


Intensive Care Unit Management of Status Epilepticus with Central Nervous System Infection: A Case Report

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ABSTRACT

Introduction: Status epilepticus (SE) is a critical neurological emergency characterized by prolonged seizure activity that poses significant neurological risks and death, requiring immediate and effective management to prevent morbidity and mortality. SE can result from a variety of causes, including primary neurological disorders, metabolic disturbances, and iatrogenic factors. In the ICU, drug toxicity and electrolyte imbalances are common triggers, accounting for over 30%–35% of seizures. Understanding the underlying causes, such as central nervous system infections or autoimmune encephalitis, is crucial for targeted treatment and improving patient outcomes. **Case:** A 21-year-old male with SE complicated by a central nervous system (CNS) infection was managed in the intensive care unit (ICU). Seizure control was achieved using continuous infusions of midazolam and phenytoin. The treatment strategy also included supportive care, including mechanical ventilation and hemodynamic stabilization with norepinephrine. The patient's condition improved significantly. **Discussion:** This case demonstrates that favorable clinical outcomes in SE complicated by a CNS infection rely heavily on continuous sedation, the selection of antibiotics with adequate CNS penetration (such as linezolid), and meticulous hemodynamic monitoring in the intensive care setting. **Conclusion:** This case highlights the importance of early diagnosis, targeted treatment, and interdisciplinary collaboration.

Keywords: Case report, central nervous system infection, intensive care unit, seizure, status epilepticus.

ABSTRAK

Pendahuluan: Status epileptikus (SE) adalah keadaan darurat neurologis kritis ditandai dengan aktivitas kejang berkepanjangan yang berhubungan dengan risiko neurologi yang signifikan dan kematian; keadaan ini membutuhkan penanganan segera dan efektif untuk mencegah morbiditas dan mortalitas. SE dapat ditimbulkan oleh berbagai penyebab, termasuk gangguan neurologi primer, gangguan metabolik, dan faktor iatrogenik. Di unit perawatan intensif (ICU), toksisitas obat dan gangguan keseimbangan elektrolit merupakan pemicu yang umum ditemukan, yang merupakan 30%–35% pemicu kejang. Memahami penyebab yang mendasarinya, seperti infeksi sistem saraf pusat atau ensefalitis autoimun, adalah krusial untuk pengobatan yang ditargetkan dan memperbaiki luaran pasien. **Kasus:** Seorang pria berusia 21 tahun dengan SE dan komplikasi infeksi sistem saraf pusat (SSP) dirawat di ICU. Kontrol kejang dicapai dengan infus *midazolam* dan *phenytoin* secara kontinu. Strategi perawatan juga mencakup perawatan suportif dengan ventilasi mekanik dan stabilisasi hemodinamik menggunakan *norepinephrine*. Kondisi pasien membaik dengan signifikan. **Pembahasan:** Kasus ini menunjukkan bahwa luaran klinis yang baik pada SE dengan komplikasi infeksi SSP sangat bergantung pada sedasi berkelanjutan, pemilihan antibiotik dengan penetrasi SSP yang optimal (seperti linezolid), serta pemantauan hemodinamik di ruang intensif. **Simpulan:** Kasus ini menyoroti pentingnya diagnosis dini, pengobatan yang ditargetkan, dan kolaborasi interdisiplin. **Novi Dwi Akhsaniati, Bowo Adiyanto. Tata Laksana Intensif Status Epileptikus dengan Infeksi Susunan Saraf Pusat: Laporan Kasus.**

Kata Kunci: Laporan kasus, infeksi sistem saraf pusat, unit perawatan intensif, kejang, status epileptikus.

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INTRODUCTION

Status epilepticus (SE) is a critical neurological emergency characterized by prolonged seizure activity that poses significant risks to life and neurological function. Annually, the United States reports between 102,000 and 152,000 cases of SE, with approximately 55,000 resulting in fatalities.[1] The condition arises when mechanisms responsible for terminating seizures fail, or when pathological mechanisms initiate sustained seizure activity. This can lead to long-term consequences such as neuronal death and structural changes in neural networks, depending on the type and duration of the seizures.[2] SE is categorized based on clinical manifestations, including the presence or absence of motor symptoms and consciousness impairment,

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leading to classifications such as convulsive SE and non-convulsive SE (NCSE).[3]

Management of SE, particularly in the intensive care unit (ICU) setting, requires a comprehensive understanding of its etiology, diagnosis, and treatment strategies. SE can result from a variety of causes, including primary neurological disorders, metabolic disturbances, and iatrogenic factors. In the ICU, drug toxicity and electrolyte imbalances are common triggers, accounting for over 30%–35% of seizures, which can exacerbate the patient's condition.[4]

A key aspect of managing SE involves rapid intervention to prevent long-term neurological damage and systemic complications. Initial treatment typically involves benzodiazepines, which are preferred for their rapid action and efficacy in terminating seizures.[5] If first-line treatments are ineffective, second-line therapies such as phenytoin or fosphenytoin are employed. In refractory cases, where seizures persist beyond 60 minutes despite appropriate treatment, intensive care management with anesthetic agents like midazolam or propofol may be necessary.[6] Understanding the underlying causes, such as central nervous system infections or autoimmune encephalitis, is crucial for targeted treatment and improving patient outcomes.[7]

The case report aims to explore the causes, diagnostic approaches, and management strategies for SE in the ICU, and to assess the prognosis of patients treated for SE in this critical care environment.

CASE

Patient Background and Initial Presentation

A 21-year-old male with a six-year history of epilepsy, was admitted to the intensive care unit (ICU) due to a decrease in consciousness and continuous generalized tonic-clonic seizures lasting over five minutes. He had been experiencing frequent auditory hallucinations and was under regular psychiatric and neurological care. Prior to admission, the patient exhibited weakness, drowsiness, and intermittent fever, but no respiratory complaints, such as cough or shortness of breath. These episodes prompted his family to seek emergency care, resulting in his transfer to RSUP Dr. Sardjito after an initial 11-days ICU stay at a psychiatric hospital.

Clinical Assessment and Initial Management

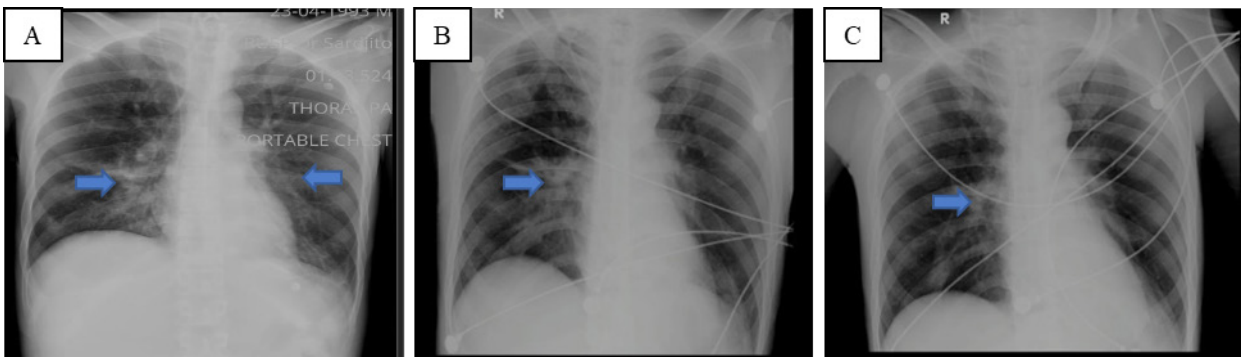
Upon arrival in the ICU, GCS E4V4Vt vital signs included blood pressure of 123/78 mmHg, maintained with norepinephrine, and a heart rate of 110 beats per minute., and a heart rate of 110 beats per minute. The patient was intubated and sedated, placed on mechanical ventilation in PSIMV mode with PEEP of 5 cmH_2O, FiO2 of 70%, and a respiratory rate of 24 breaths per minute. The patient was sedated with thiopental 4 mg/kg IV bolus, followed by 1 mg/kg repeated dose every 2–3 minutes until the seizure stopped. It was then maintained by continuous infusion at a rate of 5 mg/kg/min. Physical examination revealed neither meningeal signs nor nuchal rigidity. Initial laboratory tests showed anemia, elevated liver enzymes, and signs of bilateral pneumonia . The primary assessment included status epilepticus, sepsis, septic shock, and anemia.

Diagnostic Workup

The diagnostic workup involved a comprehensive approach to identify the underlying causes and to assess the patient's condition. Cerebrospinal fluid (CSF) analysis was a critical component used to detect markers of bacterial infection. The CSF culture identified *Staphylococcus haemolyticus*, sensitive to linezolid, indicating a central nervous system infection. Laboratory tests revealed anemia, elevated liver enzymes, and signs of bilateral pneumonia, contributing to the overall assessment of the patient's condition. Imaging studies, including a head computed tomography (CT) scan and chest x-ray (**Figure**), were utilized to evaluate intracranial pressure and identify any contraindications to lumbar puncture . The head CT scan showed cerebral atrophy and *ex vacuo* ventriculomegaly (Evans index 0.34), with no apparent abnormalities in the neurocranium or viscerocranium. Chest x-ray revealed bilateral pneumonia, normal heart size, and the presence of a central venous catheter (CVC) inserted in the right jugular vein with its distal tip directed caudally towards the superior vena cava. On follow-up, a subsequent chest x-ray indicated persistent bilateral pneumonia. The head CT scan was specifically assessed for intracranial pressure, which could indicate a risk of cerebral herniation if a lumbar puncture were performed.

Treatment Strategy

The management of status epilepticus in the context of a central nervous system infection in the ICU involved a multifaceted approach,



*Documentation by Novi Dwi Akhsaniati and Bowo Adiyanto.

Figure. Posteroanterior chest x-ray showing signs of minimal pneumonia (blue arrow). A) Day 0, B) day 4, and C) day 8.


focusing on seizure control, infection management, and supportive care. The patient was initially sedated and intubated to secure the airway and facilitate mechanical ventilation. For seizure control, a continuous infusion of midazolam was administered, with the dose titrated to achieve adequate sedation and seizure suppression. Initially, a loading dose of 0.2 mg/kg is given intravenously as a bolus, followed by continuous infusion starting at 0.05–0.1 mg/kg/hour. The dose is then titrated upwards as needed, typically in increments of 0.05–0.1 mg/kg/hour every 15–30 minutes, until seizure control is achieved or limited by side effects. The maximum recommended infusion rate is usually 0.4 mg/kg/hour, though higher doses may be used in refractory cases under close monitoring. Phenytoin was given intravenously at a dose of 100 mg every 8 hours to stabilize neuronal membranes and prevent further seizures.

Antibiotic therapy was initiated to address bacterial infection identified in the cerebrospinal fluid. Intravenous meropenem was given at a dose of 1 gram every 8 hours, targeting a broad spectrum of potential pathogens. Additionally, levofloxacin was administered at 750 mg per 24 hours, based on culture sensitivities. Once *Staphylococcus haemolyticus* was identified as the causative organism, linezolid 600 mg daily was added to the regimen due to its excellent CNS penetration and efficacy against this pathogen.[8]

Supportive care in the ICU included maintaining hemodynamic stability with norepinephrine, initially titrated at 0.1 mcg/kg/min to support blood pressure. Omeprazole 40 mg daily was given intravenously for stress ulcer prophylaxis, and paracetamol 1 gram every 8 hours was used for fever management when the temperature exceeded 37.5°C. Nutritional support was provided via nasogastric tube feeding, ensuring adequate caloric intake and minimizing the risk of aspiration.

Regular neurological assessments were conducted to evaluate the patient's response to therapy and adjust medications as needed. The use of continuous EEG monitoring, if available, would have provided additional insights into seizure activity and the effectiveness of antiepileptic treatment. The interdisciplinary team, including neurologists, intensivists, and infectious disease specialists, collaborated closely to optimize the patient's care and promptly address any complications.

Progress and Follow-Up

The patient's initial presentation featured persistent incomprehensible vocalizations despite spontaneous eye opening (E4V4Vt), suggesting intact brainstem reflexes with impaired cortical processing. Continuous propofol-midazolam infusions targeted seizure suppression while maintaining RASS score of -4 to -5 regarding to sedation depth. Paracetamol administration (3 g/24 h) addressed febrile episodes (peak 38.9°C), potentially exacerbating neuronal excitability. Ventilatory management emphasized lung-protective strategies, with an initial PEEP of 8 cmH2O to treat suspected neurogenic pulmonary edema, though a chest x-ray remained unavailable for confirmation.

Emergence of complex oculomotor signs - bidirectional nystagmus with rotational components - raised concerns for brainstem irritation or antiseizure medication toxicity. Concurrent orolingual tremors (frequency 4–6 Hz) suggested basal ganglia involvement, potentially reflecting midazolam accumulation, given the prolonged half-life of its active metabolite, α-hydroxymidazolam, in critical illness. Despite these findings, the patient demonstrated purposeful movement to pain (M6), contradicting pure brainstem dysfunction patterns .

Sputum culture on day 7 revealed *Pseudomonas aeruginosa* susceptible to amikacin (MIC 4 µg/mL), prompting targeted Gram-negative coverage while maintaining meropenem for possible ESBL producers. On day 10, CSF culture isolated *Staphylococcus haemolyticus* (oxacillin MIC > 256 µg/mL), necessitating linezolid to achieve adequate CNS penetration (CSF:serum ratio 0.6–0.8). Therapeutic drug monitoring guided amikacin dosing (peak 35 µg/mL, trough 2.1 µg/mL) to minimize nephrotoxicity risk.

By day 8, the patient exhibited visual tracking and reliable grimacing to noxious stimuli, though aphasia persisted from prolonged intubation. Transition to oral antiepileptics required careful overlap - valproic acid loading (20 mg/kg) achieved therapeutic levels (85 µg/mL) within 48 hours, permitting phenytoin tapering. Myoclonic jerks on day 12 (1–2/sec, predominantly right deltoid) were managed with clonazepam up-titration, reflecting resolved cortical hyperexcitability transitioning to brainstem-mediated movements.

Methylprednisolone (1 g/day) addressed suspected inflammatory-mediated ARDS, with subsequent FiO2 reduction from 0.6 to 0.3 correlating with resolution of pneumonia. Ursodeoxycholic acid prevented cholestasis from prolonged midazolam infusion, maintaining direct bilirubin < 2 mg/dL. Subcutaneous heparin (anti-Xa 0.3 IU/mL) prevented DVT without hemorrhagic conversion despite seizure-related endothelial injury.

The STESS score of 2 implied a 85% 30-day survival probability, which aligned with the observed GCS improvement. However, persistent myoclonus suggested residual postictal cortical dysfunction, necessitating outpatient qEEG monitoring. The planned antiepileptic drug (AED) duration of 12–24 months in this case reflects evidence-based guidelines for acute symptomatic seizures (ASS) with structural brain injury from central nervous system (CNS) infections.[9–11] Acute symptomatic seizures (ASS) occurring within 7 days of acute brain insults like infections carry a lower long-term epilepsy risk compared to unprovoked seizures (cumulative 12-month recurrence risk: 10.7% vs 65%).[12] However, combined infectious-structural etiologies significantly increase relapse risk (OR 11.1),[12] justifying extended prophylaxis. Levetiracetam is prioritized due to its favorable pharmacokinetics in critical illness, minimal drug interactions with antibiotics,[13,14] and adherence advantages over enzyme-inducing AEDs like phenytoin.[15] The cited 32% relative risk reduction (RRR 0.68) for recurrence with adherence aligns with studies showing levetiracetam's effectiveness in preventing seizure relapse when maintained at therapeutic levels,[13,15] particularly important given non-adherence increases recurrence 4.28-fold in comorbid patients.[15] This duration balances recurrence prevention (median AED course of 3.4 months in ASS)[12]


against overtreatment risks, as prolonged AED use in resolved structural lesions provides no added benefit.[15] Respiratory follow-up assessment includes diffusing capacity of the lung for carbon monoxide (DLCO) testing to assess aspiration-induced parenchymal injury. Follow-up of the patient's condition is shown in the **Table**.

DISCUSSION

Status epilepticus (SE) is a life-threatening condition characterized by prolonged seizure activity that can lead to significant morbidity and mortality if not promptly managed.[2,16] In the intensive care unit (ICU), SE often results from various etiologies, including central nervous system infections, metabolic disturbances, and drug toxicity.[4,17,18] The case presented the complexity of diagnosing and managing SE, particularly when it is associated with a CNS infection. The diagnostic process involved cerebrospinal fluid (CSF) analysis, which revealed *Staphylococcus haemolyticus* infection.[19,20]

Early lumbar puncture is crucial for diagnosing CNS infections, but it must be performed with caution in patients with suspected increased intracranial pressure.[21,22] In this case, the presence of bacterial markers in the CSF and the growth of *Staphylococcus haemolyticus* were pivotal in confirming the diagnosis of a CNS infection. This finding underscores the importance of timely and accurate diagnostic procedures in guiding effective treatment strategies.[20,23]

The management of SE involves rapid intervention with antiepileptic drugs and targeted antibiotic therapy. In this case, midazolam and phenytoin were used to control seizures, while linezolid was chosen for its efficacy against the identified bacterial pathogen.[5,24] The choice of linezolid was particularly appropriate given its excellent penetration into the CNS, making it effective for treating infections like MRSA in the brain.[23] The patient's gradual improvement, as evidenced by reduced seizure frequency and improved consciousness, highlights the efficacy of this tailored treatment approach.

SE can lead to various systemic complications, including metabolic disturbances, respiratory failure, and cardiovascular instability.[25] This case required mechanical ventilation and hemodynamic support with norepinephrine, illustrating the need for comprehensive supportive care in the ICU. The status epilepticus severity score (STESS) provided insights into the patient's prognosis; a score of 2 indicates a lower risk of mortality.[26–31] This lower score suggested a better prognosis,

Table. Follow-up of the patient's condition.

Day	GCS	Key Medications	Seizure Activity	Clinical Events/Interventions
1	E4M4Vt	Midazolam (continuous infusion), propofol (continuous infusion), norepinephrine (0.1 mcg/kg/min IV), meropenem (1 g IV q8h), levofloxacin (750 mg IV q24h), phenytoin (100 mg IV q8h), omeprazole (40 mg IV daily), paracetamol (1g IV q8h PRN)	2 generalized tonic-clonic seizures (> 1 minute each)	Intubation, initiation of dual sedative therapy, vasopressor support, empiric antibiotic coverage, antiepileptic therapy initiation
2	E4M4Vt	Midazolam infusion continued, ketamine infusion added, methylprednisolone (250 mg IV q6h), Ursodeoxycholic acid (250 mg PO q8h), heparin (5,000 U SC q24h)	1 generalized tonic-clonic seizure (< 1 minute)	Discontinuation of propofol, weaning of norepinephrine, initiation of steroid therapy and DVT prophylaxis
4–7	E4M6Vt	Midazolam + ketamine infusions continued, Nebulized albuterol/budesonide (1:1 ratio q8h), Continued meropenem/levofloxacin/phenytoin/methylprednisolone	4 focal seizures (shoulder/ eye involvement)	Horizontal/vertical/rotatory nystagmus observed, orolingual tremor noted, ventilator settings reduced
8–9	E4M6Vt	Midazolam infusion continued, amikacin (1 g IV q24h), valproic acid (500 mg PO BID), clonazepam (2 mg PO BID), levetiracetam (500 mg PO BID), N-acetylcysteine (200 mg PO q8h)	2 focal seizures (shoulder/ eye involvement)	Ketamine discontinued, oral antiepileptic transition initiated, sputum culture-guided antibiotic adjustment
10–11	E4M6Vt	Linezolid (600 mg IV q24h) added to regimen	2 focal seizures (shoulder/ eye involvement)	CSF culture-directed antimicrobial therapy initiated (*Staphylococcus haemolyticus* coverage)
12	E4M6Vt	Continued antibiotic/antiepileptic therapy	Myoclonic-like movements (focal seizure resolution)	Neurological recovery milestones achieved, sustained cardiovascular stability, progressive ventilator weaning

Prognostic Notes:
STESS Score: 2 (Generalized seizure [+1], stupor at presentation [+1], age < 65, no prior seizures)
Trajectory: Transition from status epilepticus → focal seizures → myoclonic movements → seizure resolution
Key Recovery Markers: Improved GCS (E4M4Vt → E4M6Vt), reduced FiO2 requirements, negative pneumonia imaging

Abbreviations: GCS: Glasgow coma scale; IV: Intravena; PO: Per oral; DVT: Deep vein thrombosis; BID: Bis in die: CSF: Cerebrospinal fluid.

which aligned with the patient's gradual improvement. The STESS takes into account factors such as age, seizure history, seizure type, and level of consciousness, providing a quick, practical tool for risk stratification. In this case, the score helped guide treatment intensity and recovery expectations, demonstrating its utility for clinical decision-making and resource allocation in the ICU setting.

Further research is needed to explore the optimal management strategies for SE associated with various etiologies, including CNS infections. Studies evaluating the efficacy and safety of various antiepileptic and antibiotic regimens could provide valuable insights to improve patient care. Additionally, the development of predictive tools like STESS can aid clinicians in assessing prognosis and tailoring interventions to individual patient needs.[26–31] Overall, continued advances in understanding and treating SE will enhance the ability to manage this complex and challenging condition effectively.

CONCLUSION

This case emphasizes the importance of early intervention, interdisciplinary collaboration, and continuous monitoring to optimize patient outcomes in severe neurological emergencies like SE.

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