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Staphylococcus Hominis Meningitis In A Paediatric Patient With Grade II Protein-Energy Malnutrition: A Rare Entity

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Abstract
Background: Coagulase-negative staphylococci (CoNS) are recognized as comprising the main part of human normal skin flora and are rarely associated with severe and intensive infections. However, these organisms can cause several diseases in humans, especially immunocompromised patients and neonates. For example, Staphylococcus hominis can cause meningitis.
Here, we came across a case of Staphylococcus hominis meningitis in a grade II (IAP Classification) PEM patient.
Case presentation: A 10-year-old boy with grade II protein and energy malnutrition presented to the emergency department with a history of fever (101.80 F), lethargy and headache, vomiting, photophobia, and neck rigidity. Examination of cerebrospinal fluid (CSF) revealed increased protein level, neutrophil pleocytosis, and reduced blood glucose level. Gram stain of CSF fluid showed plenty of pus cells with a few gram-positive cocci. The CSF sample was subjected to an automated aerobic culture system in BACTEC, followed by VITEK. The isolate was identified as Staphylococcus hominis with antimicrobial sensitivity. Clinical improvement was observed on treatment with antibiotics like vancomycin as a sensitive drug in the VITEK system, along with supportive treatment with corticosteroids, diuretics, fluids, and blood products. The duration of the antibiotic treatment was 14 days.
Conclusion and Result: Staphylococcus hominis meningitis, though uncommon, can cause severe disease in immunocompromised states like PEM, and it requires proper intervention following prompt diagnosis and treatment.

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I. Introduction

Coagulase-negative staphylococci (CoNS) comprise the majority of the human normal microbiota and are rarely associated with severe and intensive infections. Although these organisms can cause several diseases in humans, especially in immunocompromised patients and neonates, for example, Staphylococcus hominis can lead to meningitis [(1).]

Bacterial meningitis, as an important and acute infection in the central nervous system, is still a major global health challenge and a serious infectious disease, causing a high rate of mortality and morbidity [(2)].

Coagulase-negative staphylococci (CoNS) are a diverse group of Gram-positive cocci that comprise the majority of the human normal microbiota [(1)]. Traditionally regarded as non-pathogenic commensals and frequent contaminants in microbiological cultures, they have emerged over recent decades as significant opportunistic pathogens, particularly in immunocompromised hosts, neonates, and patients with indwelling medical devices. Common pathogenic CoNS species include *Staphylococcus epidermidis*, *S. saprophyticus*, *S. haemolyticus*, and *S. lugdunensis*. Among these, *Staphylococcus hominis is* a commensal organism predominantly inhabiting apocrine gland-rich skin areas. S.hominis is ranked third among the coagulase-negative bacteria that are of clinical importance. A previous study by Kloos et al. implicated S. hominis in the skin and soft tissue infections in immunocompromised individuals. Methicillin-resistant S. hominis (MRSHo) are all capable of causing infections and are usually more likely to show multiple resistance to antimicrobial agents than other coagulase-negative Staphylococci. In addition to its occasional pathogenicity, S. hominis may be a reservoir of specific components of the methicillin resistance genetic element, staphylococcal cassette chromosome (SCCmec), that may be transferable to more pathogenic staphylococcal species [(24)].

The pathogenicity of S. hominis in immunocompromised individuals has been of grave concern recently [6]. *Staphylococcus hominis* can form biofilm on the surface of smooth devices in the human body, and it is considered to be a potential pathogen, which can cause different infections such as infective endocarditis and meningitis in immunocompromised patients [3,10].

Meningitis caused by CoNS is relatively rare and typically associated with neurosurgical interventions, shunt placement, or head trauma. Cases of spontaneous CoNS meningitis, especially those caused by S. hominis, are exceedingly rare, with only isolated reports in the literature [4,5,7-13]. Invasion of the central nervous system (CNS) by *S. hominis* in the absence of prior neurosurgical procedures is unusual. It poses diagnostic challenges, as the organism is often dismissed as a culture contaminant [14].

Protein-energy malnutrition (PEM) is a known cause of secondary immunodeficiency in children, leading to impaired cell-mediated immunity, reduced complement activity, and diminished phagocytic function. This immunological compromise predisposes affected individuals to a broad spectrum of infections, including those caused by atypical and low-virulence organisms such as CoNS[15-16]. However, a comprehensive search of indexed medical literature reveals a scarcity of reports describing *S. hominis* meningitis in paediatric patients with PEM.

Here, we describe a rare case of *S. hominis* meningitis in a child with grade II PEM according to the Indian Academy of Pediatrics (IAP) classification. We aim to highlight the pathogenic potential of *S. hominis* in immunocompromised pediatric populations, emphasize the role of species-level identification of CoNS, and raise awareness among clinicians and microbiologists regarding the need for careful interpretation of CoNS isolates in high-risk hosts.

II. Case Presentation

A 10-year-old male child presented to the paediatric emergency department with a history of high-grade fever (101.8^0°F) for 5 days, lethargy and headache, photophobia, irritability for 3 days, and two episodes of vomiting over the past 24 hours. There was no such history of seizure, trauma, neurosurgical procedure, or prior hospitalisation. Developmental milestones were appropriate for age. Immunisation status was complete as per the National Immunisation Schedule.

Nutritional status assessment revealed grade II protein-energy malnutrition as per the Indian Academy of Paediatrics classification: weight-for-age was 65% of the expected weight, mid-upper arm circumference was 12.5 cm, and there was evidence of mild muscle wasting. Dietary history indicated inadequate protein intake over the preceding months.

On examination, the child was febrile (temperature 101.8°F), irritable, and mildly dehydrated. Heart rate was 120/min, respiratory rate 26/min, and blood pressure 100/70 mmHg. Neurological examination revealed neck stiffness and positive Kernig's and Brudzinski's signs. Fundus examination was normal. No focal neurological deficits were noted. Other systemic examinations were unremarkable.

Laboratory investigations showed haemoglobin 8.9 g/dL, total leukocyte count 40,500/mm³ with neutrophil predominance (88%), and erythrocyte sedimentation rate (ESR) of 40 mm in the first hour. C-reactive protein was elevated (16 mg/L). Serum electrolytes, renal, and liver function tests were within normal limits.

Lumbar puncture showed clear cerebrospinal fluid (CSF) with the following analysis:

- Total leukocyte count: 180 cells/mm³ (predominantly neutrophils – 85%)
- Protein: 109 mg/dL (elevated)
- Glucose: 25 mg/dL (serum glucose: 76 mg/dL)
- Gram stain: Gram-positive cocci in clusters with plenty of pus cells
- Acid-fast bacilli stain: Not found
- India ink: Negative
- MPDA- Negative
- WIDAL TEST-Negative
- CBNAAT-Negative
- CHEST X RAY-WNL
- CECT OF BRAIN-WNL

CSF was inoculated onto blood agar, chocolate agar, and MacConkey agar and incubated aerobically. After 24 hours, small, non-hemolytic colonies were observed on blood agar. Gram staining confirmed Gram-positive cocci in clusters. The isolate was catalase-positive, coagulase-negative, and identified as *Staphylococcus hominis* using the VITEK® 2 automated identification system (bioMérieux, France). Antimicrobial susceptibility testing revealed sensitivity to vancomycin, linezolid, and clindamycin, with resistance to penicillin and oxacillin (methicillin-resistant phenotype).

Blood culture performed at admission was negative after 5 days of incubation in BACTEK. Chest radiograph and cranial computed tomography were normal.

The child was started empirically on intravenous ceftriaxone and vancomycin at admission. Based on culture and sensitivity results, ceftriaxone was discontinued, and vancomycin was continued for a total of 14 days. The patient became afebrile by day 4 of therapy, with gradual resolution of irritability and meningeal signs. Nutritional rehabilitation was initiated concurrently with a high-protein diet and micronutrient supplementation.

The patient was discharged in good clinical condition after 16 days of hospitalisation. At the 4-week follow-up, the child was neurologically intact, gaining weight, and thriving well.

III. Discussion

Coagulase-negative staphylococci (CoNS) usually have low-virulence skin commensals and are often dismissed as contaminants when isolated from clinical specimens [(1)]. However, in recent decades, CoNS have emerged as significant opportunistic pathogens, especially in immunocompromised patients, neonates, and those with indwelling medical devices or post-neurosurgical interventions [(1,2,5,6).] Among the CoNS species, *Staphylococcus hominis* is infrequently reported as a cause of invasive disease, with most literature describing bloodstream infections, endocarditis, or device-associated infections [(7,19)]. Meningitis caused by *Staphylococcus hominis* is extremely rare, especially in patients without a history of neurosurgery [(14,18)].

In the present case, *S. hominis* was isolated in pure culture from cerebrospinal fluid (CSF) in a child with grade II protein-energy malnutrition (PEM). Clinical features, CSF cytochemical profile, and the absence of alternative pathogens supported the diagnosis. The possibility of contamination was considered but deemed unlikely because (i) the organism was isolated from a sterile site in a significant clinical context, (ii) Gram-positive cocci with plenty of pus cells were seen in the direct Gram stain of CSF, and (iii) the patient demonstrated clinical improvement with targeted anti-staphylococcal therapy.

PEM is a well-recognized cause of secondary immunodeficiency in children. Malnutrition impairs both innate and adaptive immune responses, including reduced complement activity, diminished phagocytic function, and atrophy of lymphoid tissues, leading to decreased T-cell numbers and impaired cytokine production [(15,16)]. This immunological compromise predisposes patients to infections by both common and opportunistic organisms, including CoNS, which would rarely cause invasive disease in immunocompetent hosts.

A literature search reveals very few documented cases of *S. hominis* meningitis in paediatric patients, and to our knowledge, this is the first case in India, bacterial meningitis with PEM. In a review by Becker et al., *S. hominis* accounted for less than 1% of clinically significant CoNS isolates in invasive infections, underscoring its rarity as a CNS pathogen. Previous reported cases have largely been associated with neurosurgical interventions, prosthetic material, or severe immunosuppression due to malignancy or transplantation [(1,5,18)]. Our case expands the spectrum of at-risk populations to include children with moderate malnutrition.

From a microbiological perspective, species-level identification of CoNS is essential for appropriate interpretation and management. Automated identification systems, such as VITEK 2, enable rapid and accurate differentiation of CoNS species. This is critical because certain species (e.g., *S. lugdunensis*) are more virulent, and antimicrobial resistance patterns vary among species. In our case, the *S. hominis* isolate exhibited methicillin resistance, necessitating vancomycin therapy, to which the patient is sensitive and responded well.

This case also highlights the importance of careful clinical correlation when CoNS are isolated from sterile body fluids. While contamination is common, the presence of compatible clinical features, relevant host factors (e.g., malnutrition), and concordant microbiological findings should prompt consideration of true infection and initiation of targeted therapy without delay.

IV. Conclusion

Staphylococcus hominis is an uncommon cause of meningitis, particularly in the absence of neurosurgical interventions or prosthetic devices. This is, to our knowledge, the first Indian report of *Staphylococcus hominis* meningitis in a pediatric patient with PEM. This case shows that in the setting of protein-energy malnutrition, the organism's low virulence can be overcome by the host's impaired immune defences, leading to severe central nervous system infection. Accurate species-level identification of coagulase-negative staphylococci, along with careful clinical correlation, is essential to differentiate true infection from contamination. Early recognition and targeted therapy can lead to favorable outcomes even in resource-limited settings.

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