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CASE REPORT

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Acinobacter Lowfii, an unusual cause of subdural empyema in pediatric

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ABSTRACT

Extradural seat empyema is a complication of mastoiditis or infected cholesteatoma. Subdural empyema, however, is synonymous with dural effraction following chronic sinusitis or may occur as a complication of meningitis or meningoencephalitis. Clinical presentation consists of signs of increased intracranial pressure, seizures, neurological deficit, and impaired consciousness. In general, an inflammatory syndrome is present; however, fever might be absent initially which may delay the diagnosis. Subdural empyema is rare and Acinetobacter lwoffii infection is even rarer. We have not found in the literature any meningitis or empyema secondary to Acinetobacter lowfii recently described in children. Therefore, we present this exceptional case , of a previously healthy 4-month-old child, who presented with bilateral empyema during an atypical evolution of meningitidis, admitted in the pediatric Hospital. The neurological weakening and tirelessness of the fever beneath twofold anti-microbial treatment drove us to perform a cerebral MRI which uncovered a subdural emyema. This child profited from a craniotomy departure of discharge, cytobacteriological analysis found an acinobacter Lowfflii. Physicians ought to consider the possibility of subdural empyema in children with meningitides.

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1. INTRODUCTION

Subdural empyema (SDE) is the most frequent type of intracranial empyema; it is more rare than cerebral abscess [1]. In general, the most frequent location is the frontal or the front-parietal regions, yet it might be found in interhemispheric and rarely in the posterior fossa. The incidence of subdural empyema is 12% [2]. The impact of the abscess, on the other hand, is constant in all the studies; it is up to 2 to 4% [3,4] with a male predominance (57.8%) [5-8]. It is rare for an infectious process to extend directly by continuity from sinusitis or osteitis, and gradually forming, leading to subdural septate [9]. Infection of the submucous veins, devoid of valves, sinus cavities [10] is transmitted to the subdural veins retrogradly. It is in this space that the infection develops, whereas a meningeal reaction tends to limit it by the formation of fibrin deposits which contributes to forming neome membranes, then an encapsulation [11]. Postmeningitic endocranial suppuration in newborn children and youthful children is concomitant with meningitis, by dividing the contamination within the arachnoid and the subdural space.

Bacteriologically, the most frequently incriminated germs are staphylococcus, streptococcus and anaerobes [12]. In half of the cases bacteriological examinations reveals a polymicrobial flora [13]. More rarely, Hemophilus Aspergillus [14,15] and mycoplasma [16] can be found. The use of neuroradiological investigations (CT and MRI) and the change of neurosurgical strategies have made it conceivable to clearly make strides the forecast of endocranial suppuration; Hence ; mortality fell from the previous high rate of 50% [17,18].

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2. CASE REPORT

A 4-month-old infant admitted for the management of an infectious syndrome. He was admitted for deterioration of general condition in a febrile context, with a temperature of 40° evolving for 48 hours, with impaired neurological exam (hyperexitability, axial hypotonia and refusal to breastfeed). His weight was 7200 g, his height 55 cm and his head circumference 42 cm. Full blood count showed increased white blood cell count at 10.94 × 109/L; with 73% neutrophils and 19.4% lymphocytes; C-reative protein was 90 mg/L. A lumbar puncture revealed a citrine aspect of the cerebrospinal fluid with a protein concentration of 1.82 g/l and glucose concentrations of 0.20 g/l with a blood sugar level of 0.98 g/l and 90 elements/mm^3 with a predominance of lymphocytes and 40 red blood cells/mm^3. Forty-eight hours later, the infant remained feverish and his neurological examination deteriorated with exaggeration of hypotonia, laterality of gaze, segmental hypertonia. The cranial circumference remained the same. the blood cultures were negative and the culture of the lumbar puncture fluid did not find pathogens.

A cerebral MRI showed a bifrontal parietal empyema with purulent intraventricular sediment(Figures 1 and 2), which has been suspected by the practice of a transfontanellar ultrasound. The infant subsequently benefited from craniotomy evacuation of pus and excision of the outer membrane at the neurosurgery department.

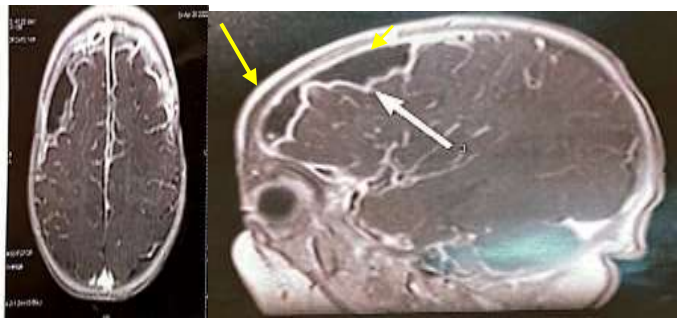

Figure 1. Rim-enhanced lesions suggestive of bifrontal parietal empyema

Even before receiving the analysis of the drainage product, antibiotic regimen was changed to ceftazidime (115 mg/kg/d) and metronidazole 30mg/kg/d, phenobarbital was added at the dosage of 5mg/kg/d. Antibiotics were rationalised and continued for a median of 6 weeks following surgery. An Electroencephalogram found paroxysmal waves in the right frontal area. Treatments were tapered when electroencephalogram returned without abnormalities (Two months). The analysis of the drainage liquid found an Acinetobacter lwoffii multisensitive according to the antibiogram, therefore we decided to keep the same therapy, which had made it possible to obtain apyrexia from 72 hour. The neurological examination normalized on 10^{th} day of treatment. The child is doing well with a follow-up of height months.

Figure 2. whitest pus collection underneath the dura.

3. DISCUSSION

Post meningitic endocranial suppuration, abscess and subdural empyema are rare. Their frequency varies from 1 to 12% depending on the field and depending on the definition chosen for empyema, it represents 4% in a series of 320 cases of bacterial meningitis [19]. The first signs appear at different times according to the authors [20-22]. The delay in the onset of symptoms is 48 hours in our patient after admission for his meningitis, compatible with a delay described by certain authors [23]. Alongside fever and signs related to the initial attack, altered consciousness is the main warning sign in 10.4% of cases of intracranial suppuration [24] with a Glasgow score ranging from 4 to 12 , found in 47.2% of cases [25]. Worsening of neurological disorders with persistence of fever in our patient, were in favor of a cerebral collection.

The complete blood count showing hyperleukocytosis, predominantly neutrophils, is often noted during SDE. It is present in 80 to 100% of cases, it can be moderate, sometimes very high, exceeding 20,000 elements/mm^3.The sedimentation rate is accelerated [26]. The cytobacteriological analysis of the pus of the empyema revealed the responsible germ. A predominant neutrophilic hyperleukocytosis was present in our patient, with a frankly positive inflammatory assessment and cytobacteriological samples have individualized Acinetobacter Lowflii.

Acinetobacter species are a frequent skin and oropharyngeal commensal and have become a prominent cause of nosocomial

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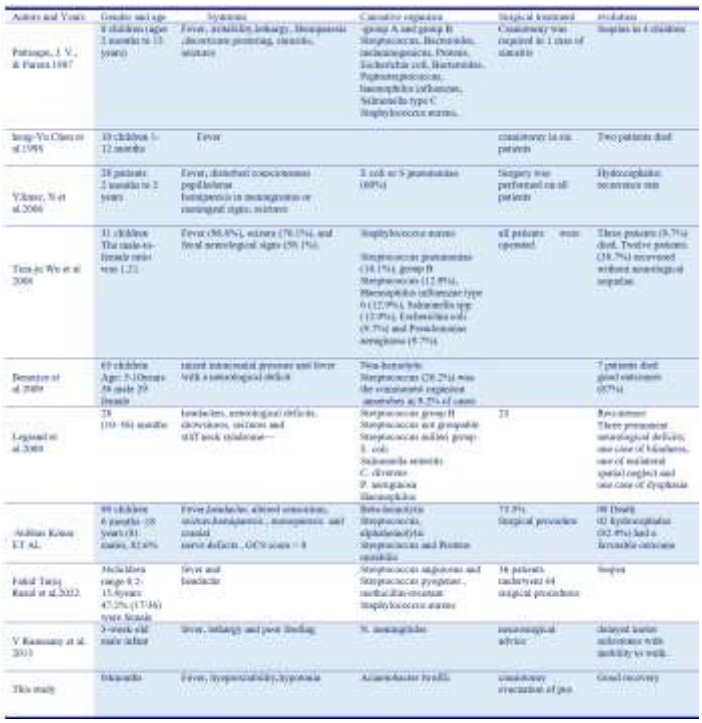
infection and, in recent years, have shown increased resistance to a broad spectrum of antimicrobials. Acinetobacter baumanni is the species of most clinical importance and is associated with hospital epidemics; however, other species have been linked to human sickness, such as Acinetobacter lwoffii [27-34]. They are also part of the commensal flora of the skin in humans since an epidemiological study revealed the presence of cutaneous colonization with Acinetobacter spp. in 43% of non-hospitalized subjects[35].The most frequently isolated species were: A. lwoffii (58%), A. johnsonii (25%), A. junii (10%) and Acinetobacter genomic species 3 (6%) [36-39]. For this purpose, we did not know the mode of contamination by this organism in our patient. The source of infection was certainly proven in only one study [40]. A Lowfii was found in the natural flora of the skin and the oropharynx and perineum of 20 -25% of the healthy individuals that was not normally pathogenic, has been implicated in infections such as endocarditis, endophthalmitis, meningitis, gastritis, bacteraemia secondary to catheter infections in immunocompromised patients or secondary to acute gastroenteritis.[41-47].

We found no reported cases of Acinetobacter Lowfii meningitis since 1965-1989 when Acinetobacter Lowfii was at the time called “Mima Polymorpha” in case reports of neonatal meningitides. So, we believe that our observation is exceptional for recent pediatric cases. [48-54] (Table 1). Two observations have been reported recently, of Acinetobacter Lowfii meningitis in adults [55- 57]. Very recently, another observation in an adult with posty covid 19 infection who had presented an acinetobacter lowfii meningitis was reported [58]. Lumbar puncture is contraindicated in cases of suspected SDE [59,60], or more generally in the presence of focal neurological signs, a state of stupor or a coma given the risks of cerebral herniation, or even death that may potentially result. In our patient, it was performed before the onset of subdural empyema. The trans-fontanellar ultrasound practiced in infants, has a major diagnostic interest in terms of SDE, because it does not require preparation, it is inexpensive and can be repeated several times. It allows to see the localization and extent of the SDE and a possible mass effect [61,62]. Magnetic resonance imaging better visualizes the empyema especially at the early stage. MRI is more precise and contrast enhancement allows better localization, it also allows a better characterization of the nature of the effusion (blood, sterile effusion or pus) [63]. SDE is shown on MRI by hypointensity in the T1 sequence and hyperintensity in the T2 sequence [64-67]. MRI is more effective in demonstrating edema and ischemic lesions. MRI angiography is also effective in terms of associated sinus thrombosis and hemorrhagic softening [68-70]. Trans-fontanellar ultrasound helped us to suspect the diagnosis of subdural empyema best described by magnetic resonance imaging.

Table 1. Literature review of published series of SDE in children.



Once the diagnosis is confirmed, medical treatment is started, based on broad-spectrum antibiotic therapy, anticonvulsants and sometimes anti-edematous drugs [71-74]. The optimal duration of antimicrobial treatment for patients with subdural empyema or effusion has not been established in trials or comparative studies, but general recommendations are to treat patients with empyema for 3–4 weeks if an empyema has been evacuated, and even longer if the patient is conservatively treated [75-78]. There is discussion over the benefits and hazards of commencing antibiotics before surgical drainage, which is said to minimize the rate of microbiological diagnosis. Our patient has benefited from surgical drainage with antibiotic therapy adapted to the antibiogram for six weeks, associated with an antiepileptic treatment maintained for one month. This therapy allowed us to have a good clinical, radiological and electrical evolution and our patient was normal after eight months.

The delay of therapeutic management remains the main factor in poor prognosis of SDE [79,80]; However, the prognosis remains burdened with a 4% mortality and frequent sequelae including venous sinus thrombosis, stroke, cerebral oedema, and seizures. Mortality is approximately 4%, and up to 35% of patients have a residual neurologic deficit [81-84]. Risk factors for mortality in children were well identified in in cases of Acinetobacter baumannii bacteremia especially [85,86].

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4. CONCLUSION

Intracranial empyema deserves to be known, researched, and treated on time. It would be necessary consider an SDE before the association of neurological signs and unexplained fever. New imaging techniques allow early diagnosis and urgent management of intracranial empyema.

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