Wernicke Encephalopathy with Atypical Findings on Magnetic Resonance Imaging

Sir,

Wernicke encephalopathy (WE) is a life-threatening condition due to thiamine deficiency, most commonly due to alcoholism. The classical clinical triad of ocular signs, encephalopathy, and ataxia for the clinical diagnosis of WE as described by Carl Wernicke in 1881, is seen only in 16%–38% of cases. [1] We report a rare case of WE with typical clinical features. Magnetic resonance imaging (MRI) of the brain showed atypical findings with extensive involvement of brainstem and corpus callosum.

A 50-year-old nonhypertensive, nondiabetic, and nonalcoholic male presented with a history of unsteadiness of gait, confusion, and blurring of vision of 3 weeks. No history of recent febrile illness. No history of seizures. He was disoriented to time and place with impaired memory. He had horizontal gaze-evoked nystagmus with conjugate gaze palsy. Fundus was normal. Tandem gait was impaired with normal power and sensation in the upper and lower limbs.

MRI brain revealed nearly symmetrical bilateral T2 and fluid-attenuated inversion recovery (FLAIR) hyperintensities in entire pons and midbrain including cerebral peduncles, thalami including pulvinar and periventricular areas of third ventricle, anterior thalamo-capsulo-ganglionic regions, corona radiata, genu and proximal body of corpus callosum extending into the centrum semiovale on the left side, anterior to the fourth ventricle, and in the periaqueductal region [Figures 1-3]. There was subtle diffusion restriction and contrast study revealed focal enhancement in the left cerebral peduncle and right half of splenium of corpus callosum. [Figures 4 and 5]. Laboratory investigations, including blood glucose, serum creatine, electrolytes, and liver function tests, were within normal limits. Antinuclear antibodies were negative, and he had nonreactive HIV-1 and HIV-2 antibody tests. Whole body 18 fluorodeoxyglucose positron-emission tomography-computed tomography (CT) scan showed no metabolically active lesions. CSF analysis was unremarkable. WE was suspected, and therapeutic doses of parenteral thiamine were administered. Craniotomy with biopsy from corpus callosal lesion was done to exclude the differentials including diffuse glioma with gliomatosis cerebri pattern of spread. Histopathological examination revealed focal areas of hemorrhage and blackish pigmentation [Figure 6]. No evidence of demyelination or neoplasia. It was concluded to be consistent with WE. Postbiopsy follow-up MRI 1 week after initiation of the treatment revealed similar findings as the initial scan. The patient recovered partially over next 2 weeks. Based on the typical clinical features, supportive biopsy findings, ruling out possible differentials, and clinical improvement with parenteral thiamine the final diagnosis of WE was made.

WE is a potentially treatable neurological emergency. A high index of suspicion is required given atypical clinical and imaging features. CT is not much of use in the diagnosis of WE. Typical MRI findings in WE include symmetrical T2 and fluid-attenuated inversion recovery hyperintensities in thalami, periventricular areas of the third ventricle, periaqueductal area,

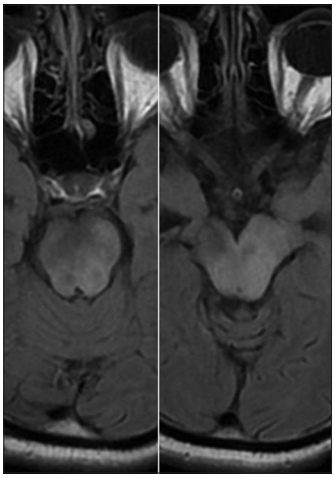

Figure 1: Wernicke Encephalopathy. T2 fluid-attenuated inversion recovery axial image reveals hyperintensity in the pons and midbrain including periaqueductal region. Such extensive signal changes in brainstem are not described under typical or atypical changes of Wernicke encephalopathy

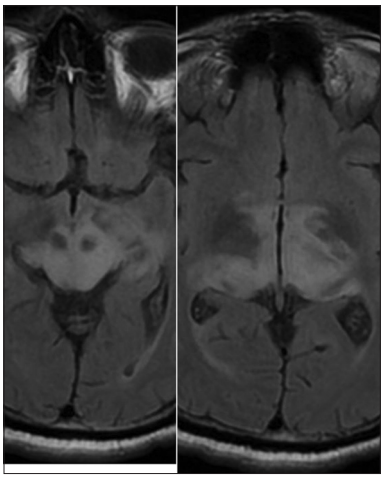

Figure 2: Wernicke encephalopathy. T2 fluid-attenuated inversion recovery axial image reveals extensive increased signal intensity in thalami around the third ventricle including pulvinar and capsuloganglionic regions

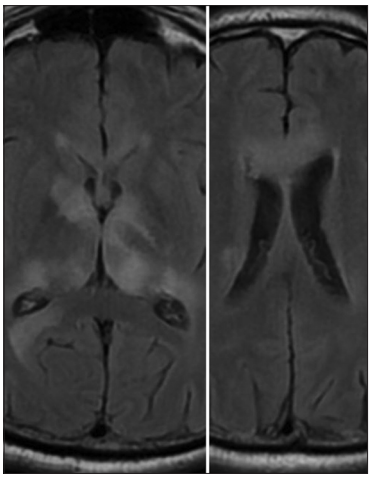

Figure 3: Wernicke encephalopathy. T2 fluid-attenuated inversion recovery axial image reveals extensive increased signal intensity in thalami around the third ventricle including pulvinar, capsuloganglionic regions with mass like lesions in the genu of corpus callosum mimicking diffuse glioma with gliomatosis cerebri pattern of spread. Involvement of genu with mass-like lesions is not previously described in Wernicke encephalopathy

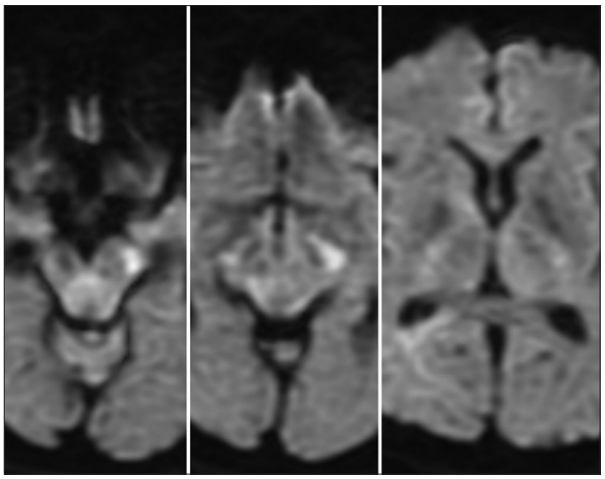

Figure 4: Wernicke Encephalopathy (WE). Diffusion weighted images reveal only faint increased signal intensity in the left cerebral peduncle and right half of splenium of corpus callosum with subtle corresponding restriction on ADC (not shown)

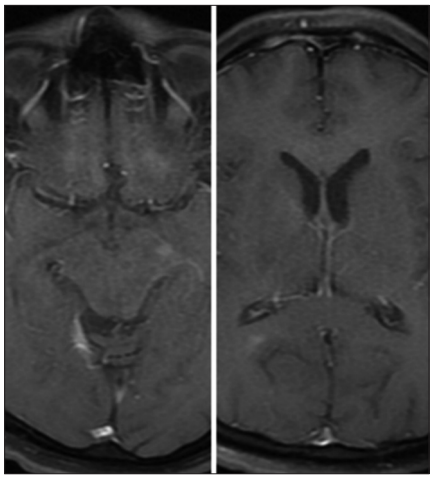

Figure 5: Wernicke Encephalopathy (WE). Post contrast fat saturated T1 weighted images reveal focal enhancement in the left cerebral peduncle and right half of splenium of corpus callosum

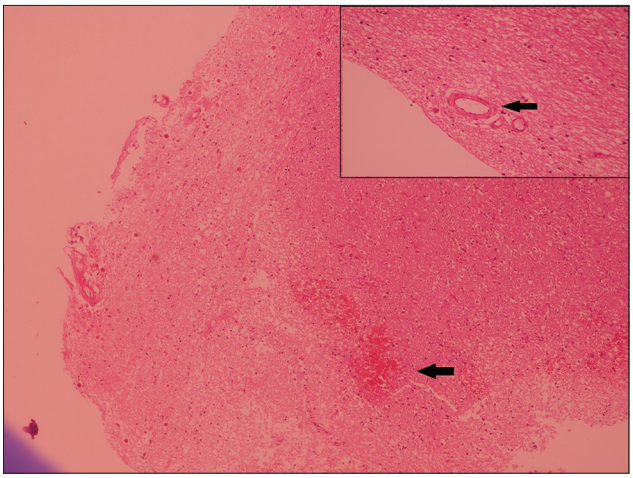

Figure 6: Wernicke encephalopathy. HPE of biopsy specimen from genu of corpus callosum reveals edematous white matter with petechial hemorrhages and thickened blood vessels (inset) changes compatible with Wernicke encephalopathy

mammillary bodies, and tectal plate and in the periventricular gray matter located anteriorly to the fourth ventricle with enhancement on contrast. T1 postcontrast imaging is recommended in suspected cases of WE even when the plain study is negative.[2,3] Mammillary bodies are not involved in our case. Atypical MRI findings have been variably described in the previous studies with symmetrical signal changes in the cerebellum, vermis, dorsal medulla, pons, red nuclei, cranial nerve nuclei, splenium of corpus callosum, and cerebral cortex and are usually associated with typical findings.[4,5] Atypical MRI findings are more common in nonalcoholic patients but have also been seen in alcoholics.[5]

The present case highlights the atypical imaging features of WE and empirical treatment with parenteral thiamine may be considered in patients with suggestive clinical features, pending the definitive diagnosis.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

Kiran Kumar Ramineni, Sravan Kumar Marupaka[1], Ravikanth Jakkani[1], Abhijeet Ingle[2]

Departments of Neurology, [1]Radiology and [2]Pathology, Yashoda Superspeciality Hospitals, Hyderabad, Telangana, India

Address for correspondence: Dr. Sravan Kumar Marupaka, Plot No. 364, 3- 7- 62, South End Park, Mansoorabad, LB Nagar, Hyderabad - 500 068, Telangana, India.
E-mail: ishasravan@gmail.com

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