated protein and low glucose levels; however, variations can occur due to the early stage of infection or individual immune responses. In the initial phase, biochemical changes may be minimal, as inflammatory processes and blood-brain barrier disruption may not yet be fully established. Additionally, the patient received antibiotics prior to the lumbar puncture, which may attenuate the inflammatory response and lead to less pronounced CSF biochemistry abnormalities (Nigrovic *et al.*, 2008).

CSF culture is the gold standard for confirming the diagnosis, however, in this patient, his CSF culture turned out to be negative. The CSF latex agglutination test was not performed in this case. We utilized the multiplex PCR method to detect *E. coli* K1 using the QIAstat-Dx® Meningitis/Encephalitis (ME) Panel by QIAGEN, Germany, which has a sensitivity of 66.7% and a specificity of 99.9% for *E. coli* K1 detection (QIAGEN, 2024). Molecular techniques, such as multiplex PCR assays, have demonstrated higher sensitivity compared to traditional cultures, especially in patients who received prior antimicrobial therapy. A study showed the sensitivity of PCR for diagnosing bacterial meningitis was superior to culture, with rates of 59% versus 43%, respectively ($p < 0.05$). PCR demonstrated high specificity at 97%, comparable to culture. When both culture and PCR were utilized, the combined sensitivity increased to 69%. Notably, among patients receiving antibiotic treatment, the sensitivity difference between PCR (79%) and culture (45%) was statistically significant ($p < 0.005$) (Welinder-Olsson *et al*., 2007). A separate study indicated that the positivity rate of PCR testing in CSF specimens surpassed that of CSF bacterial cultures, with rates of 39.5% for CSF PCR compared to 7.4% for CSF culture ($p < 0.05$) (Li *et al*., 2024).

The administration of antibiotics before CSF collection can lead to negative culture results while still allowing PCR to detect bacterial genetic material. This is especially relevant in cases of bacterial meningitis, where timely treatment is critical. Additionally, early in the infection, bacterial loads may be low, resulting in negative cultures while PCR may still yield positive results. All these factors may explain why this patient has a positive PCR result and a negative CSF culture.

It is important to note that the ability of PCR to detect non-viable bacteria can lead to false-positive results, necessitating careful interpretation in the context of the patient's clinical presentation and history (Li *et al*., 2024). Multiplex PCR can identify a range of pathogens, and certain bacteria may persist in the cerebrospinal fluid (CSF) after an infection has resolved, resulting in positive PCR results without active disease. Nevertheless, PCR results demonstrating the presence of highly virulent organisms such as *E. coli* K1, a well-established pathogen of acute bacterial meningitis characterized by rapid progression with high mortality, warrant careful consideration. Furthermore, in this case, the commencement of antibiotic therapy prior to culture collection may have contributed to the negative culture result, thereby reinforcing the reliability of the positive PCR finding. Considering the acute nature of *E. coli* K1 meningitis and the patient's condition clinically, the detection of the pathogen using PCR in this case is unlikely to be a false positive.

Unfortunately, cultures did not isolate the pathogen, which resulted in the inability to perform antimicrobial susceptibility testing (AST). However, based on the literature, *E. coli* K1-positive strains are less frequently associated with resistance modifications across multiple antimicrobial classes. In contrast, K1-negative strains exhibit chromosomal resistance mechanisms, including inhibitor-resistance TEM (IRT) and mutations in gyrA/B or frxA/rdxA, which confer resistance to quinolones and trimethoprim-sulfamethoxazole. Another study reported higher susceptibility to ampicillin in K1-positive strains. Furthermore, Cole *et al*. demonstrated that antibiotic non-susceptibility inversely correlates with the total number of virulence factors (Fujita *et al*., 1990, Cole *et al.*, 2019; Proquot *et al*., 2021).

Empirical treatment for suspected *E. coli* meningitis should not be delayed, even if lumbar puncture is postponed due to imaging studies. The preferred regimen includes ceftriaxone or cefotaxime, with alternative options for immunocompromised patients being chloramphenicol or meropenem. If no organism is isolated but the clinical picture suggests bacterial meningitis, it is recommended to continue antibiotics for a minimum of 14 days (Ministry of Health Malaysia, 2024).

CONCLUSION

This case report illustrates the rare and fatal occurrence of community-acquired *E. coli* K1 meningitis in an adult with significant comorbidities. The rapid progression of symptoms and positive PCR results highlight the importance of considering atypical pathogens in adult meningitis diagnoses. This case underscores the critical role of molecular diagnostics in confirming pathogens when cultures are negative, facilitating timely and appropriate treatment.

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Conflict of Interest Statement

The authors declare that they have no conflicts of interest.

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