Journal of Case Reports and Scientific Images

E-ISSN: 2708-0064
P-ISSN: 2708-0056
Impact Factor (RJIF): 5.21
IJCRS 2026; 8(2): 01-04
www.allcasereports.com
Received: 10-12-2025
Accepted: 13-01-2026

Mohammed Benlili
Department of Plastic and Reconstructive Surgery, Mohammed VI University Hospital, Oujda, Morocco

El Abbassi Mohamed
Department of Plastic and Reconstructive Surgery, Mohammed VI University Hospital, Oujda, Morocco

Zakaria Rahali
Department of Plastic and Reconstructive Surgery, Mohammed VI University Hospital, Oujda, Morocco

Ayat Allah Oufkir
Department of Plastic and Reconstructive Surgery, Mohammed VI University Hospital, Oujda, Morocco

Corresponding Author:
Mohammed Benlili
Department of Plastic and Reconstructive Surgery, Mohammed VI University Hospital, Oujda, Morocco

When burns affect the brain: A case of pediatric meningoencephalitis

Mohammed Benlili, El Abbassi Mohamed, Zakaria Rahali and Ayat Allah Oufkir

DOI: https://www.doi.org/10.22271/27080056.2026.v8.i2a.169

Abstract
Severe burn injuries are associated with a high risk of infectious complications due to extensive skin barrier disruption, prolonged intensive care stays, repeated surgical procedures, and burn-induced immunosuppression. While infections are common in burn patients, central nervous system involvement such as meningitis or encephalitis remains rare, particularly in the pediatric population and in the absence of cranial trauma.
We report the case of a 13-year-old child admitted for severe thermal burns involving 37% of the total body surface area. The patient initially presented with stable neurological status and no head injury. During hospitalization, the clinical course was complicated by septic shock and recurrent bacteremia caused by multidrug-resistant nosocomial pathogens, including *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Staphylococcus haemolyticus*, and *Enterobacter hormaechei*. Despite broad-spectrum antibiotic therapy, the patient developed persistent high-grade fever and acute neuropsychiatric manifestations, notably visual hallucinations and transient alteration of consciousness. Neuroimaging was unremarkable; however, cerebrospinal fluid analysis revealed marked neutrophilic pleocytosis, supporting the diagnosis of bacterial meningoencephalitis. Antibiotic therapy was subsequently adapted based on microbiological findings and central nervous system penetration. Following targeted treatment, the patient showed rapid clinical improvement, with resolution of fever and complete recovery of neurological function.
This case highlights a rare but serious complication of severe pediatric burns and emphasizes the diagnostic challenges of central nervous system infections in this context. Persistent fever and unexplained neurological symptoms in burn patients should prompt early consideration of meningoencephalitis, even in the absence of cranial trauma. Early lumbar puncture and appropriate antimicrobial therapy with adequate cerebrospinal fluid penetration are essential to improving outcomes.

Keywords: Severe burns, pediatric burns, meningoencephalitis, central nervous system infection, bacteremia

Introduction
Burn patients with extensive total body surface area (TBSA) involvement are at an elevated risk of infection due to the presence of large open wounds, frequent surgical interventions, prolonged stays in the intensive care unit, and immunosuppression [1]. Common sites of infection in burn patients include the skin, respiratory tract (including the sinuses), eyes, urinary tract, veins (septic thrombophlebitis), and heart (infective endocarditis) [2]. However, meningitis and encephalitis are rare complications [1].
Here, we report the case of a 13-year-old patient who developed meningoencephalitis following severe thermal burns, with a TBSA burn estimated at 37%.

Case report
A 13-year-old child was admitted to our unit for the management of thermal burns caused by flames. The burns included superficial and deep second-degree burns affecting the face, neck, both upper limbs, and both lower limbs, with a total body surface area (TBSA) burn estimated at 37%, with no associated cranial trauma. Upon admission, the child was conscious and hemodynamically stable. The patient received fluid resuscitation based on the Carvajal formula, along with wound cleansing and dressing with silver sulfadiazine, and elevation of the limbs.
On day 3 (D3), the patient underwent the first excision and skin grafting procedure, given the extensive nature of the burns, which could not be treated in a single operation. Antibiotic

therapy with amoxicillin and clavulanic acid was initiated.

On day 5 (D5), the patient developed hemodynamic instability, presenting with hypotension at 76/34 mmHg, oxygen desaturation at 85%, and a fever of 40.5°C. After stabilization, a chest CT scan was performed, revealing aspiration pneumonia with moderate right-sided pleural effusion. Biological tests showed white blood cell count at 25,810, CRP at 211, and albumin at 21. Blood cultures, wound swabs, and a urine culture were performed. Antibiotic therapy was switched to ceftriaxone and ciprofloxacin.

Given the complexity of the case, a multidisciplinary approach was initiated early, involving pediatricians and intensivists to guide management decisions, particularly regarding infection control and neurological monitoring.

The patient remained afebrile for three days. However, on day 9 (D9), he developed a persistent high-grade fever ranging between 39°C and 41°C, resistant to antipyretics. Infectious workup was expanded with a cardiac ultrasound to rule out infective endocarditis, which returned normal. The central venous catheter was replaced and sent for bacteriological analysis. Blood cultures were positive for Pseudomonas aeruginosa, while the urine culture was sterile.

On day 10 (D10), the fever persisted, accompanied by the onset of visual hallucinations that lasted throughout the day. The patient's Glasgow Coma Scale (GCS) was 13/15. A contrast-enhanced brain CT scan was performed, showing no abnormalities. A lumbar puncture revealed cerebrospinal fluid (CSF) with leukocyte count at 1,320/mm³, 90% of which were polymorphonuclear neutrophils (PMNs). Antibiotic therapy was switched to meningitis-dose ceftriaxone, and ciprofloxacin was discontinued.

On day 11 (D11), the patient had a single episode of fever lasting 24 hours. On day 12 (D12), the patient remained febrile at 39.5°C, with no response to antipyretics. Blood cultures were positive for Pseudomonas aeruginosa, Staphylococcus haemolyticus, Acinetobacter baumannii, and Enterobacter hormaechei. We switched the antibiotic to ceftazidime because the pathogens were resistant to ceftriaxone, and their sensitivity to ceftazidime was intermediate.

On day 13 (D13), the fever subsided and the patient achieved apyrexia. There was a notable improvement in the Glasgow Coma Scale, reaching 15/15, and a decrease in CRP levels. Additionally, there was a significant reduction in the patient's hallucinations.

After day 13, the patient remained afebrile, and neurological symptoms further improved. The hallucinations were completely resolved, and the patient regained full cognitive function with no further episodes of confusion or altered mental status. The Glasgow Coma Scale remained stable at 15/15 throughout the following days.

A multidisciplinary meeting was held with the infectiologists and pediatricians to review the progress of the case and refine the management plan. The team recommended continued monitoring of neurological status and further evaluation to ensure full resolution of any potential CNS infection.

Discussion

Infections remain the leading cause of mortality in patients with severe burns, primarily due to several contributing factors such as extensive destruction of the protective skin barrier, the presence of necrotic and edematous tissues that promote microbial colonization, the use of invasive monitoring devices during resuscitation, and burn-induced immunosuppression [3]. Burn patients are considered immunosuppressed due to impaired neutrophil function, T lymphocyte dysregulation, and an imbalance in cytokine production [4]. Post-burn immunosuppression is well documented and was likely a major contributing factor in the case of our patient.

Despite the high incidence of infections in burn patients, bacterial meningitis and encephalitis remain rare complications [1]. A post-mortem study from 1992, examining CNS complications in thermal burns, found that 10.1% of cases developed brain abscesses and identified facial burns and head trauma as potential risk factors for central nervous system infections [5]. However, no recent studies have presented evidence to support these findings.

The primary hypotheses explaining the cerebral spread of infection include septic emboli and disruption of the blood-brain barrier, secondary to the systemic inflammatory response induced by burn injury [6]. The first hypothesis is supported by the presence of documented cutaneous infection and positive blood cultures, indicating hematogenous dissemination. Although no vegetations were seen on transthoracic echocardiography, the possibility of septic emboli cannot be excluded, especially in the context of high-grade bacteremia. These mechanisms may have either facilitated direct microbial invasion or triggered a neuroinflammatory cascade, leading to central nervous system involvement.

CNS infections, although rare in pediatric burn patients without associated cranial trauma, represent a serious complication that is often difficult to diagnose due to subtle neurological signs and overlap with the systemic inflammatory responses commonly seen in burns.

In our patient, persistent visual hallucinations and a transiently reduced Glasgow Coma Scale (13/15) suggested probable meningoencephalitic involvement. These neurological symptoms, occurring alongside systemic infection with neurotropic pathogens and evidence of blood-brain barrier disruption, support the hypothesis of hematogenous CNS invasion. The progressive improvement in mental status and resolution of hallucinations following targeted antibiotic therapy further strengthens this diagnosis.

The decision to perform a lumbar puncture required careful consideration. While neurological changes in burn patients are often attributed to sepsis-associated encephalopathy, certain features in our patient suggested otherwise: persistent high-grade fever despite broad-spectrum antibiotics, new-onset visual hallucinations, and a transient decrease in the Glasgow Coma Scale. Taken together, these findings strongly indicated possible CNS involvement and justified lumbar puncture, which revealed marked pleocytosis consistent with meningoencephalitis. This is an important distinction, since sepsis-associated encephalopathy remains a diagnosis of exclusion

Identification of the causative pathogens plays a key role in understanding the severity and progression of infection in burn patients. In this case, blood cultures were positive for *Pseudomonas aeruginosa*, *Staphylococcus haemolyticus*, *Acinetobacter baumannii*, and *Enterobacter hormaechei*. These organisms are all known nosocomial pathogens, frequently associated with healthcare-related infections, particularly in immunocompromised patients such as those

with severe burns [7]. *Pseudomonas aeruginosa* and *Acinetobacter baumannii* are notorious for their intrinsic resistance mechanisms and ability to survive in hospital environments, while *Staphylococcus haemolyticus* and *Enterobacter hormaechei* are also recognized for their multidrug resistance profiles [7]. According to the same post-mortem study previously cited, *Candida* species, *Staphylococcus aureus*, and *P. aeruginosa* were responsible for 77% of CNS infections [5], highlighting the neurotropic potential of some of the organisms isolated in our patient. The presence of multiple pathogens in this case further illustrates the polymicrobial nature of bloodstream infections in burn patients and underscores the complexity of managing such infections, particularly when the central nervous system is involved.

Severe burns can lead to systemic inflammation and increased permeability of the blood-brain barrier, potentially facilitating the translocation of pathogens into the central nervous system. However, this altered permeability also affects the pharmacokinetics of antimicrobial agents, making their penetration into the cerebrospinal fluid variable and unpredictable. This necessitates careful selection and dosing of antibiotics, particularly in cases of suspected CNS involvement [8].

The choice of antibiotic therapy in burn patients with suspected central nervous system involvement must take into account both the causative pathogens and the ability of selected agents to achieve therapeutic concentrations in the cerebrospinal fluid (CSF). In this case, the antibiotic regimen was subsequently adjusted to ceftazidime and amikacin, based on the microbiological results showing intermediate sensitivity to ceftazidime and full sensitivity to amikacin. Among these, only ceftazidime has adequate CSF penetration in the context of meningeal inflammation [9]. In contrast, amikacin has poor diffusion into the CSF when administered intravenously, limiting its effectiveness for central nervous system infections [10]. The delay in obtaining culture and susceptibility results contributed to a postponed initiation of targeted antimicrobial therapy. Furthermore, CSF cultures remained sterile, most likely due to prior antibiotic administration, which may have led to microbiological sterilization and hindered identification of the responsible organisms.

Given the severity and frequent underdiagnosis of CNS infections in pediatric burn patients, we believe that those with prolonged ICU stays, recurrent or persistent bacteremia, or unexplained neurological changes warrant a more proactive and targeted evaluation. Early identification is essential to improving outcomes. We recommend systematic neurological monitoring, early neuroimaging in cases of unexplained fever or altered mental status, and prompt lumbar puncture when CNS involvement is suspected even in the absence of cranial trauma. Empirical antibiotic therapy should be adapted to ensure adequate CNS penetration, particularly when neurotropic pathogens are suspected. Implementing these strategies may facilitate timely diagnosis and treatment, ultimately reducing morbidity and mortality.

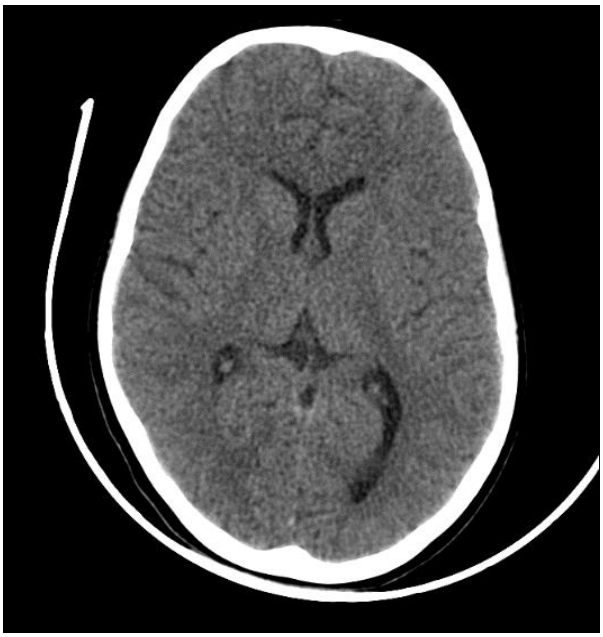

Fig 1: Contrast-enhanced brain CT scan performed to assess cerebral involvement shows no acute intracranial abnormalities, with preserved gray-white matter differentiation and symmetrical ventricles.

Patient Consent

Written informed consent was obtained from the patient's legal guardian for publication of this case report and any accompanying clinical data. All identifying information has been anonymized to ensure patient confidentiality, in accordance with ethical standards and journal requirements.

Conclusions

This case highlights a rare but serious complication of severe burns: bacterial meningoencephalitis in a pediatric patient without associated cranial trauma. Despite the high frequency of infections in burn patients, central nervous system involvement remains exceptional, particularly in children. The presence of multidrug-resistant pathogens, immunosuppression, and potential disruption of the blood-brain barrier likely contributed to the development of this infection. Diagnostic delays are common, due to the nonspecific presentation and the limitations of conventional microbiological testing after prior antibiotic administration.

References

1. Segal N, Polcz VE, McKean JA, Kariyawasam V, Carson JS, Fahy BG. Pseudomonal meningoencephalitis with ventriculitis secondary to bacteremia in a burn patient: a novel case. J Burn Care Res. 2021;42(4):832-835. doi:10.1093/jbcr/irab016.
2. Calvano TP, Hospenthal DR, Renz EM, Wolf SE, Murray CK. Central nervous system infections in patients with severe burns. Burns. 2010;36(5):688-691. doi:10.1016/j.burns.2009.08.004.
3. Ansermino M, Hemsley C. ABC of burns: intensive care management and control of infection. BMJ. 2004;329:221-223.
4. Sharma BR, Harish D, Singh VP, Bangar S. Septicemia as a cause of death in burns: an autopsy study. Burns. 2006;32:545-549.
5. Winkelman M, Galloway P. Central nervous system complications of thermal burns: a postmortem study of 139 patients. Medicine (Baltimore). 1992;71:271-283.
6. Flierl MA, Stahel PF, Touban BM, Beauchamp KM, Morgan SJ, Smith WR, Ipaktchi KR. Bench-to-bedside review: burn-induced cerebral inflammation a neglected entity? Crit Care. 2009;13(3):215. doi:10.1186/cc7794.
7. Church D, Elsayed S, Reid O, Winston B, Lindsay R. Burn wound infections. Clin Microbiol Rev. 2006;19(2):403-434. doi:10.1128/CMR.19.2.403-434.2006.
8. Sharma BR. Infection in patients with severe burns: causes and prevention. J Burn Care Res. 2010;31(1):27-35.
9. Fong IW, Tomkins KB. Penetration of ceftazidime into the cerebrospinal fluid of patients with and without evidence of meningeal inflammation. Antimicrob Agents Chemother. 1984;26(1):115-116.
10. Briedis DJ, Robson HG. Cerebrospinal fluid penetration of amikacin. Antimicrob Agents Chemother. 1978;13(6):1042-1043.

How to Cite This Article
Benlili M, Mohamed EA, Rahali Z, Oufkir AA. When burns affect the brain: A case of pediatric meningoencephalitis. Journal of Case Reports and Scientific Images. 2026;8(2):01-04.