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Case Report

CEREBRAL ABSCESS WITH HYDROCEPHALUS MANIFESTATION: A CASE REPORT IN 10-MONTH-OLD INFANT

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ABSTRACT

Background Alongside hydrocephalus, cerebral abscess poses as a space-occupying lesion altering consciousness and intracranial pressure. Cerebral abscesses commonly presented vague clinical symptoms in infants, including weeks of fever and late-onset neurologic deterioration. However, limited studies denote cerebral abscesses, especially in children under one year old.

Objective We reported a case of a 10-month-old male infant with cerebral abscess and hydrocephalus.

Case description A 10-month-old male infant was taken to the hospital due to 6 days progressing fever. Neither clinical nor laboratory findings were abnormal. After being discharged from the hospital, the patient was admitted again 2 days later, reserved for 5 days of fever until unfortunate symptoms of raised intracranial pressure coincide with hypothermic body temperature. A brain CT-Scan showed an area of hypodense lesion in the parieto-occipital region suspected of brain abscess- resulting in non-communicating hydrocephalus. After a total of 12 days of intensive treatment with antibiotics, anticonvulsant, and steroid agents, the patient was comatose and passed away due to respiratory failure.

Conclusion Brain abscesses should be investigated thoroughly despite the probable absence of a pathognomonic feature. Radiology imaging ought to demonstrate the early process of abscess pathogenesis. To the best of our knowledge, this is the first case report published in Indonesia of an infant with a cerebral abscess resulting in hydrocephalus.

Keywords: cerebral abscess; hydrocephalus; infant

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INTRODUCTION

Cerebral abscess, an infection within the brain's parenchymal, is quite a challenge to pronounce, especially for physicians. Determination of abscess etiology and its pathogenesis of acute cerebritis process to encapsulate purulent material could include unspecific symptoms and may differ in various age groups.[1] In pediatrics, a triad of fever-headache-neurologic dysfunction is present in under 14% of children with focal intracranial suppurative infection.[2] Clinical findings may be unspecific, mild, or severe, and may be influenced by the patient's age, the stage, size, and location of the abscess, the presence of meningitis, and the patient's immune status.[3] Neuroimaging techniques often signify its mass effect and complication in the clinical suspicion of brain abscess.

Commonly, bacterial meningitis or bacteremia is the highest predisposing factor of brain abscess in infants owing to higher susceptibility. The most frequent pathogen is Staphylococci and Streptococci anaerobes and Gram-negative bacteria consecutively.[4,5] Fortunately, significant decline in the mortality rate of brain abscess from 30% to 60% in the 1970s and 1980s to 4–24% in recent years has been attributed to the improvement of hygiene and vaccination in the pediatric population, advances in diagnostic imaging leading to early diagnosis, improved and rapid neurosurgical intervention techniques, and broad-spectrum antibiotics

covering both aerobic and anaerobic organisms.[3]

Nearly half of children with brain abscesses had subclinical sequelae.[1] Though scarce, hydrocephalus formation as brain abscess sequelae is possible.[2,6] further increasing intracranial pressure and giving a poorer prognosis hence the need for studies analyzing this phenomenon and its complicating factor.

Similar studies have been done in Lagos previously[3] and years ago[7] but no emphasis on brain abscesses prior to hydrocephalus. Moreover, studies about cerebral abscesses and hydrocephalus events in Indonesia are still scarce. In this case report, we reported a case of a 10-month-old male infant diagnosed with a cerebral abscess presenting ipsilateral hydrocephalus.

CASE DESCRIPTION

A 10-month-old male infant was taken to Bethesda Hospital Yogyakarta due to a history of 6 days persistent fever, lethargy, and loss of appetite. No previous history of viral or bacterial infection was noted. The temperature on admission was 36.5°C and unremarkable physical or laboratory examination. The patient was later discharged on the fifth day as the condition improved. On the second admission, the fever reached 37°C and 38°C. Still, the physical examination and other vital signs were unremarkable but apathetic consciousness. Neither blood and urine bacterial culture, NS-1 test, nor Mantoux test were positive.

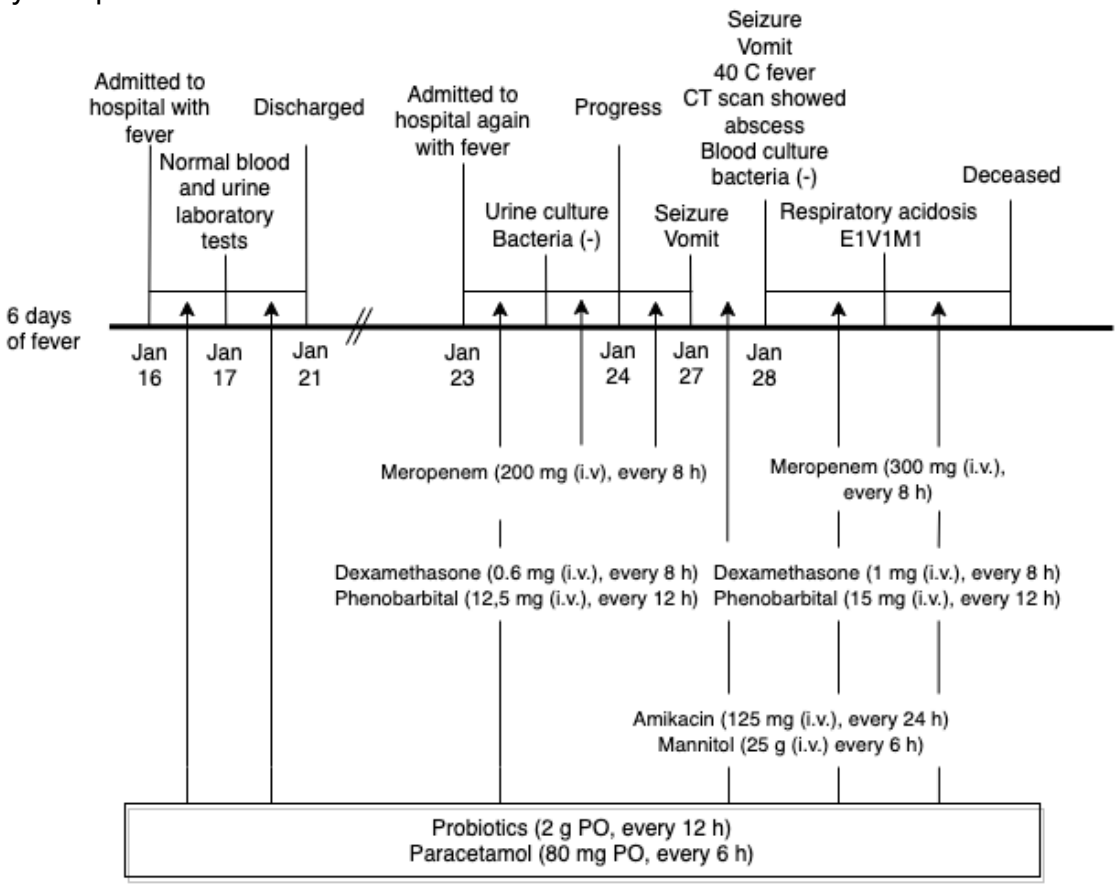


Figure 1. Timeline of the case presentation description

The temperature dropped to normal by 36°C in the next 2 days, and the patient was compos mentis. Projectile vomitus and general seizure were later reported. As the temperature increased, laboratory examination was done on the fifth day showing low hemoglobin (9.7 g/dL), high leukocyte (23.18 109/L), low eosinophil

(0.3%), high segmented neutrophil (64.5%), low lymphocyte (26.6 %), low hematocrit (31.1 %), low MCHC (31.2 g/dL), high thrombocyte (481109/L), high blood glucose (121.0 mg/dL). Oxygen saturation was still 99% until the next dawn when the respiratory rate dropped to 30x/minute and heart rate 89x/minute before 5 minutes of convulsive state. Later, there was dropping in temperature (34.7 C), mydriatic pupil (+)/ (+), tense fontanelle present, and coma state (Glasgow Coma Scale E1V1M1). Endotracheal tube and nasogastric tube were inserted. Later, the night, blood gas analysis showed respiratory acidosis (Table 2). The patient was still comatose, hypothermic (35°C), and was not breathing spontaneously. The patient had a seizure fit once more before being pronounced dead.

The complete timeline of the subject's presentation is mentioned in Figure 1.

Clinical Findings

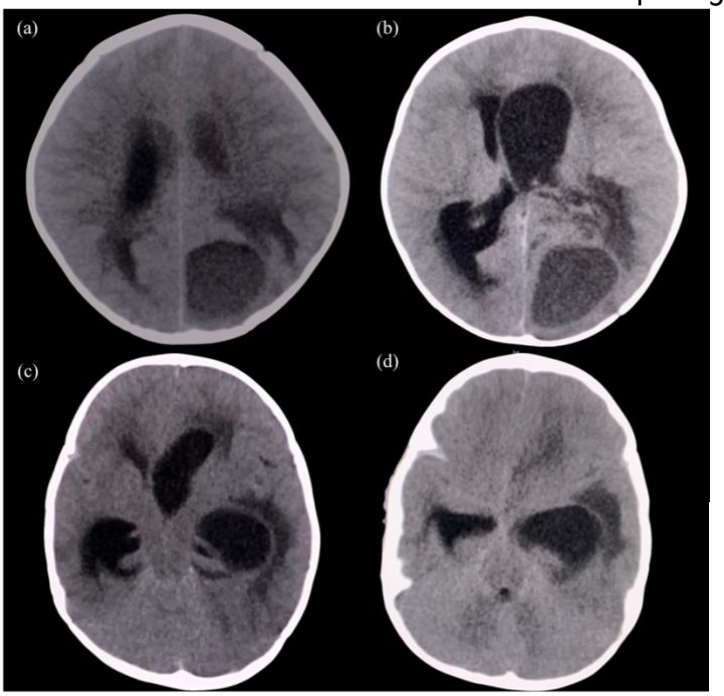
Overall, the total days of admission are roughly 12 days. Signs of intracranial pressure increase were pronounced a few days before the subject's death.

The highest recorded fever is 40°C on the tenth day, followed by a period of hypothermia of 34.7°C and 35°C respectively on the last 2 days before the patient died due to respiratory failure. There were a total of 3 seizure fits, with the seizure type being generalized tonic-clonic. Bulging of the anterior fontanelle has been prominent in the last 2 days.

Diagnostic Assessment

An axial slice 5mm head CT-Scan with no contrast was performed, showing a hypodense-cystic-lesion in the subject's left parieto-occipital region, causing the falx cerebri to deviate to the right. Periventricular edema was noted, especially in Picture 1c. The lateral ventricular system widened, giving the impression of hydrocephalus (Picture 1), in which we suspect the obstruction happened below the lateral level and just before the third ventricle; showing no involvement in the third ventricle (Picture 1b) and the fourth ventricle (Picture 1d). No atypical findings of cisterns and extracranial soft tissue.

Due to no cerebrospinal fluid analysis, we cannot determine the etiology of what pathogens cause the brain abscess.



Picture 1. Pediatric brain CT-Scan series showing large hypodense cystic-lesion in the parieto-occipital region associated to mass effect and dilatation of ventricular system with a deviation of falx cerebri.

Therapeutic Intervention

Antibiotic agents used are Meropenem (Penicillin) and Amikacin (Aminoglycoside). Corticosteroid agent Dexamethasone and anticonvulsant Phenobarbital administration was given on the seventh until the ninth day due to no noticeable progress. Mannitol was injected in the last 2 days due to symptoms of raised intracranial pressure. A detailed timeline of the medication regime is presented in Figure 1.

For maintenance, Ringer Lactate infusion was administered 15 cc/ hour. Endotracheal tube + ventilator mode bilevel PEEP 4 was inserted in the last 2 days and later changed to a bigger endotracheal tube (No. 4) inserted 13 centimeters in.

DISCUSSION

Brain abscess progression yields devastating outcomes. It generally involves 4 stages: early cerebritis, late cerebritis, early capsule formation, and late capsule formation occurring on days 1-4, day 4-10, day 11-14, and day >14, respectively. The subject's clinical history showed an early or late capsule formation phase.[8] However, we could not define whether the lesion is yet capsulated due to no contrast injected, distinguishing a thin-regular-enhanced wall[9]. Edema formation is expected in the surrounding tissue due to the vast inflammation process[8] hence the increasing pressure of intracranial, manifested in projectile vomitus, alteration of consciousness, and seizure.

In the period of inpatient, the subject's pediatrician suspected bacterial meningitis-since Indonesia possessed the eighth highest burden of meningitis disease in children under 5-year-old.[10]

The previous site of Infection could be manifested in different areas of secondary infection. Abscess in the temporal lobe and cerebellum possibly originated from otitis media and mastoiditis; abscess in the frontal lobe developed from dental infection and paranasal/ frontal/ ethmoid sinuses infection.[5,11] However, abscess in the parieto-occipital lobe is rarely mentioned. Previous studies of occipital lobe abscess were mentioned in a child with tetralogy of Fallot,[12] occipital dermal sinus,[13] and even cryptogenic.[14] No clear pathogenesis behind this was explained.

In the previous study in Lagos Nigeria over an 11- year period, 89 children presented with brain abscesses and the total duration of antibiotic therapy was 6–8 weeks. Cefotaxime (or ceftriaxone), amikacin (or gentamycin), and metronidazole were the most frequently used -unless otherwise dictated by sensitivity pattern- based on the presumed etiologic agent. Vancomycin was used in the patients with MRSA or as indicated by sensitivity pattern. Other antibiotics used were meropenem, cefuroxime, cefpodoxime, and cotrimoxazole. However, antibiotic regimes alongside minimally invasive percutaneous aspiration or craniotomy are sometimes needed as indicated.[3]

As the abscess progresses, complications might occur. Hydrocephalus is an uncommon complication of brain abscess. The previous study denotes hydrocephalus in <0.05% of children with brain abscesses[2] and its scarce nature is still unknown. The pathogenesis of liquor passage disturbance is suspected because of obstruction in one of the ventricle atriums. As the abscess and edema extend, they act as space-occupying lesions that further compress the choroid plexus's circulation, resulting in ventricular expansion and symptoms of projectile vomitus and seizure. Since the lateral ventricle gave the most significant expansion, it is believed that the compression began to disturb its surroundings-including pons-suppressing respiratory regulation, causing respiratory acidosis and eventually respiratory failure.

An infant's inadequate verbal capacity accounts for higher cerebral abscess mortality, as the delayed diagnosing by >7 days influences later sequelae.[11,15] Sudden worsening of symptoms usually indicates abscess rupture[16] Previous retrospective

studies conveyed similar cases projecting similar outcome; children with hydrocephalus tend to have unfavorable outcome.[15]

Though this might be the first study regarding this topic, there are a few limitations. Analysis of the cerebrospinal fluid as the gold standard for diagnosing suppurative intraparenchymal infections etiology[10] and CT-Scan with contrast or MRI as the modality with higher sensitivity was never done, as well as the information of subject's history of vaccination against meningeal pathogens.

Table 1. Routine blood test on the eleventh day

Examination (unit)	Results	Reference	Interpretation
Hemoglobin (g/dL)	11.6	10.4 – 15.6	Normal
Leukocyte (10^9/L)	44.89	6.0 – 18.0	High
Basophil (%)	0	0-1	Normal
Eosinophil (%)	0	1-5	Low
Lymphocyte (%)	7	37-73	Low
Monocyte (%)	10	1-11	Normal
Segmented Neutrophil (%)	72	30-40	High
Band Neutrophil (%)	5	0-8	Normal
Myelocyte (%)	5		
Metamyelocyte (%)	1		
Hematocrit (%)	39.9	35.0-51.0	Normal
Erythrocyte (10^9/L)	4.79	3.70-5.20	Normal
RDW (%)	13.4	11.5-14.5	Normal
MCV (fL)	83.3	76.0-92.0	Normal
MCH (pg)	24.2	23.0-31.0	Normal
MCHC (g/dL)	29.1	32.0-36.0	Low
Thrombocyte (10^9/L)	472	150-450	High
MPV (fL)	9.1	7.2-11.1	Normal
PDW (fL)	9.3	9.0-13.0	Normal

Abbreviations: MCH: Mean Corpuscular Hemoglobin; MCHC: Mean Corpuscular Hemoglobin Concentration; MCV: Mean Corpuscular Volume; MPV: Mean Platelet Volume; PDW: Platelet Distribution Width; RDW: Red-Cell Distribution Width

Table 2. Arterial Blood Gas test on the seventh day twelfth

Examination (unit)	Results	Reference	Interpretation
pH	6.845	7.350 - 7.450	Very Low
SBC (mmol/L)	18.8	22.0 - 26.0	Low
pO2 (mmHg)	45.1	83.0 - 108.0	Low
O2 Saturation (%)	50.8	95.0 - 98.0	Low
BE-b (mmol/L)	-5.8		Low
pCO2 (mmHg)	181.6	35 - 45	Very High
HCO3 (-) (mmol/L)	32.5	21.0 - 28.0	High
CO2 Total (mmol/L)	38.5	23.0 - 27.0	High

Abbreviations: BE-b: Base excess; CO2: Carbon dioxide; HCO3-: Bicarbonate; O2: Oxygen; pCO2: Partial pressure of carbon dioxide; pO2: Partial pressure of oxygen; SBC: Standard bicarbonate

CONCLUSION

Cerebral abscess is a devastating condition due to various complications, including hydrocephalus and other neurologic dysfunction. Identifying cerebral abscesses and their etiology as soon as possible proposes a better outcome. Diagnostic imaging, including contrast CT-Scan or MRI, helps provide clues towards any complications and treatment planning. Unfavorable outcomes in this study were mostly attributable due to delayed presentation and unsatisfying scrutiny, causing delayed diagnosis and intervention.

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CONFLICT OF INTEREST AND FUNDING RESOURCES

None declared.

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