

Listeria Rhombencephalitis

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Rhombencephalitis is the inflammation of brainstem and cerebellum (hindbrain) and has a variety of potential underlying etiologies that respond variably to treatment, including infections, inflammatory disorders, parainfectious disorders, and paraneoplastic syndromes.¹ *Listeria* is the most common infectious agent causing rhombencephalitis and mostly affects immunocompromised individuals.

A 48-year-old man with background history of multiple myeloma (on chemotherapy with bortezomib, thalidomide, and dexamethasone) was admitted with right hemiplegia and altered mental status. His symptoms began 10 days ago immediately after last course of chemotherapy, with low-grade fever and headache, followed by right hemiparesis. He was hospitalized at another center with a probable diagnosis of acute disseminated encephalomyelitis (ADEM) or herpes encephalitis and was treated with acyclovir and steroids. Subsequently his neurological status gradually worsened and he became quadriparetic and unresponsive. On arrival to our hospital, he was febrile, unresponsive (Glasgow Coma Scale [GCS] of 4/15). He had right third, fourth, and sixth cranial nerve palsy and asymmetric quadriparetic, right side more severely affected. Magnetic

resonance imaging (MRI) brain revealed irregular confluent areas of T2 and fluid-attenuated inversion recovery (FLAIR) hyperintensities involving frontal and temporal white matter, both internal capsule, midbrain, pons, and bilateral middle cerebellar peduncle (►Fig. 1). Minimal patchy diffusion restriction was noted in bilateral middle cerebellar peduncles and internal capsule (►Fig. 2). Patchy contrast enhancement was noted in dorsal midbrain, pons, and part of right internal capsule (►Fig. 3). Blood investigations revealed leukocytosis, normal renal function, and liver function tests. Cerebrospinal fluid (CSF) analysis revealed raised protein of 92 mg/dL, normal glucose, and elevated white cell count of 135 cells/mm³ (L68% and P32%). Blood culture and CSF culture grew *Listeria monocytogenes* confirming the diagnosis of *Listeria rhombencephalitis*. He was treated with ampicillin injection for 4 weeks and oral cotrimoxazole for 8 weeks, and he recovered well with mild residual quadriparetic.

Encephalitis due to *L. monocytogenes* shows predilection for the brainstem, cerebellar peduncle, and cerebellum as in this patient. Other infrequent sites of involvement include supratentorial white matter, thalamus, internal capsule, and spinal cord.^{1,2} Most common MRI feature is

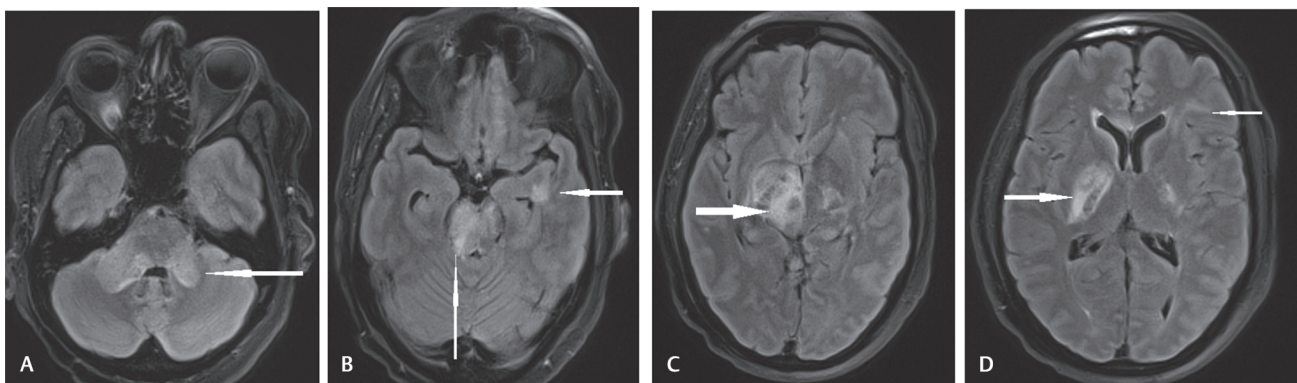


Fig. 1 MRI T2 FLAIR axial sequences showing (A) bilateral symmetrical hyperintensities involving middle cerebellar peduncle(MCP); (B) hyperintensities involving pons and left temporal white matter; (C) hyperintensities involving midbrain; and (D) hyperintensities involving both internal capsule, left frontal white matter, and corpus callosum. FLAIR, fluid-attenuated inversion recovery; MRI, magnetic resonance imaging.

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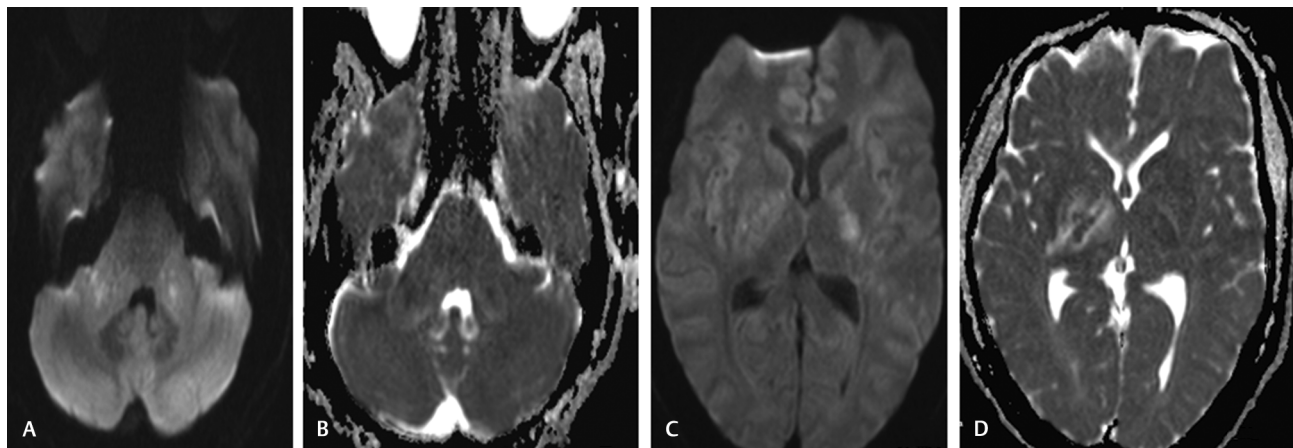


Fig. 2 MRI (A) diffusion weighted imaging (DWI) showing hyperintensities involving both middle cerebellar peduncle (MCP), (B) corresponding apparent diffusion coefficient (ADC) sequence showing hypointensity of MCP suggestive of diffusion restriction, (C) DWI showing hyperintensities involving both internal capsules, and (D) corresponding ADC showing corresponding hypointensities involving both internal capsule. MRI, magnetic resonance imaging.

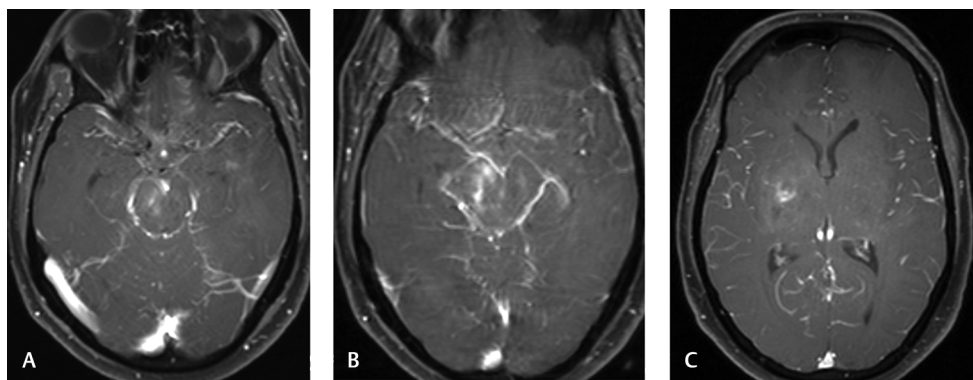


Fig. 3 MRI Postcontrast T1 sequence showing patchy contrast enhancement involving (A) pons (B) midbrain (C) right internal capsule. MRI, magnetic resonance imaging.

the T2 hyperintensity followed by contrast enhancement as seen in this patient.¹ Other MRI findings include abscess formation, hydrocephalus, edema, and hemorrhages. We noted patchy areas of diffusion restriction indicating areas of necrosis. The regions of restricted diffusion were surrounded by hyperintense signal on T2-weighted images that most likely denoted as edema.^{2,3} The typical distribution patterns in *L. rhombencephalitis* may be due to the proliferation and spread of *L. monocytogenes* along the intracerebral tracts.^{4,5} In rhombencephalitis, lesions are usually confined to brainstem and cerebellum. Rarely, it can involve internal capsule, frontal lobe, and temporal lobe. This might lead to misdiagnosis as herpes encephalitis or demyelination as in our patient. MRI has a major role in early diagnosis of *L. rhombencephalitis*, as isolation of *L. monocytogenes* by culture or PCR may be possible only in up to 50 to 60% of cases.⁶

Conflict of Interest

None declared.

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