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Anesthesia

Fusobacterium Nucleatum and Meningitis

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Abstract **Case Report**

Fusobacterium nucleatum is a rare cause of meningitis in adults, yet it is associated with significant morbidity and mortality. We present a case of meningitis due to Fusobacterium nucleatum in a 28-year-old male with a history of head trauma two years prior, recurrent pneumococcal meningitis, and a cerebrospinal fluid leak. Initial cerebrospinal fluid (CSF) cultures were sterile despite severe inflammatory abnormalities, and the patient was treated with vancomycin and penicillin. His condition worsened neurologically, requiring intubation, mechanical ventilation, and sedation. A subsequent anaerobic culture identified Fusobacterium nucleatum, and metronidazole was added to the treatment. The patient passed away on the third day due to cerebral edema and intracranial hypertension.

Keywords: meningitis, Fusobacterium nucleatum, cerebrospinal fluid leak.

INTRODUCTION

Fusobacterium nucleatum is a strictly anaerobic, Gram-negative, fusiform bacillus of variable size and non-sporulating [1,2]. Although meningitis caused by Fusobacterium nucleatum is rare, it can become severe if not promptly treated [3].

Management involves a combined antibiotic therapy, including a beta-lactam (cephalosporin, penicillin) and an agent targeting anaerobic microbes (metronidazole, clindamycin), for 15 days [1,5]. Delayed treatment of Fusobacterium nucleatum meningitis can lead to severe complications and even death [9]. Hospital monitoring is essential.

CASE PRESENTATION

The patient, a 28-year-old male, had a history of head trauma two years ago, recurrent pneumococcal meningitis, and a cerebrospinal fluid leak. He was admitted to the emergency department with a 5-day history of headaches, vomiting, and fever.

Vital signs upon admission:

temperature 39°C, heart rate 100 bpm, respiratory rate 18/min, blood pressure 125/60 mmHg, oxygen saturation 98% on ambient air, blood glucose 1.8 g/L. Neurological examination revealed impaired consciousness (Glasgow Coma Scale 12/15), neck stiffness, and no focal deficits.

Biological findings:

leukocytosis (21,000 cells/mm³), elevated C-reactive protein (292 µg/L), normal electrolytes. CSF analysis showed cloudy fluid with 22,000 cells/mm³ (95% neutrophils), albumin 5.78 g/L, glucose 0.02 g/L, and negative direct examination. Chest X-ray was unremarkable.

The treatments with ceftriaxone (70 mg/kg/day), vancomycin (60 mg/kg/day), and dexamethasone was initiated. Four days later, the patient's neurological condition deteriorated (Glasgow 10/15), requiring intubation, mechanical ventilation, and sedation with midazolam and fentanyl. Antibiotics were maintained. Computed tomography (CT) and magnetic resonance imaging (MRI) revealed a brain abscess and ventriculitis, with meningitic changes (Figures A, B, C and D). CSF anaerobic culture identified Fusobacterium nucleatum. Consequently, metronidazole (1.5 g/day) was added. Unfortunately, the patient's condition worsened, and he died three days later from cerebral edema and intracranial hypertension.

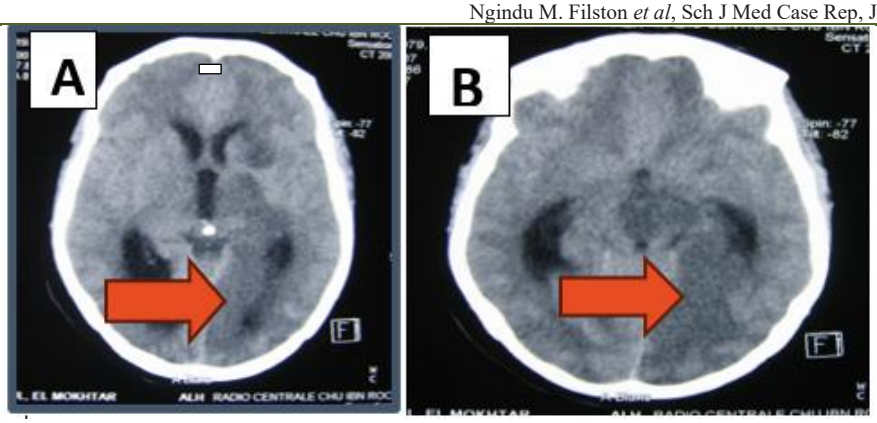

Figure A, B: Computed tomography (CT)

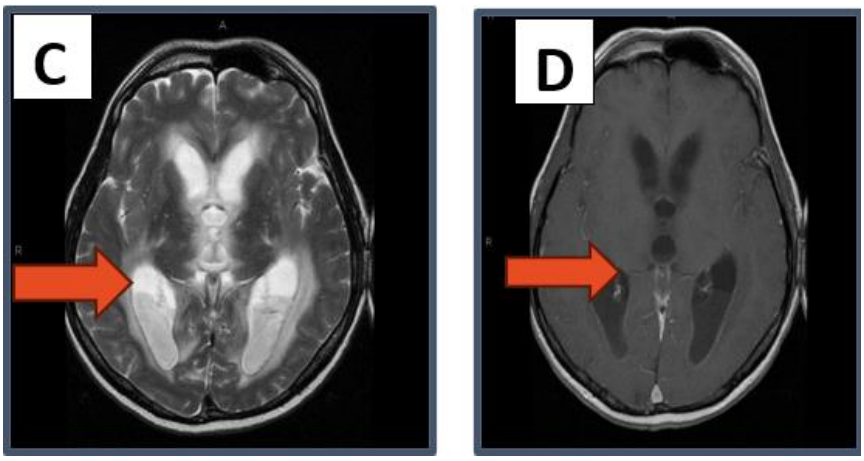

Figure C, D: magnetic resonance imaging (MRI)

DISCUSSION

Fusobacterium is a strictly anaerobic, Gram-negative, fusiform, non-sporulating bacillus found in the human oral cavity, intestinal tract, and genital tract at low levels [2,4,11]. It primarily produces butyric acid, which has a characteristic odor [3,6]. Fusobacterium includes 16 species, the most pathogenic being F. nucleatum, isolated from clinical samples in various conditions such as appendicitis, osteomyelitis, pericarditis, Vincent's angina, lung abscesses, oral-dental infections, genital and intestinal infections, meningitis [12-14], and adverse pregnancy outcomes like chorioamnionitis [15]. F. nucleatum can cause severe complications, including epilepsy, brain abscess, and cerebrovascular diseases [9,10].

Epidemiology

Anaerobic germs account for 10% of bacterial infections overall. Anaerobic meningitis, estimated at less than 1% of bacterial meningitis cases, appears underreported [2,7]. This may be due to the lack of systematic use of techniques for detecting and identifying anaerobic germs. Typical clinical features of bacterial meningitis include headaches, fever, vomiting, neck stiffness, or altered consciousness [9]. Our patient presented all these signs, consistent with bacterial meningitis. Microbiological diagnosis can be delayed or missed because F. nucleatum requires strict anaerobic conditions and prolonged incubation for isolation [9]. This explains the initially sterile CSF culture in our case, despite cytological evidence of bacterial meningitis. CSF infection may occur through contiguity or, rarely, via bacteremia [16]. In this case, the patient had a cerebrospinal fluid leak, a known risk factor.

Prognosis and Treatment:

The prognosis for anaerobic meningitis is excellent if treated promptly. Most clinical isolates of F. nucleatum are sensitive to antibiotics, including metronidazole, clindamycin, and beta-lactams. Resistance to penicillin has been reported [8]. Beta-lactam antibiotics are becoming less effective due to the production of beta-lactamases by some strains, necessitating the use of beta-lactamase inhibitors like clavulanic acid, sulbactam, or tazobactam, which do not penetrate the meninges. Metronidazole, however, is highly effective against anaerobic bacteria, with good meningeal penetration and tolerability [1,5]. It should be combined with third-generation cephalosporins due to the possible presence of other aerobic or anaerobic pathogens and beta-lactamase production. Antibiotic therapy is typically administered intravenously for 1–2 weeks, followed by approximately 10 days of oral treatment [5]. Fusobacterium is sensitive to beta-lactams, metronidazole, tetracyclines, and colistin, but resistant to neomycin and vancomycin [8], which explains the patient's initial deterioration.

CONCLUSION

Fusobacterium nucleatum is a rare but severe and often underdiagnosed cause of meningitis [7]. Its detection should be systematic in patients with risk factors for the disease. Rapid diagnosis and early treatment are essential.

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