



# Cauda Equina Enhancement in Wernicke Encephalopathy: An Atypical Radiologic Manifestation

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Indian J Radiol Imaging 2026;36:270–273.

## Abstract

A 13-year-old female with restrictive eating and associated weight loss presented with progressive neurological decline, including bilateral ptosis, gait disturbance, dysphonia, and hyporeflexia. Brain magnetic resonance imaging revealed T2 and FLAIR (fluid-attenuated inversion recovery) hyperintensities in the medial thalami, periaqueductal gray matter, and mild diffuse cerebral and cerebellar volume loss characteristic of Wernicke encephalopathy (WE). Additionally, smooth enhancement of the cauda equina nerve roots was observed, a finding not usually seen in thiamine (vitamin B1) deficiency. Infectious, metabolic, and autoimmune causes were ruled out, and low serum thiamine levels established WE. This case highlights an atypical radiologic feature of WE—cauda equina enhancement, suggesting broader neurological involvement. Early diagnosis and thiamine repletion therapy are required immediately to prevent long-term neurological damage.

## Keywords

- ▶ Wernicke encephalopathy
- ▶ thiamine deficiency
- ▶ cauda equina enhancement

## Introduction

Wernicke encephalopathy (WE) is a thiamine (vitamin B1) deficiency-related neurological disorder, commonly seen in individuals with alcohol use disorder, which can also be seen in individuals who are malnourished and suffering from restrictive eating disorders.<sup>1,2</sup> WE typically presents with ocular dysfunction, ataxia, and altered mental status, with characteristic T2 and fluid-attenuated inversion recovery (FLAIR) hyperintensities of medial thalami, periaqueductal gray, and mammillary bodies on brain magnetic resonance imaging (MRI).<sup>3</sup>

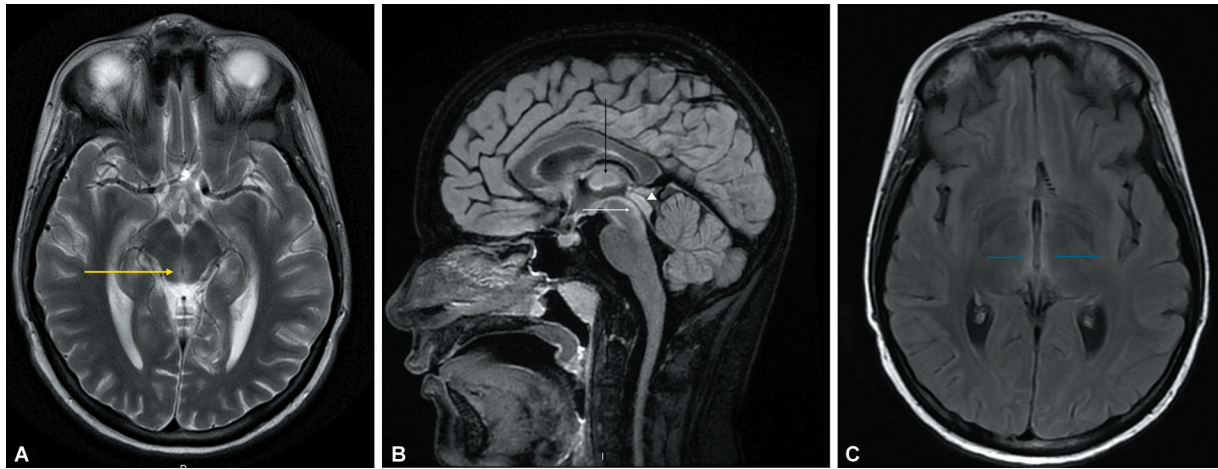
## Case Report

A 13-year-old female patient with past medical history of Hashimoto thyroiditis, asthma, and alopecia presented with gait difficulty, slurring of speech, forgetfulness, blurred vision, dizziness, and paresthesia of 2 weeks' duration. She also had abdominal pain, nausea, vomiting, and restrictive eating behavior resulting in about 15-lb weight loss over several months. On initial neurologic examination in another hospital, she was found to have bilateral ptosis, worsening nystagmus on upward gaze, diplopia, dysphonia, upper extremity hyporeflexia, lower extremity areflexia, intermittent confusion, and

article published online  
August 18, 2025

DOI <https://doi.org/10.1055/s-0045-1810626>.  
ISSN 0971-3026.

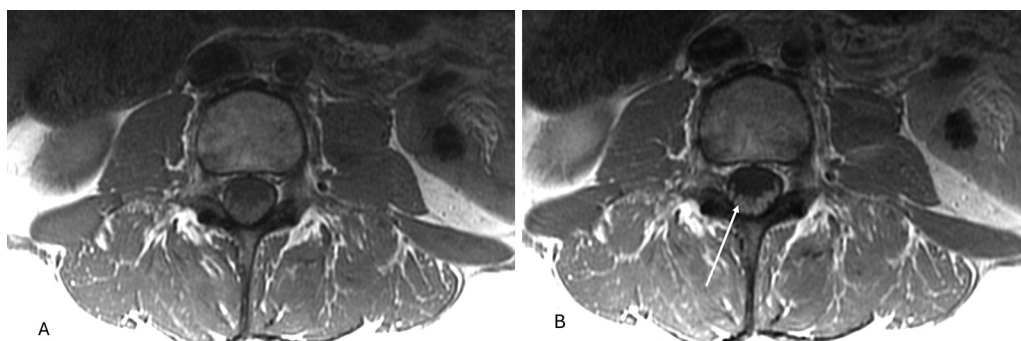
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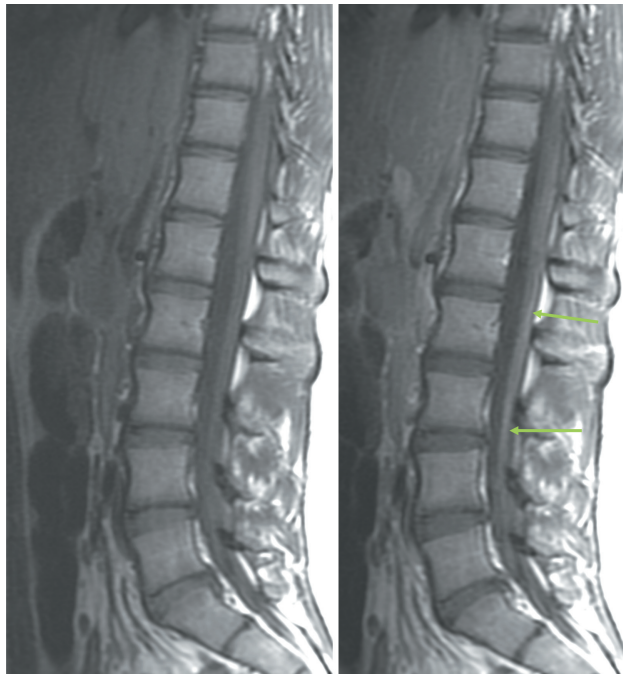
**Fig. 1** Brain MRI in Wernicke encephalopathy. (A) Axial T2-weighted image showing hyperintensity surrounding the cerebral aqueduct (yellow arrow). (B) Sagittal volumetric post contrast FLAIR image demonstrating hyperintensity in the massa intermedia (black arrow), periaqueductal region (white arrow), and tectum (arrowhead). (C) Axial FLAIR image showing bilateral medial thalamic hyperintensities (blue arrows) and mild cerebral atrophy evidenced by widened sulci and cisterns. FLAIR, fluid-attenuated inversion recovery; MRI, magnetic resonance imaging.

inability to walk. Differential diagnoses considered were viral encephalitis, metabolic derangements, mitochondrial disease, and WE. A nerve conduction study demonstrated normal right median and ulnar motor responses including conduction velocities and F-waves. The lower extremity nerve conduction studies were mildly abnormal. The right fibular motor response to the extensor digitorum brevis is borderline low in amplitude with normal distal latency and conduction velocity. The right fibular motor response to the tibialis anterior muscle is borderline in amplitude with normal distal latency and conduction velocity. Brain MRI demonstrated T2 and FLAIR hyper-intensities in periventricular white matter surrounding the third ventricle, bilateral medial thalami, and cerebral aqueduct, with mild diffuse cerebellar and cerebral parenchymal volume loss. The mammillary bodies demonstrated a normal signal (► **Fig. 1**). The optic nerves demonstrated normal signal, but there was minimal optic nerve head protrusion. Proton magnetic resonance spectroscopy utilizing short echo time (TE) of 35 ms from left thalamus, left parietal lobe white matter, and left occipital lobe was normal. Spinal MRI did not reveal spinal cord signal abnormality or findings of subacute combined degeneration, but there was smooth enhancement

or nodularity of the nerve roots (► **Fig. 2** and **3**). There was no cervical spinal cord or nerve root thickening or enhancement. Serum thiamine level was low (<6 nMol/L, normal 8–30 nMol/L), confirming the diagnosis of WE. The patient also had vitamin D deficiency (11 ng/mL—deficient is less than <20 ng/mL) and folate deficiency (5.7 ng/mL; normal: greater than 5.9 mg/mL). B12, vitamin E, zinc, Cu, Fe, selenium, serum iron binding capacity, transferrin, magnesium, calcium, and phosphorus levels were normal. Given the patient's history of Hashimoto thyroiditis, Hashimoto's associated encephalopathy was also considered in the differential, but her normal thyroid function tests made it less likely. Fundus examination demonstrated normal optic disc. Serological test for myasthenia gravis was negative. A computed tomography (CT) of abdomen and pelvis, upper gastrointestinal endoscopy, and colonoscopy were obtained due to recurrent vomiting, which were normal. A lumbar puncture was performed after MRI of brain and spine revealing normal cell count and protein along with negative for oligoclonal bands and NMO/AQP4 antibodies. Flexible fiberoptic laryngoscopy demonstrated left vocal cord paralysis. MRI of entire spine did not reveal any pathology along the course of vagal or recurrent laryngeal nerve or obvious neck or mediastinal mass. The medulla oblongata



**Fig. 2** Pre-contrast (A) and post-contrast (B) gadolinium T1-weighted MRI of the lumbar spine L3 level shows enhancement of the cauda equina nerve roots without thickening (white arrow). MRI, magnetic resonance imaging.



**Fig. 3** Sagittal T1-weighted MRI images of the lumbar spine, shown pre-contrast (left) and post-contrast (right). The post-contrast image demonstrates smooth linear enhancement of the cauda equina nerve roots (green arrows). MRI, magnetic resonance imaging.

appeared normal on brain MRI. The exact cause of the left vocal cord paralysis remains unknown.

The patient received 3 weeks of thiamine replacement as well as vitamin D, vitamin B complex, and folate supplementation. There was some improvement in muscle weakness and paresthesia, and she required inpatient rehabilitation with physical therapy because of persistent neurological impairment. No follow-up imaging was performed to assess radiologic response; however, clinical improvement was observed following thiamine repletion.

## Discussion

WE, a thiamine (vitamin B1) deficiency associated neurologic disorder, presents with a triad of mental status changes, ophthalmoplegia, and ataxia.<sup>1,2</sup> Most frequently, it is encountered in patients with chronic alcoholism, but is also frequently seen in malnourished patients, those with restrictive eating disorders, persistent vomiting, and gastrointestinal tract disorders.<sup>2,3</sup> The MRI findings of WE are pathognomonic and typically show T2 and FLAIR hyperintensity of the periaqueductal gray matter, thalami, and mammillary bodies, with contrast enhancement.<sup>4</sup> However, involvement of the spine, particularly cauda equina nerve root enhancement, is rarely described in the literature.<sup>5</sup> Unlike typical cases of WE, our patient's mammillary bodies did not show signal alteration, which is considered an atypical finding in nonalcoholic or pediatric presentations.

The cauda equina nerves consist of lumbar, sacral, and coccygeal roots, and smooth enhancement is typically

suggestive of infection like Lyme disease or autoimmune pathologies, such as seen in Guillain-Barré syndrome (GBS). In chronic inflammatory demyelinating polyneuropathy (CIDP), sarcoidosis, and leptomeningeal carcinomatosis, the nerve roots may be thickened and demonstrate nodular enhancement or smooth enhancement.<sup>6</sup> In our case, normal cerebrospinal fluid (CSF) examination, absence of infectious findings, negative autoimmune antibodies, and the coexistence of WE on brain MRI and low serum thiamine levels suggest thiamine deficiency as a cause of cauda equina involvement.<sup>5</sup>

The pathophysiological basis of cauda equina nerve root enhancement in thiamine deficiency is yet to be elucidated. Thiamine is crucial for neuronal metabolism, particularly in the pentose phosphate pathway and Krebs cycle, and thiamine deficiency can lead to neuronal energy failure, oxidative stress, and demyelination.<sup>1</sup> It is possible that cauda equina nerve roots are susceptible to demyelination with severe thiamine deficiency.<sup>1</sup>

In addition to the classic imaging features, WE has also been associated with several atypical neuroimaging findings, including cortical involvement, red nucleus, dentate nucleus, splenium, caudate nucleus, cranial nerve enhancement and cranial nerve nuclei signal changes, cerebellar atrophy and signal changes in vermis, and spinal cord lesions like cervical spinal cord central grey matter T2 hyperintensities.<sup>4,5,7</sup> In addition to imaging findings, atypical clinical manifestations of WE including diplopia, nystagmus, slurred speech, visual loss, fever, and seizures have also been reported in both adult and pediatric populations.<sup>8,9</sup> Our patient presented several of these, including diplopia, nystagmus, and slurred speech. In our case, normal CSF examination, absence of infectious findings, negative autoimmune antibodies, low serum thiamine levels, and features of WE on brain MRI suggest thiamine deficiency as a possible cause of cauda equina involvement.<sup>5</sup> Importantly, spinal MRI showed no T2 signal changes or enhancement in the cervical or thoracic cord or roots, ruling out subacute combined degeneration and other myelopathies.<sup>6</sup> Nerve conduction studies showed a mildly abnormal pattern consistent with right distal fibular, primarily axonal neuropathy, while the upper limb responses were normal. Other micronutrient levels including B12, vitamin E, magnesium, zinc, and copper were within normal limits. Although the patient had coexisting deficiencies of vitamin D and folate, to the best of our knowledge, neither of these has been associated with cauda equina enhancement. Papilledema is rarely reported in pediatric WE, along with CT findings of low attenuation in the basal ganglia and thalami, enhancement in thalamus and hypothalamus, and MRI evidence of T2 hyperintensities in periaqueductal gray matter, mammillary bodies, medial thalami, and basal ganglia.<sup>10</sup> The combination of clinical findings, low serum thiamine, and characteristic MRI changes in the brain support a diagnosis of WE, and the coexisting cauda equina involvement may reflect a broader manifestation of nutritional neuropathy.<sup>1,5</sup>

## Conclusion

This case highlights an atypical radiologic finding of WE namely cauda equina nerve root enhancement, which expands the currently recognized spectrum of thiamine (vitamin B1) deficiency-related neuroimaging findings. For cauda equina nerve root smooth enhancement, thiamine deficiency should be considered as one of the differentials, alongside other causes like GBS, CIDP, sarcoidosis, leptomeningeal carcinomatosis, mitochondrial diseases, and infections such as Lyme disease or bacterial/viral meningitis. Early recognition of such imaging characteristics is crucial for early diagnoses and treatment to prevent permanent neurological impairments.

### Declaration of GenAI use

During the preparation of this manuscript, AI tool Grammarly was used for grammar correction, language refinement, and editing support. No AI tool was used for the generation of scientific content or interpretation of clinical data. All clinical findings, analysis, and conclusions were authored by the listed authors.

### Ethical Approval

Institutional ethics committee approval was obtained for this case report.

### Patient's Consent

Informed consent was obtained from the patient's guardian for publication of this case and accompanying images.

### Conflict of Interest

None declared.

## Acknowledgments

The authors would like to thank the Pediatric Radiology and Neurology Department at UPMC Children's Hospital for their guidance and support and providing access to clinical resources during the preparation of this case report.

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