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A rare case of peripheral facial paralysis associated with meningococcal meningitis - case report

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ABSTRACT

Introduction. Meningococcal meningitis is a severe form of bacterial meningitis caused by the bacterium *Neisseria meningitidis (N. meningitidis).* It is a medical emergency and requires prompt treatment to prevent severe complications or death. Peripheral facial paralysis is one of the rare complications that can occur in this condition.

Aim. This paper aimed to draw attention to the possibility of an unexpected complication in a child with meningococcal meningitis, which requires prompt clinical and paraclinical management.

Materials and methods. We present the case of a 2-year and 8-month-old female patient who, in the context of meningococcal meningitis, develops peripheral facial paralysis, a very rare complication, especially in the pediatric population.

Results. The patient initially presented with clinical and paraclinical signs of a respiratory viral infection. However, in a febrile context, she has two generalized tonic-clonic paroxysmal episodes, characterized by gaze fixation, each lasting 2-3 minutes, followed by spontaneous recovery. Laboratory tests revealed neutrophilia, anemia, and elevated inflammatory markers, while the cranio-cerebral CT scan showed no structural changes. Blood cultures were also collected, and treatment was initiated with third-generation cephalosporin antibiotics, along with supportive and symptomatic therapy. The following day, the patient exhibited signs of right-sided peripheral facial paralysis, and intravenous corticosteroid therapy was added to the treatment. Approximately 24 hours after admission, the blood culture tested positive for gram-negative cocci, suggesting meningococcal meningitis. A cerebral MRI was performed, revealing changes indicative of acute meningoencephalitis. The clinical and paraclinical course was slowly favorable, with a slight persistence of facial asymmetry and intermittent ataxic syndrome.

Conclusions. Meningococcal meningitis remains a relevant pathology in the pediatric population, able of causing severe complications that can result in long-term disability for the patient. Prompt treatment is crucial when this condition is suspected.

Keywords: *N. meningitidis,* meningitis, child, facial paralysis

INTRODUCTION

Neisseria meningitidis (N. meningitidis) is a Gram-negative diplococcus that can cause invasive meningococcal disease, such as bacterial meningitis. It is a member of the normal nasopharyngeal microbiome and can spread through aerosols or oral and nasal secretions [1].

The most frequent clinical presentations of meningococcal infection are meningitis and septic shock, though in certain instances, both conditions may occur simultaneously. Early symptoms, like a runny nose and sore throat, can mimic those of typical viral respiratory infections. The incubation period typically spans from 1 to 14 days, but in most cases, it is shorter than 2 days. [2].

The clinical presentation of the illness can differ based on age, and children are frequently misdiagnosed in the early stages. In young children, symp-

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toms may be more subtle, with vague signs that make diagnosis more difficult compared to older children or adolescents. Irritability and lethargy are common at this age. Seizures may also occur in some cases, initially presenting as focal and later becoming generalized [2]. Furthermore, neck stiffness is uncommon in children under 2 years of age. In infants younger than 18 months, the anterior fontanelle may bulge. Typically, infants experience a faster progression of the disease compared to older children [3]. Similar to adults, older children most commonly present with symptoms such as fever, nausea, vomiting, photophobia, headache, agitation, decreased consciousness, and neck stiffness. However, seizures and focal neurological signs are less frequent in this age group [2-4]. Septic shock is more prevalent in children and tends to progress rapidly, frequently resulting in multiple organ failure and death within 24 hours. Frequently, nonspecific symptoms such as fever, drowsiness, nausea, vomiting, irritability, and poor feeding are present within the first 4-6 hours of illness onset. A rapidly progressing hemorrhagic rash is one of the most common symptoms associated with sepsis, typically beginning on the lower extremities, though it can also affect mucous membranes or even the sclerae [2-3]. Skin lesions may present as macules, papules, petechiae, purpura, and ecchymoses. The purpuric rash can advance to purpura fulminans, a severe skin manifestation of disseminated intravascular coagulation (DIC). These cases are frequently linked to septic shock and may result in skin necrosis, as well as ischemia in the extremities, which usually necessitates amputation [4-5].

Bacterial meningitis can lead to numerous complications. Short-term issues may include seizures, focal neurological deficits, and subdural effusions, while long-term effects can involve hearing loss, cognitive impairment, hydrocephalus, learning difficulties, and epilepsy [6].

It is crucial that when bacterial meningitis is suspected, antibiotic treatment is started immediately, without delay. Empirical antibiotic choice should be based on the most likely pathogen depending on the patient's age [7]. In children, third-generation cephalosporins, such as cefotaxime or ceftriaxone, are commonly used as the empirical treatment to cover the most frequent pathogens – *S. pneumoniae* and *N. meningitidis.* Ampicillin should be included to cover *L. monocytogenes* in very young children. Some guidelines recommend this for children under 3 months of age, while others advise its use for those under 1 month old [8].

Humans are the only hosts for *N. meningitidis.* Nasopharyngeal colonization is quite common, with carrier rates ranging from 10% to 35%. While the disease is most common in infants, carriers are most frequently young adults (some university students in the UK having carrier rates up to 55%). Invasive disease is rare and occurs after the microorganisms translocate into the bloodstream, a process whose mechanism remains unclear [9].

Epidemiological data from 25 European countries have shown an annual incidence of invasive meningococcal disease ranging from 0.3 to 2.9 cases per 100,000 inhabitants, with a significant annual declining trend in most countries. However, *N. meningitidis* is associated with substantial mortality (5%-10%) and severe permanent disabilities, such as cognitive deficits, hearing loss, visual impairments, and epilepsy [10-12].

The most effective way to prevent neurological complications from bacterial meningitis is through vaccination programs for infants and children. Although several vaccines have been developed against the pathogens that cause bacterial meningitis, outbreaks of vaccine-preventable meningitis still occur [13]. Currently, vaccines are available for three key organisms: Hib, *N. meningitidis* (capsular groups A, B, C, W, and Y), and 23 of the more than 90 serotypes of *S. pneumoniae.* There are two types of vaccines for *N. meningitidis:* conjugate vaccines targeting capsular groups A, C, W, and Y, and protein-based vaccines for group B [14-15].

Facial paralysis in children can be either acquired or congenital. Acquired causes include infections, Bell's palsy, trauma, and tumors. In a multi-year review of facial paralysis cases at Boston Children's Hospital, the most common cause was infectious, with most of these cases being complications of acute otitis media. Other infectious causes of facial paralysis include herpes simplex virus, Epstein-Barr virus, human immunodeficiency virus, cytomegalovirus, mumps virus, and rubella virus [16].

CASE REPORT

Reasons for Admission

We present the case of a 2-year and 8-month-old female child with a medical history of multiple upper respiratory tract infections, who presented to the local Emergency Department with mucopurulent rhinorrhea and dry cough that began approximately 3 days prior to the day of admission. On the day of presentation, she developed a fever and, in the context of the fever, experienced a paroxysmal episode characterized by generalized tonic-clonic movements, with gaze fixation, lasting 2-3 minutes, without urinary incontinence, and with spontaneous recovery, preceded by an episode of vomiting. This manifestation repeated upon arrival at the local emergency service, leading to the administration of rectal Diazepam and antipyretics. The patient was then transported to the Pediatric Emergency Service of the Tg. Mures County Emergency Clinical Hospital, where laboratory tests were performed, and admission to the Pediatric Clinic I was decided.

Physical examination

Upon admission, the clinical examination revealed influences general condition, ailing face, pale skin, hyperemic pharynx, mucopurulent rhinorrhea, bilateral basal lung crackles, oxygen saturation: 89% on room air, 99% with oxygen via mask, heart rate 110 beats/minute, weight 11 kg.

Evaluation and diagnosis

Laboratory tests performed at admission revealed marked leukocytosis (40,210 × 10^3/μL) with neutrophilia (35,580 × 10^3/μL, 88%), slightly decreased hemoglobin (9.3 g/dL) and albumin (3.7 g/dL) levels, significantly elevated inflammatory markers (ESR: 68 mm/h, CRP: 264.8 mg/L). The cranial CT scan showed a large fluid collection in both maxillary sinuses, with no other pathological changes. Other infectious causes were also excluded: TORCH profile was negative, as were tests for SARS-CoV-2 and other respiratory viruses (Influenza A and B, RSV). Given the affected general condition, clinical, and paraclinical findings, symptomatic treatment, hydration and electrolyte rebalancing, and antibiotic therapy with a third-generation cephalosporin were initiated. On the second day of admission, the clinical examination revealed right-sided facial asymmetry, right-sided lagophthalmos, the loss of nasolabial fold and right forehead wrinkles, oral commissure deviated to the left, findings that suggested right-sided peripheral facial paralysis, as well as neck stiffness and ataxic gait with a wide base of support. The blood culture collected at admission identified gram-negative cocci. However, since the partial result was communicated approximately 12 hours post-admission, ceftriaxone was already initiated with favorable evolution, and considering the increased sensitivity of *N. meningitidis* to ceftriaxone, a lumbar puncture was deemed no longer necessary. Based on the anamnesis, clinical, and paraclinical elements, the diagnosis of meningococcal meningitis was established.

Management. Monitoring. Evolution.

The patient was hospitalized in the Pediatric Clinic I for 16 days, during which she was monitored both clinically and paraclinically. Throughout the hospitalization, paraclinical reassessments showed slow but favorable progress, with normalization of the leukocyte count and inflammatory markers, as well as serial neurological evaluations. A cerebral MRI revealed T2 FLAIR hyperintense areas in the occipital-parietal gyri bilaterally, cerebellar gyri bilaterally, and the walls of the occipital horns of the lateral ventricles, all suggesting acute meningoencephalitis (Figure 1 and Figure 2). The patient received antibiotic treatment, steroid anti-inflammatory treatment, symptomatic therapy, depletive treatment, and electrolyte rebalancing. She showed slow but favorable evolution, with no seizures and significant improvement in facial paralysis. However, a mild residual asymmetry and intermittent ataxic syndrome persisted, without gait imbalance or signs of meningeal irritation. On the 16th day of hospitalization, the patient was discharged in good general condition, afebrile, with stable cardiac and respiratory status, and neurologically with intermittent ataxic syndrome and mild facial asymmetry. Recommendations included continued antiepileptic treatment with Phenobarbital and periodic neurological reevaluation. Unfortunately, due to low parental compliance, the case was lost to follow-up.

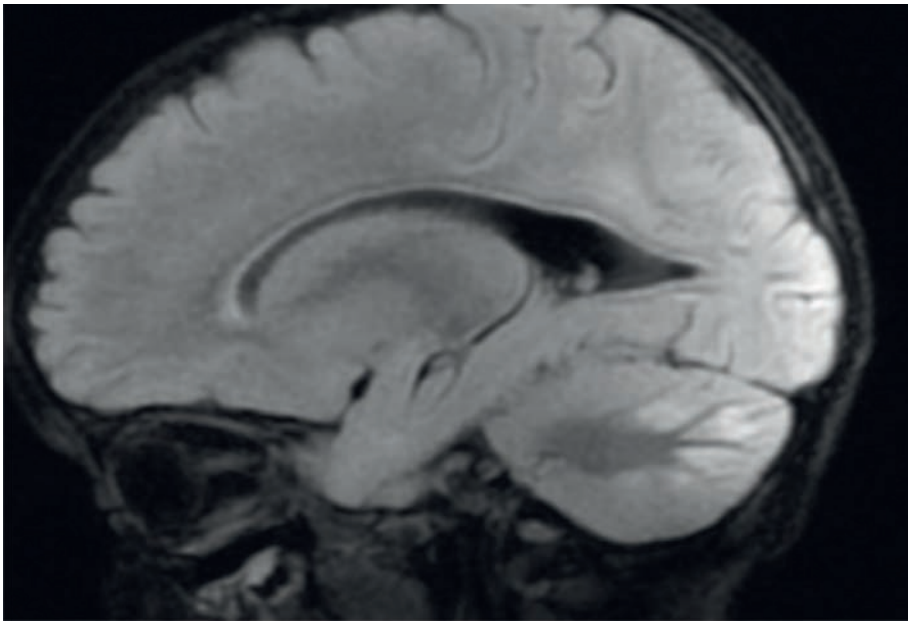

FIGURE 1. T2 FLAIR hyperintense areas in the occipital-parietal gyri bilaterally, cerebellar gyri bilaterally

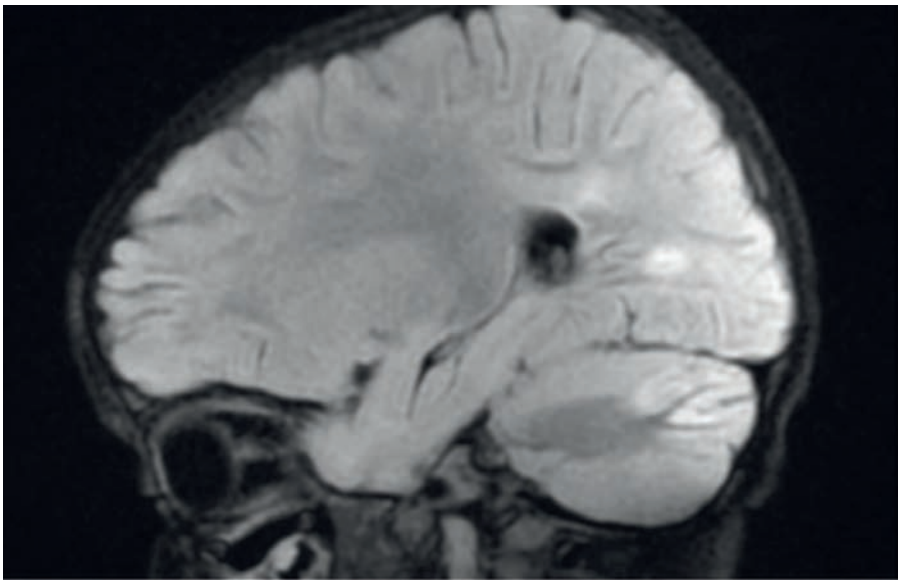

FIGURE 2. T2 FLAIR hyperintense areas cerebellar gyri bilaterally, and the walls of the occipital horns of the lateral ventricles

DISCUSSIONS

N. meningitidis is a Gram-negative diplococcus responsible for meningococcal disease, a major cause of sepsis and community-acquired meningitis in children. While relatively uncommon, meningococcal disease remains a significant global health concern in pediatric populations, with an overall mortality rate of about 8% and the potential for serious neurological complications [17-18]. *N. meningitidis* is classified into 13 capsular groups based on its polysaccharide capsule. However, only six of these groups – A, B, C, W, X, and Y – are most commonly linked to disease in humans [19]. Meningococcal disease arises when the pathogen enters the bloodstream, causing systemic infections that can include meningitis, sepsis (meningococcemia), or both [20].

Facial nerve paralysis is the most prevalent cranial nerve dysfunction in both children and adults. Although it is less common in children, it can significantly affect the child and cause considerable anxiety for parents. The causes of facial paralysis in children often differ from those in adults. When the underlying cause can be determined, acute bacterial otitis media is the most frequent condition leading to facial paralysis in children, due to anatomical and immunological factors [21]. The facial nerve (cranial nerve VII) has motor, sensory, and parasympathetic functions and is particularly vulnerable to injury because of its extended bony pathway. It occupies 25-50% of the total diameter of the Fallopian canal. This rigid bony canal may not expand to accommodate nerve inflammation, potentially leading to vascular compromise of the nerve [22]. There are six segments of the facial nerve within the temporal bone: intracranial, meatal, labyrinthine, tympanic, vertical and extratemporal [23].

Cranial nerve paralysis during acute bacterial meningitis is not uncommon, as it can be present at the onset of the infection or during its course in 9%-12% of adult patients. Specifically, disturbances in the oculomotor, abducens, facial, and glossopharyngeal nerves are often observed [24-26]. However, cranial nerve paralysis in infections caused by *N. meningitidis* is rarely described. Literature searches reveal only a few cases in adult patients and one pediatric case. In 1995, Chiu et al. reported the first case of cranial nerve paralysis in a 30-day-old male infant, in whom *N. meningitidis* was isolated from cerebrospinal fluid and blood cultures. Thus, four days after onset, the patient developed left-sided oculomotor and facial nerve paralysis, and cranial CT revealed an infarct in the bilateral frontal lobes [27]. In our case, we are dealing with a young child diagnosed with meningococcal meningitis based on clinical and paraclinical findings, who subsequently developed peripheral facial paralysis. Peripheral facial paralysis is a rare complication but can occur in the context of inflammation and edema of the meninges that may extend to the cranial nerves, including the facial nerve. This complication is particularly associated with bacterial meningitis, as is the case here. Given that the clinical and paraclinical progression was favorable following antibiotic treatment and especially after initiating corticosteroid therapy, we interpreted this clinical manifestation as a complication of meningococcal meningitis.

As previously mentioned, primary prevention can be achieved through vaccination. Unfortunately, in our country, the meningococcal vaccine is not part of the mandatory vaccination schedule, and most children do not receive this vaccine. In this case, the patient came from a disadvantaged social background and was unvaccinated, placing her in a higher risk category for developing this often fatal disease.

As indicated above and according to the CDC, the incidence of meningococcal meningitis varies across different regions of the world, with a global estimate of 0.3 to 3 cases per 100,000 people per year outside of epidemic periods. The global mortality rate is approximately 10-15% despite treatment, however, without treatment, mortality exceeds 50% [15]. In the case presented, fortunately, early intervention with antibiotic therapy led to a favorable outcome, despite the complications that occurred.

CONCLUSIONS

Meningococcal meningitis remains a significant concern for pediatricians worldwide, especially in developing countries where vaccination rates are very low, leading to a higher risk of this often fatal infection. It is crucial to consider this diagnosis as soon as a child presents to the Emergency Department, as prompt and correct treatment can be life-saving. Among the numerous acute and long-term complications, peripheral facial nerve paralysis is a very rare complication. Both *N. meningitidis* infection and its complications require prompt recognition and treatment, and the involvement of a multidisciplinary team is essential to minimize long-term sequelae.

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