CASE REPORT

Neurolisteriosis in an immunocompromised patient

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SUMMARY

Neurolisteriosis is a rare but severe manifestation of Listeria monocytogenes infection, particularly affecting immunocompromised individuals. We present the case of a 33 year-old female with systemic lupus erythematosus (SLE) who developed left-sided hemiparesis with pneumonia. Despite initial treatment with antibiotics and corticosteroids, her condition worsened, requiring neurosurgical intervention. Brain biopsy revealed Listeria monocytogenes, confirming the diagnosis of neurolisteriosis. She received prolonged antibiotic therapy and supportive care, leading to clinical improvement. This case highlights the challenges in diagnosing and managing neurolisteriosis in immunocompromised patients and underscores the importance of early recognition and intervention for optimal outcomes.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease in which the immune system attacks its own tissues, causing widespread inflammation and tissue damage in the affected organs. A dysregulated immune response, the severity of the disease, and immunomodulatory agents used significantly increase the susceptibility to infection in individuals with SLE.

CASE PRESENTATION

We describe a 33 years old lady with underlying SLE with Lupus Nephritis. She presented with worsening left-sided hemiparesis for 3 days, associated with fever and productive cough. Notably, her prednisolone was increased to 1mg/kg/day for a recent lupus nephritis flare, which increases her risk for opportunistic infection.

On examination, her Glasgow Coma Scale (GCS) was 15/15 with equal and reactive pupils. She was febrile but saturating well under room air. Neurologically, she had evidence of left hemiparesis with Medical Research Counsil (MRC) power of 4/5 over the upper limb and 3/5 over the lower limb, associated with increased muscle tone and hyperreflexia over the left upper and lower limbs, which progressed to dense hemiplegia within 2 weeks. Her sensation was intact throughout. Other systemic examination was unremarkable. She had no clinical features suggestive of active SLE.

Upon presentation, laboratory examination revealed raised WBC (13.5 x 10^3/UL) and CRP (23.2mg/L) with mild renal derangement (creatinine 124umol/L) .Other blood parameters were unremarkable. A contrasted CT brain revealed right frontal and posterior parietal enhancing hypodense lesions (4.7 × 2.3 x 5cm), causing mass effect to adjacent sulci with another small peripherally enhancing lesion seen at the right frontal region, 0.8 x 1cm.There was no abnormal leptomeningeal enhancement. Her MRI brain revealed multiple irregular peripherally enhancing intra-axial lesions with vasogenic oedema and mass effect.

She was started on intravenous (IV) Ceftriaxone 2gm BD for 4 days, then escalated to IV Meropenem 2gm TDS due to persistent fever. She was also started on dexamethasone to reduce the mass effect. Lumbar puncture was contraindicated in view of serial imaging showing lesions causing mass effect.

She was intubated for airway protection after 2 weeks of admission in view of drop in GCS (E2V1M1). A repeated CT brain showed worsening white matter oedema at right frontal and posterior parietal lobe, with worsening hypodensity at right basal ganglia with increasing mass effect which have contributed to her drop in GCS. She had no documented seizure.

A right decompressive craniectomy, image-guided stereotactic biopsy and fasciduroplasty was done on her. Intra-operatively, the dura was thinned out with clear, high-pressure cerebrospinal fluid (CSF). Brain biopsy HPE revealed rod-shaped bacterial colonies consistent with Listeria monocytogenes, which was also identified from the CSF cultures and blood cultures done via conventional method.

She was given IV Ampicillin 2gm 4 hourly for a total of 6 weeks. Dexamethasone was tapered off and maintenance IV Hydrocortisone was given to control her SLE. A repeated CT brain after 6 weeks of antibiotic showed resolving right frontal and posterior parietal lesions. Upon discharge, she was bedbound with improving left hemiparesis (MRC power 2/5) and full GCS level.

A repeated CT brain 6 months later showed reduced hypodensity over right frontal and parietal vasogenic oedema with no significant enhancement suggestive of residual vasogenic oedema.

DISCUSSION

Neurolisteriosis is an uncommon but severe manifestation of Listeriosis, caused by the bacterium Listeria monocytogenes.1 Listeria monocytogenes is prevalent in the environment, with a widespread presence in soil, water and the digestive tracts

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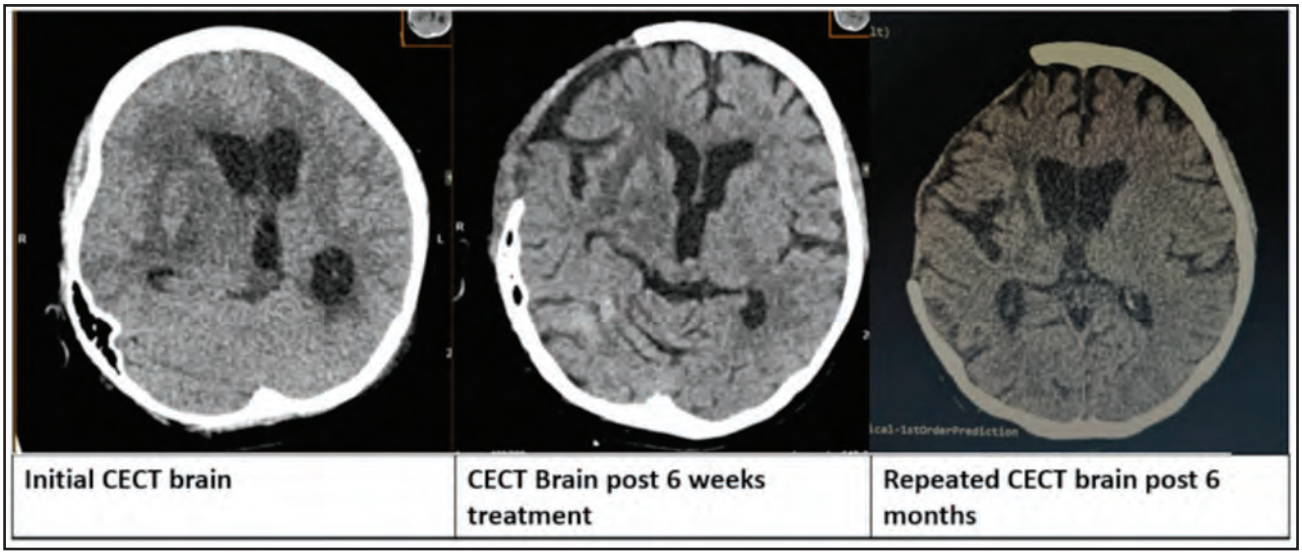

Fig. 1: Serial Contrasted Enhanced CT Brain.

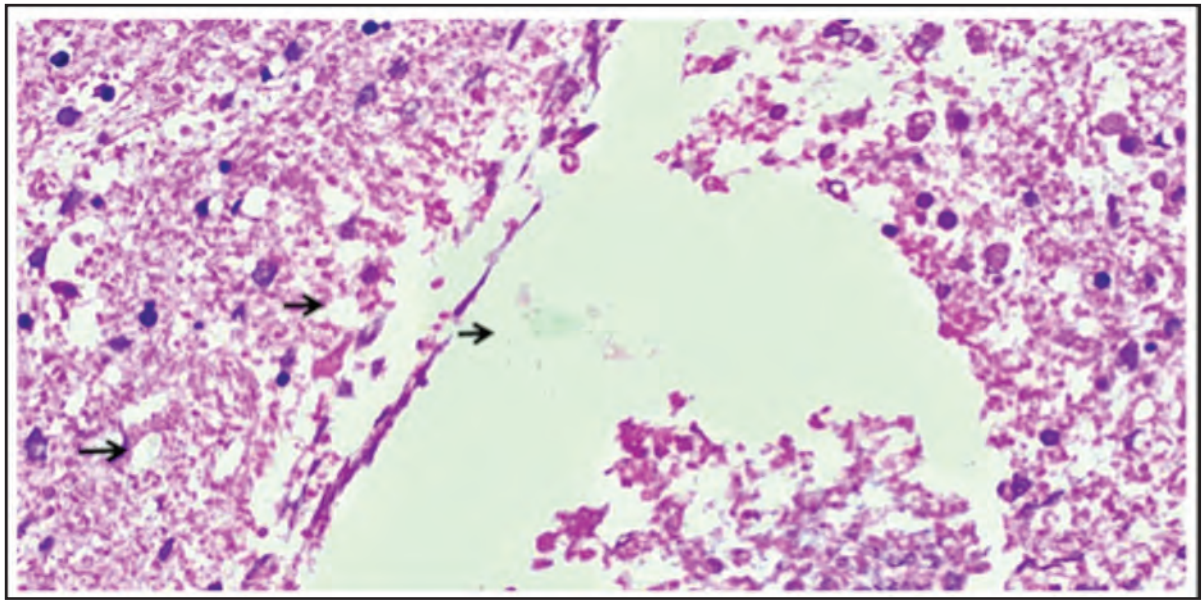

Fig. 2: Multiple foci of rod-shaped bacterial colonies are seen within the glial tissue (black arrow) from the brain tissue biopsy.(Images provided by Pathologist- in- charge Dr Ang Yee Ping (Anatomic Pathologist)).

of animals. Contamination of vegetables can occur through contact with contaminated soil or the application of manure as a fertilizer.[1] Moreover, ready-to-eat foods are susceptible to contamination at the processing stage, and the bacteria have the potential to proliferate to hazardous levels during both distribution and storage.[1]

In contrast to numerous other prevalent foodborne bacteria, L. monocytogenes exhibits the ability to endure and replicate at the low temperatures typically present in refrigerators.[1] Consuming contaminated food with high numbers of L. monocytogenes is the main route of infection.[1] There are two types of infection: non-invasive, like febrile enteritis, and the invasive type that usually affects immunocompromised patients, pregnant women and their newborns.[2] While listeriosis typically manifests as a foodborne illness with symptoms like fever, muscle aches, and gastrointestinal issues, neurolisteriosis specifically involves the central nervous system, leading to neurological symptoms.[3]

Neurolisteriosis typically manifests in three forms: meningitis/meningoencephalitis, rhombencephalitis, and cerebritis, often progressing to the development of cerebral abscesses.[4] The examination of cerebrospinal fluid yields limited information and exhibiting a low success rate in isolating the pathogen through fluid cultures. Listeria is more frequently detected in blood culture.[2] For assessment of lesions and accurately evaluate the extent of the disease, as well as to monitor treatment, a cranial magnetic resonance imaging (C-MRI) should be conducted.[2]

Treatment for Neurolisteriosis is Ampicillin 2g IV every 4 hours (or penicillin G 4 million units IV every 4 hours) with or without gentamicin 5mg/kg/day IV in three divided doses for a duration of at least 1 week.[5]

However, close monitoring of renal function is essential when using gentamicin. It is advised to avoid the use of dexamethasone due to its association with worse outcomes. For cerebral abscess, treatment duration of 4-6 weeks is recommended based on clinical response.[6]

CONCLUSION

This case report sheds light on the complexities and challenges associated with neurolisteriosis. Through the detailed analysis of the patient's presentation, diagnostic procedures, and treatment course, we emphasize the importance of early recognition and intervention in managing this rare but serious neurological infection especially in immunocompromised patients.

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DECLARATION

The authors have no conflict of interest to disclose.

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