HK J Paediatr (new series) 2026;**31**:44-47

Case Report

Enterobacter cloacae Septicaemia Complicated by Mild Encephalitis with a Reversible Splenial Lesion of the Corpus Callosum (MERS)

K ***Jagadish Kumar***, VG ***Manjunath***, HR ***Nandish***, A ***Ratna***

Abstract ***Background:*** *Enterobacter cloacae (E. cloacae)* has been recognised as one of the important nosocomial pathogens in recent times and causes a broad spectrum of infections involving the lower respiratory tract, urinary tract, central nervous and musculoskeletal system. *E. cloacae* causing systemic bacterial infection is uncommon in children and primarily affects younger children or those with associated underlying medical problems. ***Case Presentation:*** We are reporting a 5-year-old immune-competent boy with community-acquired *E. cloacae* septicaemia, who presented with septic shock requiring vasopressor therapy, and mild encephalitis with a reversible splenial lesion of the corpus callosum. He recovered completely without any sequelae after treatment. ***Conclusions:*** Even though an *E. cloacae* bloodstream infection is typically a nosocomial pathogen that affects those with co-morbidities; it can rarely lead to severe and life-threatening community-acquired septicaemia.

Key words Community-acquired; *Enterobacter cloacae (E. cloacae)*; Mild encephalitis with a reversible splenial lesion of the corpus callosum (MERS); Septicaemia

Introduction

Enterobacter cloacae (E. cloacae) is a gram-negative bacterium that belongs to the genus Enterobacter.[1,2] *E. cloacae* is one of the commensal flora of the human intestinal tract and is also widely distributed in environmental sources such as water, soil, and food.[1,3] Nowadays, *E. cloacae* has been recognised as an important nosocomial pathogen and causes a broad spectrum of infections involving the lower respiratory tract, urinary tract, central nervous system, joints, bones, skin and soft tissues.[1,3] *E. cloacae* causing systemic bacterial infection is rare in children and usually affects younger children or those with underlying medical problems.[4] Even though most of the cases are due to nosocomial infections, community-acquired infections can also occur and can be equally fatal.[2] We are reporting a 5-year-old healthy male child with community-acquired *E. cloacae* septicaemia who presented with septic shock, and mild encephalitis with a reversible splenial lesion of the corpus callosum (MERS). He recovered completely without any sequelae following prompt treatment.

Department of Pediatrics, JSS Medical College, JSS Academy of Higher Education and Esearch, Mysore, India

K **Jagadish Kumar** *MBBS, MD*
VG **Manjunath** *MBBS, DCH, DNB*
HR **Nandish** *MBBS, MD*
A **Ratna** *MBBS*

Correspondence to: Dr K **Jagadish Kumar**
Email: jagdishmandya@gmail.com

Received July 8, 2024

Case Report

A 5-year-old developmentally normal boy presented with fever, loose stools, vomiting for 1 day, and convulsions over 6 hours. Loose stools were watery, and profuse with no blood in them. He had 2 episodes of tonic-

clonic seizures for 20 minutes each with loss of consciousness. There was no history of trauma/ear discharge/headache/ body ache. On examination, he was dehydrated with a Glasgow coma scale (GCS) of 7/15. He had a temperature of 99.6°F, Pulse Rate (PR) of 148/ minute, low volume and feeble with cold extremities, Respiratory Rate (RR) of 40/minute, only manually recordable systolic Blood Pressure (BP) of 74 mm of Hg, and Capillary Filling Time (CFT) of 6 seconds. Per abdomen examination revealed 4 cm hepatomegaly. Respiratory (RS) and Cardio Vascular System (CVS) examinations were unremarkable. Immediately oxygen, IV (Intra venous) Levetiracetam, and two fluid boluses (20 ml/kg of normal saline) were initiated along with correction of dehydration. However, despite fluids infusion, he continued to have feeble peripheral pulses, cold extremities and CFT of 3 seconds, and hence a preliminary diagnosis of shigella gastroenteritis, with encephalopathy and septic shock was made. Inj Noradrenaline at a dose of 0.1 mg/kg/minute was started along with Inj Ceftriaxone and Inj Meropenam. Investigations are depicted in Table 1 and Figure 1. MRI brain revealed hyperintense signal changes of the splenium of the corpus callosum (SCC) visible on diffusion-weighted imaging (DWI) sequences. Twelve hours after admission, the patient continued to have fever spikes. His GCS improved to 15/15 and urine output to 2 mL/kg/hour. Vital signs were as follows: PR 128/min with good volume and warm peripheries, RR 32/min, BP of 80/42 mm Hg, and a CFT of 2 seconds. At 24 hours post-admission, the child was conscious with a GCS of 15/15 with normal urine output. His vitals are as follows: Temperature 100°F, RR 30/minute, PR 120/minute, good volume with warm peripheries, BP of 98/72 mm of Hg, and CFT 2 seconds. Inj noradrenaline infusion was slowly tapered and stopped while antibiotics continued for ten days. He gradually improved and was discharged on the 10th day of hospitalisation.

Discussion

The clinical significance of *E. cloacae* is that it is frequently multidrug-resistant and associated with a mortality risk of up to 40%, thus making it a huge challenge for the treating clinician.[2,5] Factors associated with mortality due to invasive *E. cloacae* include low haemoglobin levels, increased C-reactive protein (CRP), septic shock, and renal failure.[2] Our case hailing from a rural area of south India presented with acute gastroenteritis, shock and status epilepticus. There was no history of previous hospitalisation, underlying medical conditions, or immunodeficiency. The presenting symptoms at admission made us think of a provisional diagnosis of shigella diarrhoea with encephalopathy. He had leucopenia, thrombocytopenia, elevated CRP, and pneumonia on chest X-ray. Blood culture grew *Enterobacter cloacae* sensitive to most of the drugs. His stool culture was sterile. Even though he presented with septic shock with MERS, he responded well within 2 days of starting inj meropenem and inj ceftriaxone as *E. cloacae* is sensitive to both antibiotics.

The classic triad of muscle pain, weakness, and dark urine for the diagnosis of rhabdomyolysis was not present in our child. Serum CK levels in rhabdomyolysis are usually at least five times the upper limit of normal but range from approximately 1500 to 100,000 units/L.[6] Serum CK levels were 2434 U/L at presentation and decreased to 81 U/L after recovery in this child. Falasca et al reported myositis and rhabdomyolysis in association with *E. cloacae* infection.[7]

MERS is characterised by a transient mild encephalopathy and a reversible lesion in the splenium of the corpus callosum on MRI.[8] In MERS, hyper-intense signal changes in the splenium of the corpus callosum are visible on DWI in the brain MRI is associated with various other neurological symptoms like disturbed consciousness, behavioural disorders, incoherent speech, seizures, drowsiness, headaches, and hallucinations. The present case had gastroenteritis, altered consciousness, seizures and MRI features of MERS (Figure 1). There is a well-known association between MERS and hyponatremia, which was also seen in our patient, who presented with a serum sodium level of 129 mEq/L. A variety of other infectious agents have also been known for the development of MERS, including Rotavirus, Cytomegalovirus, Adenovirus, Influenza virus, Epstein-Barr virus, Mycoplasma pneumoniae, Salmonella, Escherichia coli and Shigella.[8] For a decade, *E. cloacae* has emerged as one of the most common nosocomial pathogens causing outbreaks in neonatal units.[9]

Bonadio et al reported 30 children with *E. cloacae* bacteraemia over 12 years. Among the 30 cases, 14 were infants less than one year of age, out of which the majority were younger than two months. In 17 children, bacteraemia was nosocomial acquired in the ICU. Four

Table 1 Investigations

Investigations	At admission	Day 3	Day 7
Hb (g/L)	91	98	
PCV proportion of 1.0	0.28	0.29	
Total Counts [cells x10^9/L]			
DLC	2.580 [N61.3%, L37.6%, E0.2%, M0.8%, B0.1%]	7.220 [N76.6%, L19%, E2.6%, M1.5%, B0.2]	
Platelets [cells x 10^9/L]	128	161	
CRP [U/L]	168		5.56
ESR [mm/hr]	10	50	
Blood Urea [mmol/L]	12.49	3.93	
Serum Creatinine [mmol/L]	46.85	38.01	
Serum Sodium [mmol/L]	129	137	
Serum Potassium [mmol/L]	3.7	3.7	
Blood Sugar [mmol/L]	8.1	5.94	
Serum Bilirubin [µmol/L]	4.96		
AST [U/L]	33		
ALT [U/L]	9		
Total protein [gm//L]	41		
Serum albumin [gm//L]	26		
Serum Mg [mg/dl] mmol/L	0.74		
Serum Phosphorous [mmol/L]	1.36		
Serum Calcium [mmol/L] [Ionised]	1.77 [0.27]		
CK-NAC [U/L]	2434		81
CKMB [N=1-4.9 U/L]	28.02		
Troponin-T [N=0.0127-0.024 ng/L]	0.007		
ABG=pH=7.34, pO_2=196 , pCO_2=31.7, HCO_3=18.2, Base excess=7.7 , Lactate=-2.3			
CSF analysis=Cell count=1 cell, lymphocyte. Protein=23.7 mg/dl , sugar=30.9 mg/dl , Cl=126.8, CSF Culture=Sterile			
Blood Culture grew *Enterobacter claocae*			
Sensitive to ceftriaxone, amikacin, cefipime, Cotrimaxazole, meropenam, piperacillin+tazobactum, Resistant to ciprofloxacin, amoxicillin-clavulanicacid, cefuroxime			
Stool microscopy=Plenty of inflammatory cells, no RBCs, no parasitic forms seen			
Stool hanging drop–negative, Stool culture sterile			
RT-PCR for COVID 19, Influenza A, B, H1N1=Negative, Dengue NS1=negative, HIV antibodies negative			
Echocardiography=Normal, Chest X-ray right paracardiac pneumonia			
Urine culture sterile			
MRI Brain=Hyperintense signal changes of the splenium of the corpus callosum (SCC) visible on diffusion weighted imaging (DWI) sequences.			
EEG=Normal			

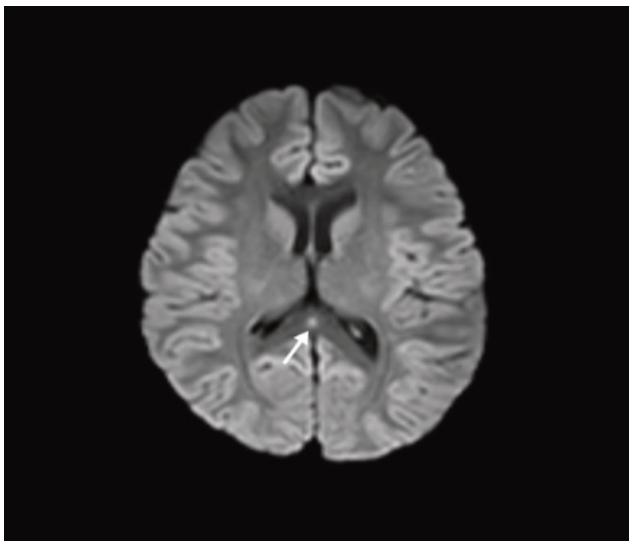

Figure 1 MRI Brain: Hyperintense signal changes of the splenium of the corpus callosum (SCC) visible on diffusion-weighted imaging (DWI) sequences.

children had hypotension requiring vasopressors and 3 patients died. The most common underlying condition was immune deficiency followed by gastrointestinal tract lesions.[4] In another study, all children with *E. cloacae* bacteraemia had at least one underlying condition, the most common being neuromuscular (35%), end-stage renal disease (27%), and short bowel syndrome (15%). In 75% of cases, a central venous catheter was present and hypotension was noted in 38% of cases. One-third of them had severe sepsis and mortality was 8%.[5] This child also presented with septic shock, requiring noradrenaline infusion for hemodynamic support. Öz et al reported a child who was treated with ACTH for infantile spasm developing rotavirus gastroenteritis complicated by nosocomial *E. cloacae* bacteraemia.[10] Wutor et al reported the unsuccessful treatment of sepsis due to multidrug-resistant *E. cloacae* sepsis in a 25-month-old child.[2]

E. cloacae have the ability to secrete various cytotoxins (enterotoxins, haemolysins, pore-forming toxins) and to form biofilms, which are important in the pathogenicity of *E. cloacae* infections.[1]

Conclusions

Although *E. cloacae* bloodstream infections are classically associated with nosocomial settings and primarily affect individuals with underlying comorbidities, they can rarely, cause community-acquired septicaemia, which may manifest as a severe and potentially life-threatening condition.

Conflicts of Interest

None

References

1. Davin-Regli A, Pagès JM. Enterobacter aerogenes and *Enterobacter cloacae*; versatile bacterial pathogens confronting antibiotic treatment. Front Microbiol 2015;6:392.
2. Wutor BM, Adefila WO, Lamin KM, et al. Community-acquired multidrug-resistant *Enterobacter cloacae* sepsis in a 25-month-old child in rural Gambia: A case report. IDCases 2024;37:e02018.
3. Huang J, Xu Q, Liu F, Xiong H, Yang J. *Enterobacter cloacae* infection of the shoulder in a 52-year-old woman without apparent predisposing risk factor: a case report and literature review. BMC Infect Dis 2021;21:13.
4. Bonadio WA, Margolis D, Tovar M. *Enterobacter cloacae* bacteremia in children: a review of 30 cases in 12 years. Clin Pediatr (Phila) 1991;30:310-3.
5. Alhajri N, Hamdy R. 2429. The Epidemiology and Outcomes of *Enterobacter cloacae* Bloodstream Infections in Children. Open Forum Infect Dis 2018;26;(5 Suppl 1): S726.
6. Bhai S, Dimachkie MM. Rhabdomyolysis: Clinical manifestations and diagnosis. In Targoff I R, Shefner J M.(Ed) UpToDate. Retrieved June 1, 2024, from https://www.uptodate.com/contents/Rhabdomyolysis: Clinical manifestations and diagnosis.
7. Falasca GF, Reginato AJ. The spectrum of myositis and rhabdomyolysis associated with bacterial infection. J Rheumatol 1994;10:1932-7.
8. Le Soudéer L, Truong J, Le Gal J, Escoda S. Shigella-associated mild encephalitis with a reversible splenial lesion in Hospital Center Delafontaine, Saint-Denis, France: a case report. BMC Pediatr 2022;22:421.
9. Ferry A, Plaisant F, Ginevra C, et al. *Enterobacter cloacae* colonization and infection in a neonatal intensive care unit: retrospective investigation of preventive measures implemented after a multiclonal outbreak. BMC Infect Dis 2020;20:682.
10. Öz FN, Koca SB, Tanır G, Çiçek D, Acar M, Zorlu P. *Enterobacter cloacae* septicaemia complicating rotavirus gastroenteritis: a case report. East Mediterr Health J 2014;20:514-6.