



Case Report

Post-neurosurgical meningitis by *Naegleria fowleri* and *Staphylococcus aureus* in Ecuador: A case report

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ABSTRACT

This report describes the first documented case in Ecuador of primary amoebic meningoencephalitis caused by *Naegleria fowleri*, in coinfection with methicillin-resistant *Staphylococcus aureus* (MRSA) associated with a ventriculoperitoneal shunt (VPS). A 23-year-old male with a history of severe traumatic brain injury developed central nervous system infection symptoms after VPS placement. Despite targeted antibiotics for MRSA, clinical deterioration continued. Whole genome sequencing confirmed *N. fowleri*, prompting antiparasitic treatment with amphotericin B and fluconazole, resulting in partial improvement. This case underscores the need to consider rare pathogens in atypical neurological cases and highlights the diagnostic value of genome sequencing in complex infections.

1. Introduction

N. fowleri (Orden Schizopyrenida, familia Vahlkampfiidae) is a free-living, thermophilic amoeba found in warm freshwater and soil environments such as hot springs, ponds, rivers, lakes, and poorly chlorinated water [1]. It presents three parasitic forms: trophozoites (10–25 µm), non-replicating flagellates (10–16 µm), and cysts (8–20 µm). Infection occurs when trophozoites enter the nasal mucosa and migrate to the brain via the olfactory nerves, causing primary amoebic encephalitis (PAM) [1,2].

PAM is a rare but nearly always fatal central nervous system infection, with a mortality rate of 98 %. The amoeba induces hemorrhagic necrosis in the brain, and death usually occurs within 3–7 days after symptom onset. Immediate treatment with multiple antibiotics is essential, although therapeutic options remain limited. The high fatality rate is largely due to delayed diagnosis and lack of effective therapies

[1,3]. Clinical awareness is critical for improving patient outcomes [4].

Globally, at least 380 cases of PAM have been reported, mostly in the United States, Pakistan, and Mexico [5]. In Latin America, 26 fatal cases of PAM have been reported in several countries, but none previously in Ecuador [6–9]. This case represents the first documented *N. fowleri* infection in Ecuador associated to a coinfection with MRSA, complicating management and prognosis. This report contributes to national epidemiological surveillance and raises important questions about the local ecology of this parasite. The case underscores the need for accurate microbiological and molecular diagnosis, as initial symptoms often mimic meningitis [3].

2. Case presentation

The patient was a 23-year-old male with a history of severe head trauma from a traffic accident in October 2023. Initially treated at a

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second-level health center in Latacunga, Ecuador, he underwent three neurosurgeries and was left with neurological sequelae and a 90 % disability. He required tracheostomy and gastrostomy procedures and was discharged under palliative care. Months later, he moved to Quito, Ecuador's capital, for outpatient follow-up in palliative care, pulmonology, gastroenterology, neurology, and neurosurgery. He remained at home with permanent family care for nutrition and hygiene.

In February 2025, during a neurology consultation, suspected cerebrospinal fluid (CSF) accumulation in the craniectomy area prompted a CT scan, which revealed ventricular dilation. The patient was admitted for VPS placement. Initial labs showed leukocytes $8.40 \times 10^3/\text{mm}^3$, neutrophils $6.41 \times 10^3/\text{mm}^3$, platelets $209 \times 10^3/\text{mm}^3$, and C-reactive protein (CRP) 0.59 mg/dl. A follow-up CT after eight days confirmed proper catheter placement and lab values within normal range. Four days later, a new bulging appeared, requiring VPS replacement due to clot blockage. The patient was discharged under neurosurgical supervision with the VPS functioning correctly and unchanged neurological status.

Twenty-seven days later, the patient returned with purulent discharge at the VPS site, new bulging in the craniectomy area, and CT showing catheter contact with ventricle walls and vacuum in the VPS. A lumbar puncture was performed, and CSF analysis included cytochemical tests (Auto Hematology Analyzer, Mindray CB-6200), bacterial culture, FilmArray meningitis/encephalitis panel (BIOFIRE® FILMARRAY® TORCH System), Xpert MTB/Rif Ultra (Cepheid GeneXpert Systems), and secretion culture. Based on altered CSF parameters (Table 1, Initial CSF), empirical treatment with vancomycin (500 mg/8 h IV) and ceftriaxone (1 g/12 h IV) was started. CSF and secretion cultures confirmed MRSA, identified via Vitek® 2 GP and AST-663 card (Biomérieux, Marcy l'Etoile, France), with resistance to oxacillin, erythromycin, and tetracycline, but sensitivity to vancomycin, ciprofloxacin, trimethoprim/sulfamethoxazole, linezolid and clindamycin. A definitive diagnosis of MRSA infection of the VPS was established.

The patient underwent VPS removal and external ventricular shunt (EVS) insertion CSF was collected for monitoring on the following days (controls #1 and #2), while cultures from the catheter tip confirmed the presence of MRSA. Considering the risk factors for multidrug-resistant organisms, the antimicrobial regimen was adjusted: ceftriaxone was discontinued after three days and replaced with meropenem (2 g every 8 h IV), while vancomycin was continued. Further CSF samples were collected for follow-up (Controls #3 and #4), and MRSA was once again detected in Control #4 (Table 1). Additionally, microbiological analysis of tracheostomy secretions revealed *P. aeruginosa* producing Verona integron-encoded metallo-beta-lactamase (VIM), identified through the

Vitek® 2 GN and AST-402 systems. The strain exhibited resistance to most antibiotics except colistin. As a result, vancomycin was discontinued after eight days and replaced with linezolid (600 mg every 12 h IV for 20 days). Following eight days of meropenem, antimicrobial therapy was further modified to cefepime (2 g every 8 h IV) and imipenem/cilastatin (500 mg every 6 h IV) in response to evolving clinical and microbiological findings. Two additional EVS replacements were performed, with no subsequent pathogen growth observed in CSF cultures.

Persistent hypoglycorrhachia, proteinorachia, and pleocytosis despite treatment led to CSF analysis by the National Genomics Reference Center “Leopoldo Izquieta Pérez” using whole genome sequencing (WGS). DNA was extracted with the Qiagen DNA Mini Kit (Qiagen, Alemania), quantified using the Quantus fluorometer (Promega, E.E.U. U), and sequenced on the Illumina MiSeq platform (MiSeq-Illumina, San Diego, California, USA). Ribosomal regions 16S, 23S, and Internal Transcribed Spacer (ITS) were amplified using specific primers, and WGS provided general pathogen identification. FASTQ data were processed using Unicycler v0.5.1 for de novo assembly and BLASTn comparison with the NCBI database, identifying *N. fowleri*. A reference genome (accession: OD957045.1) was selected for reference-guided assembly using BWA-MEM v0.7.19, and a consensus sequence was generated with iVar v1.4.2. Final confirmation was performed by BLASTn with 100 % coverage and an average sequencing depth of 1458×. The de novo assembly yielded 67 contigs, 47 of which matched *N. fowleri*. Fig. 1 summarizes the clinical events leading to this diagnosis.

After identifying *N. fowleri*, antiparasitic therapy was initiated according to Burqi et al. [10], amphotericin B deoxycholate, azithromycin, rifampicin, and miltefosine. Due to the patient's functional and nutritional status, amphotericin B deoxycholate (40 mg) was administered in 500 ml of 5 % dextrose over 6-h infusions. Fluconazole (800 mg every 12 h) was added via gastrostomy tube, along with linezolid for MRSA. Treatment led to decreased pleocytosis and proteinorachia and increased CSF glucose (Table 1). During therapy, the patient experienced electrolyte imbalances, bradycardia, and anemia, which were corrected with replacements and transfusions. Despite treatment, the patient remains with severe neurological sequelae, the patient was referred to a more complex healthcare facility where he died 35 days later due to sepsis and meningitis caused by *P. aeruginosa* producing a VIM-type metallo-beta-lactamase.

3. Discussion

The diagnosis of VPS-associated meningitis represents a clinical

Table 1
Cytochemical and microbiological analysis of CSF across sequential controls.

Laboratory parameter	INITIAL CSF	Control #1 (post VPS removal)	Control #2	Control #3	Control #4	Post treatment control
Cytochemical analysis						
Leukocytes (/uL)	1508	154	502	1121	248	129
Polymorphonuclear (/uL)	1268	108	406	881	167	90
Mononuclear (/uL)	240	46	96	240	81	39
Red blood cells (/uL)	200	600	200	600	1200	2800
Glucose (mg/dL)	8.78	16.3	9.19	5.5	13.9	32.4
Proteins (mg/dL)	70.9	48.1	94.3	108	186	140
Lactate dehydrogenase (U/L)	242	84.8	104	150	96.5	–
Lactate (mmol/L)	8.5	7.6	8.4	6.8	6.3	6.2
VDRL	No reactive	–	–	–	–	–
Microbiological analysis						
Bacterial culture	<i>S. aureus</i> (SARM)	Without growth	Without growth	Without growth	<i>S. aureus</i> (MRSA)	–
Filmarray meningitis/encephalitis panel	No detected	–	–	–	–	–
Xpert® Mtb/Rif Ultra	No detected	–	–	No detected	–	–

(–) Not done.

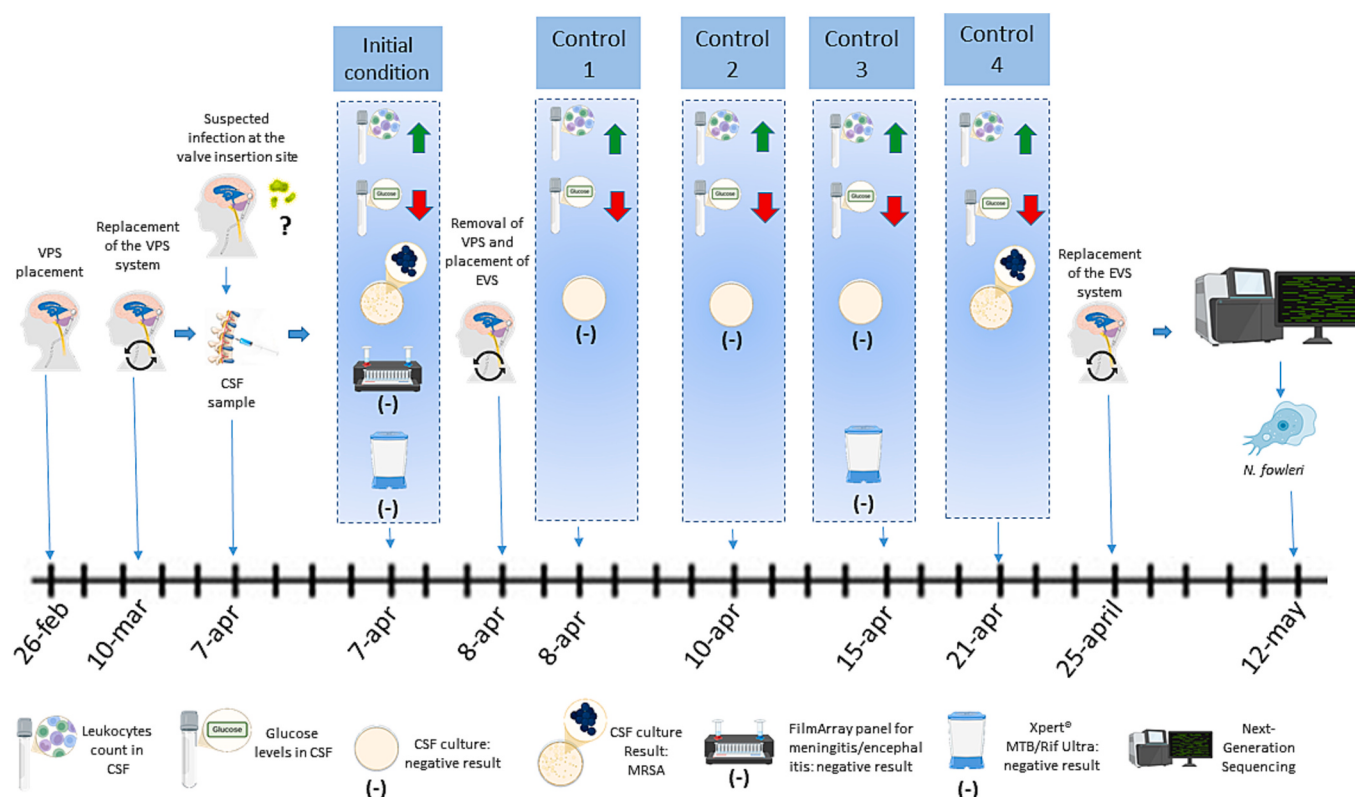


Fig. 1. Summary of the main clinical and laboratory events leading to the diagnosis of *N. fowleri* in the case. Red arrows indicate glucose levels below the normal range, while green arrows indicate an increase in leukocyte count above normal values. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

challenge, as the inflammatory response following neurosurgical procedures can mimic infection associated with the presence of meningeal signs and cytochemical alterations in the CSF. The clinical presentation of fever, purulent discharge from the valve insertion site, right parietal bulging, seizure, CSF pleocytosis and hypoglycorrhachia, in this case, was related to a VPS-associated infection, and empirical antibiotic treatment targeting the most common etiological agents was implemented [11].

The pathogenesis of *N. fowleri* begins with accidental inhalation of contaminated water. The trophozoite penetrates through the cribriform plate and exhibits selective chemotaxis toward the CNS. Depending on the inoculum size and strain virulence, PAM symptoms typically appear within 1 to 12 days post-exposure. Key clinical features manifestations include elevated intracranial pressure, cerebral edema, necrosis, and characteristic CSF abnormalities such as pleocytosis, proteinorachia, and hypoglycorrhachia [2,12]. MRSA-associated VPS infections presented with clinical signs similar to PAM, precluding early suspicion of *N. fowleri*, and making it difficult to identify the initial pathogen. The progressive evolution of the symptoms despite targeted MRSA therapy prompted further investigation through WGS.

The microbiological diagnosis of PAM due to *N. fowleri* relies on direct observation of motile trophozoites, which requires high clinical suspicion and has limited sensitivity. Techniques like indirect immunofluorescence or immunohistochemistry are rarely available in laboratories. Nucleic acid amplification for PCR offers greater precision and faster confirmation [4]. However, in cases of VPS-associated meningitis, the suspicion for *N. fowleri* is low, so routine diagnostic protocols often exclude this pathogen from differential testing. Therefore, WGS proves to be a valuable alternative, as it enables accurate, untargeted pathogen identification—even in atypical cases or when clinical suspicion is low.

The mortality rate for PAM is over 98 %, with death occurring within five days of symptom onset; only four out of 157 American patients

survived between 1962 and 2022 [13]. From the onset of symptoms, the patient remained alive for 81 days until his death associated with sepsis and meningitis associated with VPS. This unusually prolonged survival may be attributed to the continuous drainage of CSF through the EVS, which likely reduced intracranial pressure and mitigated the rapid progression of cerebral edema, a major contributor to mortality in PAM [5]. Previous studies have demonstrated similar benefits of ventricular drainage in other severe forms of meningitis, such as cryptococcal meningitis [14].

In this case, it was not possible to determine the exact epidemiological link or the time of infection. However, a possible infection associated with home care was presumed, involving exposure to domestic water sources. The patient's relatives reported routine hygiene practices at home, including bed baths with damp cloths and towels moistened with tap water, nasal washing, and daily tracheostomy suctioning, all of which may have contributed to the exposure risk.

In conclusion, this case highlights the diagnostic and therapeutic complexity of *N. fowleri* infections, particularly with coinfection by *S. aureus*. It emphasizes the value of whole genome sequencing in atypical meningitis cases and underscores the need for early suspicion, improved diagnostics, and public health awareness to reduce PAM-related mortality.

CRedit authorship contribution statement

Eduardo Montalvo: Writing – review & editing, Supervision, Methodology, Investigation, Formal analysis. **Camila Lara-Cazorla:** Writing – original draft, Investigation, Conceptualization. **V. Neira:** Writing – original draft, Conceptualization. **Eder Cano-Pérez:** Writing – review & editing, Formal analysis. **M. Páez:** Investigation. **Byron Fuentes-Flores:** Writing – original draft, Investigation. **Tatiana Guervara-Bahamonde:** Writing – review & editing. **Gabriela Echeverría:**

Garcés: Writing – original draft, Investigation. **E. Castro-Lalangui:** Writing – original draft. **E. Rivadeneira:** Writing – original draft, Investigation. **Diego Álvarez Pico:** Writing – original draft. **Nery Giler Zambrano:** Writing – original draft. **Génesis García-Díaz:** Writing – review & editing, Visualization, Methodology, Investigation.

the preparation or publication of this case report.

Declaration of competing interest

The authors declare that they have no conflict of interest.

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Appendix

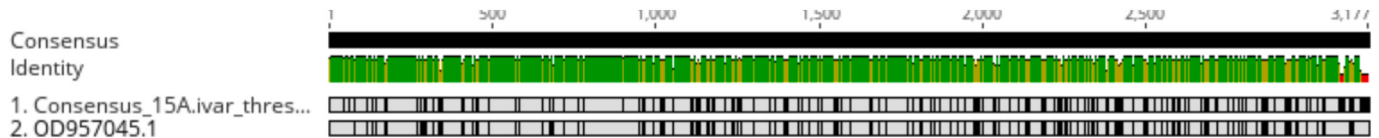
Appendix 1

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Appendix 2

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Appendix 3



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>OD957045.1 *Naegleria fowleri* Karachi NF001 strain

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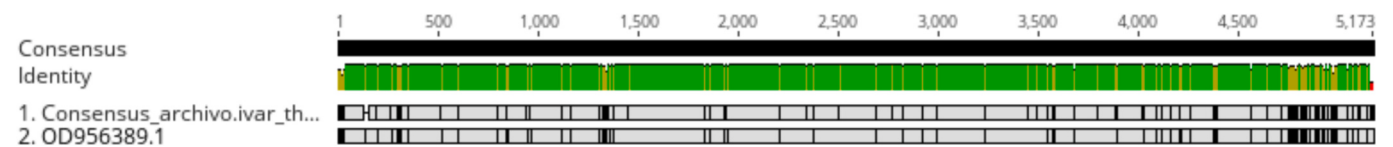
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Appendix 6.1



Appendix 7

QUAST

Quality Assessment Tool for Genome Assemblies by [CAB](#)

10 September 2025, Wednesday, 16:01:16

[View in Icarus contig browser](#)

All statistics are based on contigs of size ≥ 0 bp, unless otherwise noted (e.g., "# contigs (≥ 0 bp)" and "Total length (≥ 0 bp)" include all contigs).

2 442 716 reads

Statistics without reference ☐ **itsdenovo**

# contigs	67
Largest contig	4449
Total length	85 585
Total length (≥ 1000 bp)	77 705
Total length (≥ 10000 bp)	0
Total length (≥ 50000 bp)	0

Reads mapping

Mapped (%)	50.12
Properly paired (%)	39.5
Singletons (%)	1.46
Misjoint mates (%)	5.95
Avg. coverage depth	1458
Coverage $\geq 1\times$ (%)	100

Mismatches

# N's per 100 kbp	0
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[Extended report](#)

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