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Case Report Open Access

A Case Report of Brain Abscesses Caused by *Enterobacter cloacae*

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Abstract

In immune-competent youngsters, brain abscesses caused by Enterobacteriaceae are uncommon, and those caused by Enterobacter cloacae are even more so. We describe an intriguing case of numerous brain abscesses caused by a community-acquired E. cloacae neuroinfection in a young boy who had no predisposing risk factors. A 10-year-old child arrived at the hospital with a low-grade fever, a headache, neck pain, and gradual sensorium degeneration. He had photophobia, normal fundi, meningeal symptoms, moderate hypertonia, quick muscular stretch reflexes, and extensor plantar responses when examined. He was aware but sleepy. Brain MRI results revealed bilateral, numerous pyogenic abscesses. E. cloacae was growing in the abscess material that was aspirated during surgical drainage. During an 18-week period, he received intravenous imipenem under clinical and radiological monitoring. a methodical strategy combining early. In such challenging instances, a practical strategy combining early surgical drainage, focused antimicrobial treatment, and patient-tailored duration depending on the clinico-radiological response is required.

Keywords: Neuroinfection; Brain abscess; Enterobacter; Intravenous; Antimicrobial

Introduction

It is exceedingly rare for children to suffer brain abscesses brought on by Enterobacteriaceae or Enterobacter cloacae. In intensive care units (ICUs), the gram-negative bacillus E. cloacae is frequently seen. Transmission is almost entirely nosocomial in the absence of predisposing conditions like haematological malignancies, immunosuppressive drugs, post-transplant or burn complications, cerebrospinal fluid (CSF) shunt complications, prolonged hospitalization, or invasive procedures [1,2]. We address a youngster with immune-competent multiple brain abscesses caused by community-acquired E. cloacae and no known risk factors. The clinical issues that arise when handling these situations are highlighted.

Case presentation

An eight-day bout of widespread, dull-aching headache and intermittent low-grade fever in a 10-year-old child was followed by neck discomfort, increasing sensorium impairment, and irrelevant speech. There was no history of injury, ear discharge, respiratory issues, interaction with a patient who had TB, a persistent condition that was incapacitating, or many hospitalizations. History of the family was uneventful. He was awake but sleepy at examination, with symmetrical and evenly responding pupils, photophobia, normal fundi, meningeal symptoms, slight hypertonia, rapid muscular stretch reflexes, and extensor plantar responses. Examination of the sensory, cerebellar, extrapyramidal, and rest of the cranial nerves revealed no abnormalities. The system analysis was not helpful. It was thought that sub acute meningoencephalitis may be seen clinically. Haemoglobin levels were 107 g/L, total leukocyte count 22,400 cells/L (neutrophils 82%, lymphocytes 11%, monocytes 6%, and eosinophils 1%), platelet count 405,000 cells/L, activated partial thromboplastin time (aPTT) 25, prothrombin index 61%, international normalised ratio 1.4, and fibrinogen and D-dimer levels were all within normal ranges. The results of the pee microscopic examination, renal and liver function tests, and serum electrolytes were all normal. Repeated blood cultures and CSF samples were sterile. Multiple brain abscesses were discovered during contrast-enhanced magnetic resonance imaging (CE-MRI) of the brain. Ceftriaxone (100 mg/kg/day in two split doses), cloxacillin (200 mg/kg/day in four divided doses), and metronidazole (45 mg/kg/day in three divided doses) were experimentally begun in him. In the serum, there were no antibodies against toxoplasma, hydatid, amoeba, or human immunodeficiency virus. No occult source of infection was found by echocardiography, paranasal sinus radiography, abdominal ultrasonography, otologic, or dental tests. The results of the nitrosozo blue tetrazolium and dihydrorhodamine tests, the IgG-subclass, serum C3 and C4 levels, CH50 activity, and the immunoglobulin profile were all normal. After drainage and excision of the abscess, a left frontal microcraniotomy revealed 8 mL of frank pus. Gram-staining revealed gram-negative bacteria, and E. cloacae (sensitive to pipercillin, amikacin, and imipenem; resistant to cefotaxime) was growing in culture. A pyogenic abscess was suspected after a histopathological examination of the abscess-wall revealed neutrophils, fibrin deposits, proliferating blood vessels, and reactive gliosis in the nearby brain tissue. Ipenem was then injected into the infant (60 mg/kg/day in 3 separate dosages, 8 hours). He recovered gradually. Repeat CE-MRI imaging after eight weeks of intravenous antibiotic therapy revealed smaller abscesses, but the perilesional edoema persisted. So, the course of antibiotics was prolonged for an additional 10 weeks. After receiving antibiotics for a total of 18 weeks, CE-MRI results showed a considerable shrinkage of the brain abscess and partial healing of the superficial lesions. The youngster is presently asymptomatic and neurologically healthy after 18 months of follow-up.

Discussion

As numerous brain abscesses caused by community-acquired E. cloacae neuroinfection without any predisposing conditions are highly unusual, this case deserves consideration. Although a term infant's case of fast progressing, community-acquired E. cloacae meningo-

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encephalitis has been documented as a case study [3], the majority of cases that occur outside of the neonatal period are nearly usually linked to an underlying predisposing disease [4]. Due to his immunological competence and lack of earlier E. cloacae colonization, our patient was exceptional. There was no prior hospitalization history. We consider our case to be due to an unrecognized bacteremia from the throat, intestine, or urinary tract disseminating to the brain because E. cloacae infection is typically nosocomial or dissemination from an endogenous reservoir such as the gastrointestinal tract in healthy adults, the urinary and respiratory tracts, as well as surgical sites and burn wounds. Due to its highly impermeable blood-brain barrier, the non-predisposed brain is often extraordinarily resistant to bacterial and fungal infections, even occult bacteremia. Even in experimental models of brain abscess, the production of an abscess frequently requires direct injection of organisms into the animal's brain [5]. Thus, the reason why our patient developed several brain abscesses with only a brief history of symptoms is noteworthy. A peripherally-enhancing T2-isointense wall made of a well-defined rim of collagen and inflammatory cells, which is thinner than in tuberculous abscesses, and a central area of liquefaction showing T1-hyppointensity, T2-hyperintensity, and diffusion restriction are distinctive MRI features that aid in the identification of a "pyogenic abscess." Vasogenic edoema that appears as finger-like hyper intensity on T2 and FLAIR images is typically present around the lesion [6]. The host brain reacts by trying to separate from the inflamed tissue, which first manifests as cerebritis, and a mature abscess develops as a result. Nevertheless, it has to be distinguished from other causes including hematoma, metastasis, and granuloma, which have a suitable clinical history and particular imaging appearances. We can identify a specific aetiology for a lesion based on its characteristics, such as the thickness, irregularity, and nodularity of the rim, the kind of central necrosis, and the presence of certain materials in the wall [6]. It might be challenging to determine the cause of a brain abscess. The effectiveness of CSF and blood cultures is limited, and performing a lumbar puncture might be risky. The greatest chance to determine the aetiology of the abscess and start targeted antibiotic therapy is to carefully culture the abscess material aspirated during surgical surgery.

Uncertainty exists regarding the ideal length of antibiotic treatment for paediatric brain abscesses. If the bacterium and its sensitivity are known, conventional treatments of parenteral antibiotics for 6–8 weeks may be sufficient. According to parameters such the etiological agents and their antibiotic susceptibility, the size and number of abscesses, the effectiveness of surgical drainage, and the patient's clinical and radiological response, a pragmatic approach would be to determine the length on a case-by-case basis [7]. Due to many abscesses, an innately resistant organism, insufficient surgical drainage, and sluggish resolution on serial neuroimaging, we maintained intravenous antibiotics for 18 weeks.

Conclusion

Children may acquire E. cloacae in the community, and underlying risk factors must be ruled out. It is necessary to use a practical strategy that combines early surgical drainage, focused antimicrobial treatment, and patient-tailored duration depending on the clinico-radiological response.

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Not applicable.

Conflict of Interest

Author declares no conflict of interest.

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