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Case Report

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Recurring *E. coli* meningitis in an infant

Abstract

Bacterial meningitis is a life threatening disease, and although most cases present in a single episode, a few cases of acute bacterial meningitis can be recurrent. We review the case of a 5-month-old infant who presented recurrent meningitis by *E .coli*, in association to a pilonidal sinus within the center of a lumbosacral angioma. Meningitis produced due to congenital defects in the caudal neural tube communicating the skin with the meninges are usually associated to skin bacteria, most frequently by *S.aureus,*[1,5] however, the particularity of our case resides in that being an infant using diapers and having a congenital defect in the caudal neural tube,[2] the stools become a potentially infective inoculums, and *E. coli* should be taken into consideration as well as other enterogenic bacteria.[1,3] We will review the etiology of recurrent bacterial meningitis and discuss the antibiotic guidelines, the amount of days of treatment, and the time schedules of the neurosurgical repair of the congenital defect.

Keywords: selective transfer, superficial layer, structural analysis, intensity x-rays, width of diffraction lines, crystalline network constant

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Introduction

Despite advances in diagnostic approach, antibiotic therapy and intensive care, bacterial meningitis is still associated with a significant mortality and a high number of complications and neurological sequelae. Although the vast majority are single episodes, approximately 1-4.8% of all cases of acute bacterial meningitis are recurrent, which is defined as the reappearance of signs and symptoms of meningitis by the same microorganism or a new episode by a different microorganism that occurs at least 3 weeks after the sterilization of the cerebrospinal fluid (CSF). Different factors predisposing to recurrent meningitis have been identified, such as deficits in humoral and cellular immunity and congenital or acquired structural defects that establish an anatomical communication between the subarachnoid space and the skin or the middle ear and the paranasal cavities. The clinical history and the findings of the physical examination and the microbiological data can help guide the origin of meningitis. A prompt identification of immunological deficiencies or anatomical defects is important in preventing new episodes and potential sequelae.

Clinical case

A 5-month-old infant that arrives to the pediatric emergency room (ER) with a chief complaint of fever of 12 hours (38ºC axillary), associated to vomits and irritability. A perinatological history of a pilonidal sinus within the center of a lumbosacral angioma detected at birth stands out, with an initial medullar ultra sonography that did not detect any communication. The rest of variables were within normal range. The physical exam revealed an angiomatous cutaneous lesion in the lumbosacral region L5-S1 with a pilonidal sinus and a hair follicle in the center of the lesion (Figure 1). Blood work showed WBC: 21300/mm3 (PMN 59%, Bands 3%, Lymphocytes 29%, Monocytes 9%), CRP: 143, 2mg/L and Procalcitonine: 2, 39ng/mL; VGB and the rest within the normal range. The CSF obtained was of purulent aspect, CSF cell count: WBC 8240/mm3 (PMN: 82%), CSF glucose: 0.18mg/dL, CSF protein: 0.86g/L, CSF gram stain: Gram negative bacillus, CSF culture grows an *E. coli*. Immunologic study without alterations. The patient was admitted and started treatment with cefotaxime (300 mg/kg/day),[3] showing a clinical improvement within 6 hours of starting the treatment. The antibiotic was maintained for 22 days, with normalization of the CSF values by the end of the treatment.[5] A new medullar ultrasound in the lumbosacral region was performed finding a hypoechoic tubular image starting form a hyperechoic subcutaneous nodular lesion in the sacral region that continues in depth into the sacral osseous elements. An MRI was then performed observing a fistula starting in the lumbosacral pilonidal sinus and continuing into the dura mater with a sacral osseous dysrafia (Figure 2). Within a month of discharge, and while waiting for the corrective neurosurgery, the patient presents with a new meningitis by *E. coli*, starting treatment again with cefotaxime (300mg/kg/day), receiving antibiotic treatment for 30 days in this second episode. A few days before ending the second course of antibiotics the surgical correction was successfully performed, with a resection of the cutaneomeningeal fistula and closure of the plains from skin to sacral meninges. Following the surgery, the infant has remained asymptomatic and has not presented any complications or new infections.

Figure 1 Cutaneous pilonidal sinus within the center of a lumbosacral angioma.

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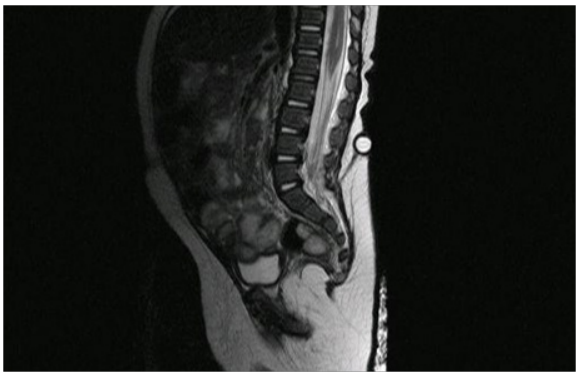

Figure 2 Medullar MRI. Cutaneomeningeal lumbosacral fistula.

Discussion

A review by Trebuegge and Curtis of a total 363 cases of recurrent meningitis in children and adults in 144 publications between 1988 and 2008,[4] found that 214(59%) were related to anatomical problems, 132(36%) were related to immunodeficiency, and 17(5%) were related to parameningeal infections. Streptococcus pneumoniae was the most commonly isolated organism, responsible for almost 60% of the cases reviewed, followed by Neisseria meningitides, with most cases associated to complement-deficient patients, and only a few cases by Haemophilus influenza, without any relevant immunological or anatomical association, and finally with only a few cases caused skin flora and gram negative bacteria, usually associated to cutaneomeningeal defects, with Staphylococcus aureus being most commonly implicated in occipital lesions and Escherichia coli and enterococcus in lumbosacral lesions. In the presence of a patient with recurrent meningitis, it is important to rule out both anatomical defects and immunological deficiencies as an underlying cause and identifying the bacteria responsible for the infection can help guide in pointing to the underlying cause. In our patients' case, the presence of a flat angioma in the lumbosacral region was the entrance to the subarachnoid space, causing two episodes of *E. coli* meningitis. The discussion of this case resides on three main aspects: the amount of days of intravenous antibiotic treatment are needed, the amount of days a patient can wait between end of the course of intravenous antibiotics[5] and the corrective neurosurgery, and finally when the corrective surgery should be performed. After reviewing the literature, some authors state that 21 or more days are recommended for *E. coli* meningitis,[1,3,6] while others recommend 10-15 days[4] and some even give a 10 day option followed by a CSF control and, if sterile and with normal values, treatment can be stopped.[5] In reference to the corrective surgery of a fistula communicating the skin and meninges, there is no general consensus for how much time should pass until the correction, but literature agrees that the corrective neurosurgery is recommended as soon as the meningitis is healed (with a normal CSF study and negative CSF culture) because its high risk of recurrence[2]. Additionally, there is no International consensus about whether a congenital defect of this type once diagnosed could wait for its corrective surgery for a determined amount of time.[2]

Conclusion

In patients with recurrent meningitis the possibility of an anatomical defect should be considered. The isolated microorganism should help to locate it. It is essential to know the normal flora of the different anatomical sites. The definitive treatment is usually surgical.

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Conflict of interest

The author declares there is no conflict of interest.

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