

Bithalamic Dysfunction in Wernicke's Encephalopathy

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We describe a 65-year-old, nonalcoholic, right-handed female with multiple vascular risk factors who developed transient visuospatial hemineglect and global aphasia after presenting with the classic triad of Wernicke's encephalopathy (mental status changes, nystagmus/ophthalmoplegia, and ataxia). A brain MRI showed no evidence of acute infarction, but demonstrated signal

change in the medial thalami and mammillary bodies. Intravenous thiamine therapy was given, and visuospatial hemineglect and ophthalmoplegia disappeared while the aphasia improved. The occurrence of these acute transient clinical features has not been previously reported in Wernicke's encephalopathy.

A 65-year-old, nonalcoholic, right-handed female with hypertension, diabetes, hyperlipidemia, and a history of ischemic strokes with residual right-sided hemiparesis initially presented with nausea, vomiting, poor oral intake and a 43-kg weight loss (25% of baseline weight) over several weeks. On admission, she received a continuous intravenous glucose infusion. Four days later she developed disorientation. On initial evaluation, she did not exhibit neglect, but was observed to be inattentive and only followed simple requests. The patient was oriented only to her name and identified few simple objects. Further examination revealed binocular abduction paresis with nystagmus, bilateral upper limb dysmetria, and mild residual right-sided hemiparesis. A clinical diagnosis of Wernicke's encephalopathy was made and she was given intravenous thiamine at 1500 mg/day. However, on the following day, the patient was unable to express, comprehend or repeat language (global aphasia). Her gaze deviated to the right and she only attended to examiners on the right side, not on the left (profound visuospatial neglect). Although significant inattention was present, she followed simple requests at times, and her language deficit was constant and out of proportion to inattention. She had notably decreased limb movement on the left side, even in comparison to her previously known right hemiparetic side. The patient did not withdraw, regard, or visually track noxious stimuli in the left arm, which we interpreted as another sign of visuospatial hemineglect on the left side. Seven hours after examination, a brain MRI obtained to rule out an acute stroke showed no evidence of acute infarction (diffusion weighted imaging MRI was negative) and instead demonstrated typical MRI findings for Wernicke's encephalopathy, including signal change in the medial thalami and mammillary bodies (Figure 1). Intravenous thiamine therapy was continued and, three days later, visuospatial hemineglect and ophthalmoplegia abated while aphasia improved. Intravenous thiamine was reduced to 500 mg/day. After seven days of intravenous thiamine therapy, she was transitioned to oral thiamine at 100 mg/day. Two weeks into thiamine therapy, the patient was fully oriented, speaking fluently and following all requests.

Extensive gastrointestinal work-up revealed gastroesophageal reflux disease, gastritis/duodenitis, and gallstones with chronic cystitis. The patient required total parenteral nutrition for several days, but gradually tolerated an oral diet. She was discharged on oral thiamine at 100 mg/day.

DISCUSSION

Wernicke's encephalopathy is characterized by a triad of mental status changes, nystagmus or ophthalmoplegia, and ataxia due to thiamine (vitamin B1) deficiency (Sechi & Serra, 2007). The syndrome occurs due to selective dysfunction of brain regions including the thalami, mammillary bodies, pontine tegmentum, abducens and oculomotor nuclei, and cerebellum. Our patient initially presented with the classic triad of Wernicke's encephalopathy. This was followed by onset of unexpected visuospatial hemineglect on the left side and global aphasia, leading us to suspect a stroke. A brain MRI revealed classic radiographic findings of Wernicke's encephalopathy, including signal changes in the medial thalami and mammillary bodies (Zuccoli & Pipitone, 2009) and confirmed the diagnosis. It is well known that additional neurologic signs including hyperthermia, increased muscle tone, paresis, choreic dyskinesias, and coma may appear a few days after the onset of initial symptoms (Sechi & Serra, 2007). However, a transient appearance of dense visuospatial hemineglect or global aphasia has not been previously reported. We speculate that this may have been caused by a transient dysfunction of the bilateral thalami, given the striking signal alteration in this region on MRI. Dysfunction of the right thalamus can cause visuospatial hemineglect on the left side (Bogousslavsky, Regli, & Uske, 1988; De Witte et al., 2011; Watson & Heilman, 1979), and dysfunction of the left thalamus can cause aphasia (Bogousslavsky et al., 1988; De Witte et al., 2011; Kumar, Masih, & Pardo, 1996). For example, in the literature a case has been described where a patient with deep cerebral venous thrombosis with bithalamic infarction presented with a transient left-side visuospatial neglect, aphasia and amnesia (Benabdeljlil et al., 2001), which resembles our case. Another consideration is our patient's prior history of lacunar infarcts in subcor-

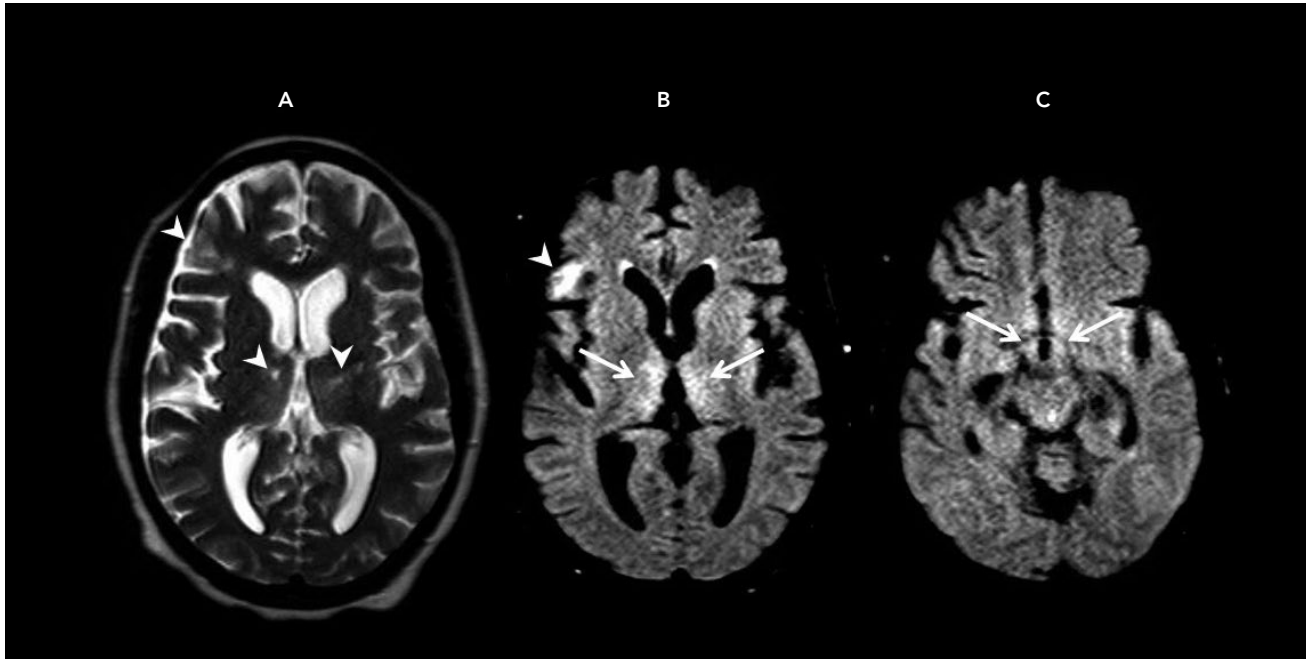


Figure 1 | (A) Axial T2 weighed image shows chronic lacunar infarcts in the right frontal operculum and thalami (arrowheads). (B) Axial FLAIR image reveals hyperintensities in the bilateral paramedian thalami (arrows). An old lesion from ischemic stroke is seen in the right frontal operculum (arrowhead). (C) Axial FLAIR image shows signal intensity alterations of mammillary bodies (arrows).

tical structures, including the bilateral thalami and the left corona radiata, resulting in residual right-sided hemiparesis. Thus the patient's remaining thalamic function may have been more vulnerable to transient metabolic insults related to thiamine deficiency, and her symptoms could have partially stemmed from an anamnestic response to old lacunes in the thalami. Transient gaze deviation and aphasia could be ictal or post-ictal phenomena, but we did not obtain an EEG on this patient since we felt her series of symptoms were not epileptic in nature. In elderly patients, Wernicke's encephalopathy can be seen in a nonalcoholic setting and can present with atypical features, possibly modified by multiple medical comorbidities.

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Author Contributions: HN and EM drafted the manuscript. RLZ and MSR revised the manuscript.

Disclosure: The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. HN, EM, CF, and RLZ report no conflicts of interest to disclose. MSR has received honoraria for educational activities from the American Headache Society, American Academy of Neurology, Medlink, and Springer as a section editor for *Current Pain and Headache Reports*. MSR also receives book royalties from Wiley. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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