Journal of Neuroinfectious Diseases

Lisandra et al., J Neuroinfect Dis 2014, 5:2
http://dx.doi.org/10.4172/ 2314-7326.1000154

Research Article Open Access

Cranial Epidural Abscess Successfully Treated with Conservative Measures: Case Report

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Received date: March 15, 2014; **Accepted date:** May 21, 2014; **Published date:** May 29, 2014

Abstract

Acute Bacterial Meningitis (MBA) is important causes of morbidity and mortality. Suppurative complications of MBA are frequent, especially in children, but are poorly described in the literature. Epidural abscess usually occurs as a result of otorhinolaryngological infections, cranial trauma or neurosurgical procedures and is rarely associated with MBA. Epidural abscess evolves to presents with focal neurological deficits due to compression process and extension to the brain parenchyma, requiring neurosurgical treatment most of the time. We report a case of meningococcal meningitis complicated with an extensive cranial epidural abscess. Clinical treatment with antibiotics was sufficient for a complete resolution of the disease.

Keywords: Acute bacterial meningitis; Meningococcal meningitis; Epidural abscess

Introduction and Objective

The Acute Bacterial Meningitis (MBA) is a serious public health problem, especially in developing countries, where vaccines are not yet available for all the population. The most common etiologic agents are *Haemophilus influenzae, Neisseria meningitidis* and *Streptococcus pneumoniae.* MBA is characterized by high morbidity and may result in sequelae [1-3]. Suppurative complications are frequent, however, poorly described in the literature.

Epidural abscess is a relatively rare condition and refers to the infection located between the duramater and the skull (cranial epidural abscess - CEA) or spine (spinal epidural abscess - SEA). The initial focus of CEA is usually in the paranasal sinuses, the mastoid cells and the middle ear. The SEA, in turn, usually stems from hematogenous spread or by extension of vertebral osteomyelitis [1].

The development of CEA as a complication of MBA occurs most commonly among children under 1 year of age [2]. Among adolescents and adults CEA generally occurs as a result of otorhinolaryngological infections, particularly infections of the paranasal sinuses, head trauma or neurosurgical procedures [1-4].

This paper describes a case of extensive CEA as a complication of acute meningococcal meningitis.

Case Report:

A 17 years old male, previously healthy, was admitted to São José Hospital of Infectious Diseases in north-eastern of Brazil. The patient presented with a history of two days severe headache associated to fever, chills and vomiting, progressing to confusion and lethargy. On examination axillary temperature was 38.3ºC, heart rate of 120 bpm, arterial blood pressure of 130/80 mmHg; the patient was agitated; cardiopulmonary auscultation was normal; abdomen examination was normal. Stiffness of neck was present and Kerning and Brudzinski's signs were positive. A Computerized Tomography (CT) of brain was no abnormalities.

Laboratory tests revealed hemoglobin of 14 mg/dL, hematocrit of 41.2%, platelets of 181,000/mm3, leukocytes of 22,100/mm3 (91% neutrophils, 4% lymphocytes, 2% basophils, 3% monocytes). A first cerebrospinal fluid (CSF 1) obtained by lumbar puncture was consistent with a bacterial infectious process (Table 1). Treatment with ceftriaxone was readily established (2g, IV, 12/12h), and the patient responded with remission of fever. However, headache and stiffness of neck persisted. Culture of CSF 1 resulted in isolation of *Neisseria meningitidis serogroup* B. Antibiogram showed sensitivity to ceftriaxone, carbapenems and aminoglycosides, by disk diffusion method. After 7 days of treatment there was a recrudescence of fever. A clinical and neurological examination revealed persistence of neck stifiness, without neurological focal deficit.

A new study of the Cerebrospinal Fluid (CSF 2) showed increased cellularity (Table 1). A new CT of brain revealed the presence of a homogeneous epidural collection without contrast uptake, localized in the right temporal lobe without involvement of the paranasal sinuses (Figure 1A).

Blood cell counts and chest x-ray were normal. Culture for bacteria in the blood and urine were negative. Meropenem (1 g IV, 8/8h) replaced ceftriaxone at this moment, based on the antibiogram. Patient was assessed by medical team of infectious diseases and neurological surgeon. Neurosurgery drainage was suggested. However, due to clinical improvement without significant neurological damage, the medical staff decided to keep conservative measures and require further investigations (CT and CSF) after 10 days of treatment with meropenem.

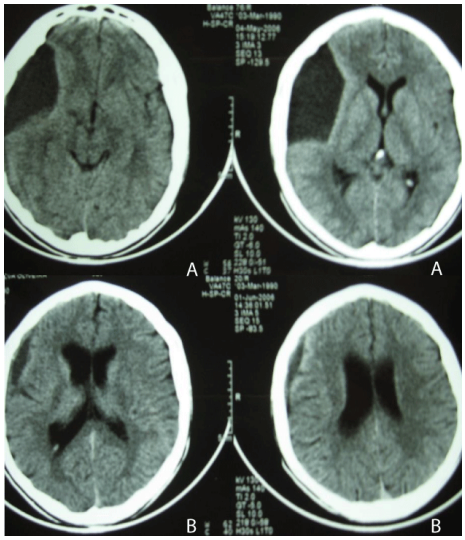

Figure 1: CT of brain with contrast (axial cuts) showing the evolution of epidural abscess in the right temporal lobe: A) homogeneous epidural collection, not captante of contrast medium, measuring 6.24 x 3.48 cm; B) cortico-subcortical hypodensity with peripheral uptake of contrast medium, measuring 3.78 x 1.39 cm.

A third CSF was collected (CSF 3, Table 1) and an improvement of pleocytosis was noted. The second CT of brain showed significant radiological improvement (Figure 1B). Cultures of CSF 2 and 3 were negative for pyogenic bacteria. Patient responded with progressive improvement of symptoms. Meropenem was held after 28 days and patient was discharged without any neurological deficit. He returned in an outpatient visit after 30 days, with no complaints or neurological deficits and was finally discharged.

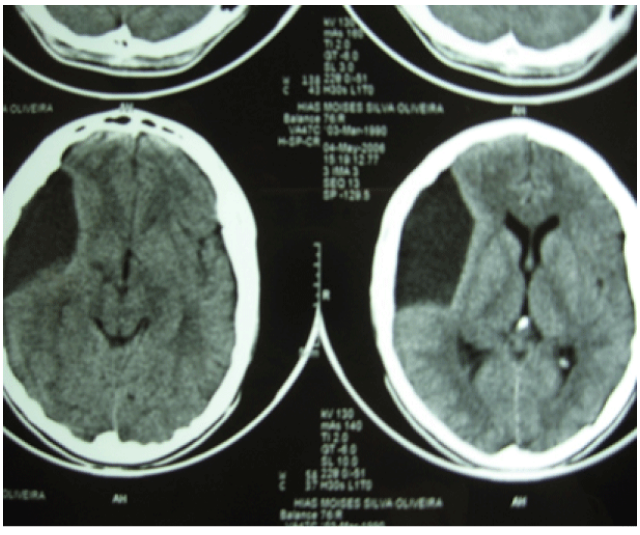

Figure 2: A second CT of brain showing a cortico-subcortical hypodensity measuring 3.78 x 1.39 cm, located in the right temporal lobe.

	CSF 1	CSF 2	CSF 3
Day of hospitalization	D0	D7	D17
Antimicrobial therapy	------	Ceftriaxone	Meropenem
Appearance	Cloudy	Clear	Colorless
Cellularity (cells/mm3)	380	1,253	218
Lymphocytes (%)	4	77	88
Neutrophils (%)	94	18	8
Monocytes (%)	2	5	4
Red blood cells (million/ml)	768	1	0
Glucose (mg/dl)	0	43.5	42.6
Protein (mg/dl)	175	117	45.6
Bacterioscopy by Gram	Gram negative diplococci	Absence of bacteria	Absence of bacteria
Culture	Neisseria meningitidis serogroup B	Negative	Negative

Table 1: Cellular and biochemical characteristics of cerebrospinal fluids analyzed

Discussion

Significant morbidity and mortality occurred in patients with MBA before the advancement of antimicrobial treatment. Neurological sequel occurred more often in children [3]. Christmas et al. found a complication rate of around 14% in children older than 6 months old with an MBA, where most were ventriculites, subdural hygroma, hydrocephalus, subdural empyema and brain abscess. Some authors have found low frequency of epidural abscess as localized infections of central nervous system, usually occurring as a complication of sinusitis. About 50% evolved to severe neurological deficits [5,6].

Complications of suppurative MBA are suspected by the persistence of fever, sensory changes or appearance of neurological focal signs despite adequate antimicrobial therapy [1,2]. Onset of symptoms is insidious and occurs through demonstrations of focal or primary infection. Because the duramater is entrapped by the opposite surface of the skull, a CEA usually causes focal neurological deficits and focal or generalized seizures. In the absence of treatment, papilledema and other signs of increased intracranial pressure may develop suggesting the presence of a large collection [1].

Computerized tomography and nuclear magnetic resonance are the methods of choice for diagnosis of CEA, since both shows a circumscribed area with decreased superficial density. In general the therapy of cerebral epidural abscess requires a combination of medical and surgical treatment [1,7-13]. For large collections with mass effect associated with neurological disorders such as motor deficit, decreased sensations or seizure. The neurosurgical drainage may be delayed if the collection is small and there is no neurological damage in asymptomatic patients. Conserved treatment can be considered in this scenario, although the complications rate is very high [14,15].

According to Oban and Rosenblum (1992), the indications for non-surgical approach to infections of the CNS associated with purulent

collections are limited. In general, this conservative option should be restricted to patients with neurological disorders and unable undergo to a surgical procedure. The case reported here had an unusual and surprising evolution because an extensive CEA was not associated to corresponding clinical and neurological findings. The recrudescence of fever after 7 days of treatment with ceftriaxone was the main factor for the suspicion of antimicrobial resistance by mechanism of selective pressure or pyogenic complication, which led the medical staff to replace the antimicrobial drug and implement an imaging examination.

Few reports have been published in the literature describing CEA as a complication of MBA in young-adult patients. In the case reported here the favorable outcome and considerable regression of the collection was a determining factor in the postponement of surgery. This case illustrates that the volume of the collection should not be used alone as criteria for surgical drainage of CEA. Other parameters such as neurological disorders (motor deficit, decreased sensations or seizures) should be considered.

Conclusion

This case reveals an atypical evolution with epidural abscess in a patient with meningococcal meningitis. Conservative treatment should be considered only in asymptomatic patients without neurological injury. Regular monitoring of the clinical and radiological status is necessary due to the high possibility of a surgical intervention.

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