Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 14s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

A Rare Presentation Of Sepsis Associated Metabolic Encephalopathy With Superadded Uremic Encephalopathy – A Case Report

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Cite this paper as: Sachdev Shobha Pravin, Ashwin Kumar Azhagarasan, Murugan Gopalakrishnan, Vishal Ramnath Chanan, (2025) A Rare Presentation Of Sepsis Associated Metabolic Encephalopathy With Superadded Uremic Encephalopathy – A Case Report. *Journal of Neonatal Surgery*, 14 (14s), 870-872.

ABSTRACT

Background: Sepsis-associated metabolic encephalopathy is seen in critically ill patients. Uremia worsens sepsis, causing significant cognitive and mental status changes.

Objective: To highlight the imaging patterns of septic and uremic encephalopathy.

Methods: The patient was brought to the emergency with decreased responsiveness for 2 days. Comprehensive lab evaluations and imaging studies were conducted.

Results: Imaging diagnosis was made based on the characteristic brain parenchymal changes in correlation with clinical and lab parameters. On follow up post treatment changes revealed resolution of brain parenchymal changes and improved patient's clinical status.

Discussion: Imaging in septic and metabolic encephalopathy reveals bilateral symmetrical and characteristic changes such as diffuse white matter hyperintensities in brain stem in septic and the Lentiform fork sign in uremic encephalopathy.

Conclusion: Clinicians should correlate lab parameters and imaging patterns with altered mental status to optimize treatment.

Keywords: *Metabolic encephalopathy; Sepsis-associated encephalopathy; Uremic encephalopathy; Lentiform fork sign; White matter disease; Central nervous system; Procalcitonin.*

1. INTRODUCTION

This case underscores the importance of imaging findings in association with clinical and lab parameters in encephalopathic patients who present with clinical features of sepsis associated with co-added metabolic abnormalities.

Initially, the patient displayed significant lethargy and drowsiness. Imaging performed confirmed changes indicative of septic and uremic encephalopathy with findings as mentioned in the methods above. After receiving treatment with broad

spectrum antibiotic and supportive measures, his BUN and procalcitonin levels decreased, leading to a marked improvement in neurological symptoms.

2. METHODS

A 71-year-old male was brought to casualty with decreased responsiveness for 2 days. The

patient had a history of multiple episodes of vomiting on and off for the past one month

associated with decreased appetite. No history of trauma/ numbness or tingling sensation in lower limbs. No known comorbidities.

On admission, Glasgow coma score was E4V1M5 (10/15).

Blood reports revealed leucocytosis (WBC: 16000), raised procalcitonin (2.37 ng/ml), C-

reactive protein of 32.0 mg/dl, increased blood urea (serum urea: 92 mg/dl) and serum

creatinine (1.3 mg/dl).

MRI brain showed multiple punctate and confluent T2 FLAIR hyperintensities in bilateral

fronto-parietal and occipital subcortical and deep white matter and periventricular regions.

Symmetrical T2 FLAIR hyperintensities were also seen involving bilateral thalami, external

capsules, head of caudate nucleus, brainstem and dentate nucleus of cerebellum (See Figure 1 and Figure 2). However, no abnormal foci of diffusion restriction were seen.

3. RESULTS

Initially, the patient displayed significant lethargy and drowsiness. Imaging performed confirmed changes indicative of septic and uremic encephalopathy with findings as mentioned in the methods above. After receiving treatment with broad spectrum antibiotic and supportive measures, his BUN and procalcitonin levels decreased, leading to a marked improvement in neurological symptoms.

4. DISCUSSION

Sepsis associated encephalopathy is characterized by diffuse brain dysfunction secondary to infection elsewhere in the body without overt CNS infection. [(1)] Uremic encephalopathy presents with symptoms such as lethargy and confusion, progressing to seizures or coma with chronicity. [(2)] The impact of uremic toxins is observed in the basal ganglia and can also affect cortical, subcortical regions and white matter. Imaging reveals bilateral changes, with the “Lentiform Fork Sign” as a significant indicator (Figure 2). This sign appears on T2-weighted and FLAIR MRI images as hyperintensities surrounding the medial and lateral aspects of the lentiform nuclei, defining their boundaries. [(2)] This distinctive imaging feature is notably observed in diabetic patients. [(3)] The uniqueness of this case lies in the presence of septic component in metabolic encephalopathy. Raised procalcitonin levels primarily indicate sepsis among other causes. The presence of symmetrical lesions indicated a metabolic origin, helping to rule out traumatic causes. Septic-associated encephalopathy is characterised by diffuse white matter hyperintensities on T2 and FLAIR sequences in bilateral periventricular and deep white matter and brainstem (Figure 3). [(4)] In this case, we observed similar findings consistent with septic encephalopathy. Post treatment follow up imaging showed reduction in the white matter hyperintensities as well as those in brainstem and cerebellum (Figure 4 and Figure 5). Radiological assessments are vital for guiding treatment strategies and monitoring the progression of the disease. [(5)] Bilateral basal ganglia and brainstem hyperintensities are also seen in Wernicke’s encephalopathy (which also shows involvement of mammillary bodies and periaqueductal grey matter), Wilson’s disease (which shows intrinsic T1 hyperintensity) and osmotic demyelination (which has extrapontine involvement as well).

Sources of Funding: The authors received no specific funding for this work.

Conflict(s) of Interest and any Disclosure(s): The authors declare no conflicts of interest.

Declaration of patient consent:

The authors declare that informed consent was obtained from the patient or their legal representative for the publication of this case report. The patient has been informed about the nature of the report, its purpose, and the potential for identification through the details provided. All personal identifiers have been removed to ensure confidentiality.

REFERENCES

[1] Mahoney CA, Sarnacki P, Arieff AI. Uremic encephalopathy: role of brain energy metabolism. *Am J Physiol.* 1984;247(3 Pt 2).

[2] Olano C, Akram S, Bhatt H. Uremic encephalopathy. 2024. [PubMed].

[3] M. D. G, et al. Metabolic encephalopathy: a review. *J Neurol.* 2021.

[4] Gofton T, Young G. Sepsis-associated encephalopathy. *Nat Rev Neurol*. 2012;8:557–66. https://doi.org/10.1038/nrneurol.2012.183.

[5] Kumar G, Goyal MK. Lentiform fork sign: a unique MRI picture. Is metabolic acidosis responsible? *Clin Neurol Neurosurg.* 2010;112(9):805–12. doi:10.1016/j.clineuro.2010.06.006. [PubMed].