Sir,

Bacterial meningitis is associated with high mortality and morbidity, especially in children. Most common pathogens causing bacterial meningitis in children are *Haemophilus influenzae* type B (Hib) and *Streptococcus pneumoniae*, accounting for up to 75% of cases.[1] From March 2010 to April 2011, 400 children between 0 and 15 years old with symptoms of acute central nervous system (CNS) infection were screened, of which 54 who had cerebrospinal fluid (CSF) white blood cell count ≥10 cells/µl were recruited while those with history of road traffic accidents, CNS anomalies, and postoperative meningitis were excluded.

Conventional CSF culture, BacT/ALERT for CSF, latex agglutination, and conventional polymerase chain reaction (PCR) to detect the capsular polysaccharide gene of *S. pneumoniae* and TaqMan real-time PCR to detect the *bcs B* gene of Hib were performed with the sequences of the primers and PCR protocol described by Pai et al.[2] and Mayer,[3] respectively.

Fever was observed in 90% of children, vomiting and seizures in 51% of children. In 23 of the 54 children, a cause of CNS infection could be established. Thirteen (24%) of the 54 children were confirmed to have pyogenic bacterial meningitis, with eight due to *S. pneumoniae*, four due to Hib, and one case of *Streptococcus pyogenes* meningitis was detected by BacT/ALERT culture.

Culture, PCR, and latex agglutination detected five cases of *S. pneumoniae* and one case of Hib, whereas six cases were detected by nonculture methods, three pneumococcal meningitis by PCR only, one Hib meningitis by PCR and LA, and one case by LA alone. Spike and dilution tests performed on this sample ruled out PCR inhibitor; this was considered a false negative PCR test. Studies have shown that nonculture methods have performed better, as demonstrated by Kennedy et al.[4]

In the 41 children who did not fit in the pyogenic group, three children were diagnosed as tuberculous meningitis based on a standard case definition and response to anti-tuberculosis treatment, four as rickettsial meningitis with Weil-Felix test positive along with the presence of eschar in three of them and all four showing dramatic response to doxycycline, three children as neurocysticercosis by standard diagnostic criteria including computed tomography scans showing typical ring-enhancing lesions. Interestingly, at 6 months follow-up, sequelae were seen in children who did not have a definite diagnosis; this reiterates that identification of the agent leads to effective treatment which reduces mortality and morbidity.[5]

In conclusion, culture and nonculture methods including radiological methods are necessary for the diagnosis of meningitis. This study reiterates the need for a battery of tests for the detection of these etiological agents causing CNS infections. Further studies to identify other etiological agents, especially viral causes need to be evaluated to diagnose meningitis in children.

**Acknowledgments**

We acknowledge Dr. Maria Carvalho and Dr. Leonard W. Mayer, Meningitis and Vaccine Preventable Diseases Branch, Division of Bacterial Diseases, National Centre of Immunization and Respiratory Diseases, Centre for Disease, Atlanta, GA, USA, for providing the protocol and reagents for PCR.

**Financial support and sponsorship**

Fluid Research Grant of Christian Medical College, Vellore.

**Conflicts of interest**

There are no conflicts of interest.

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Sir,

Achromobacter is a Gram-negative aerobic bacilli known to cause opportunistic infections in neonates and patients with HIV infection,[1] cancer,[2] cystic fibrosis,[3] and hyper-immunoglobulin M syndrome.[4] We report a case of Achromobacter xylosoxidans sepsis with septic arthritis in a child with suspected severe combined immunodeficiency (SCID).

A 2-year and 11-month-old male child presented with complaints of high-grade fever with swelling and restriction of movement of bilateral knee joints for 10 days. There was a history of multiple respiratory infections starting at 1 year of age, including an episode of empyema thoracis. Initial investigations revealed total leukocyte count 28,360 cells/mm$^3$, neutrophil 65%, lymphocyte 25%, monocyte 7%, eosinophils 3% (absolute neutrophil count = 18,434/mm$^3$ and absolute lymphocyte count = 7090/mm$^3$), and C-reactive protein 290 mg/dl. Bilateral knee aspirates revealed acute inflammatory exudate. The child was empirically started on injection ceftriaxone and cloxacillin; however, there was no response in 72 h.

On day 4, his initial blood culture and knee aspirates showed growth of A. xylosoxidans. All the isolates were sensitive to ceftazidime, ciprofloxacin, and levofloxacin and resistant to gentamicin, amikacin, ceftriaxone, and cotrimoxazole. The antibiotic was changed to ceftazidime. Patient’s joint symptoms improved and he became afebrile within 7 days of starting ceftazidime, which was continued for 6 weeks.

In view of a history of recurrent respiratory infection and disseminated infection with an opportunistic pathogen, possibility of immunodeficiency was considered. HIV serology was negative. His immune globulin profile showed severe panhypogammaglobulinemia and lymphocyte subset study was suggestive of T−NK−B+ SCID [Table 1].

**Table 1: Immunoglobulin Profile and Lymphocyte Subset Analysis of the Patient**

| Immunoglobulin Profile | Lymphocyte Subset Analysis by Flow Cytometry |
|------------------------|--------------------------------------------|
| IgA level: <10 mg/dl (14‑159 mg/dl) | T-cell markers |
| IgG level: <75 mg/dl (345‑1236 mg/dl) | CD3$^+$: 21.2% (control: 84.7%) |
| IgE level: <2 IU/ml (0-230 IU/ml) | CD4$^+$: CD8$^+$ ratio: 0.97 |
|                                    | B-cell marker |
|                                    | CD45$^+$CD19$^+$: 18.3% (control: 16.9%) |
|                                    | NK-cell markers |
|                                    | CD3$^−$CD56$^+$: 0.56% (control: 4.03%) |
|                                    | CD3$^−$CD16$^+$: 0.18% (control: 26.5%) |
|                                    | CD3$^−$CD56$^+$CD16$^+$: 0.05% (control: 21.3%) |
| Neutrophil oxidative index: 26.5% (control: 7.1%) | |

Intravenous immunoglobulin was given as a replacement therapy for panhypogammaglobulinemia. The child was advised monthly intravenous immunoglobulin replacement therapy and cotrimoxazole prophylaxis. He was referred for genetic testing and stem cell transplant. Mother was advised carrier testing for genetic counseling to explain the risk in future pregnancy.

A. xylosoxidans infections are more common in immunocompromised hosts with indwelling catheters, endotracheal tubes, or other medical devices. It may disseminate and cause sepsis, meningitis, and death. It is a very rare cause of septic arthritis. A. xylosoxidans is found in water sources, and well water is considered to be the source of community-acquired infections and intravenous fluid, ventilator, or dialysis fluid in nosocomial infection. It is usually multiresistant to antimicrobial therapy.

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