



Wernicke's Encephalopathy Following Gastrointestinal Surgery: MRI and DWI Findings

Gastrointestinal Cerrahi Sonrası Gelişen Wernicke Ensefalopatisi: Konvansiyonel ve Difüzyon MRG Bulguları

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Summary

Wernicke's encephalopathy (WE) is a neurologic disorder resulting from thiamine (vitamin B1) deficiency characterised by the ocular signs, altered consciousness and ataxia. It is usually associated with chronic alcohol abuse. Recently, it emerged as a complication following bariatric surgery. The classical clinical triad occurs in a small group of patients, and early replacement of thiamine is an important prognostic factor. Hence, magnetic resonance imaging (MRI) is useful to support the diagnosis. In this case, we report MRI and diffusion weighted imaging findings of WE in a patient without history of alcoholism after gastrointestinal surgery. (Turkish Journal of Neurology 2014; 20:84-86)

Key Words: Gastrointestinal surgery, magnetic resonance imaging, thiamine, Wernicke's encephalopathy

Özet

Wernicke ensefalopatisi (WE), tiamin eksikliği sonucu oluşan, oküler bozukluk, bilinç değişikliği ve ataksi ile kendini gösteren klinik bir antitedir. Sıklıkla kronik alkol kullanımı ile birlikte görülmekle beraber son yıllarda bariyatrik cerrahilerin bir komplikasyonu olarak da karşımıza çıkabilmektedir. Klasik klinik bulgular hastaların küçük bir bölümünde izlenebildiğinden ve erken tiamin replasmanının hastalığın prognozunda belirleyici olması nedeniyle erken tanıda manyetik rezonans görüntüleme (MRG) önem taşımaktadır. Bu yazıda gastrointestinal cerrahi sonrasında kronik alkol kullanımı olmayan bir hastada gelişen WE'nin MRG ve difüzyon ağırlıklı görüntüleme (DAG) bulguları sunulmuştur. (Türk Nöroloji Dergisi 2014; 20:84-86)

Anahtar Kelimeler: Gastrointestinal cerrahi, manyetik rezonans görüntüleme, tiamin, Wernicke ensefalopatisi

Introduction

Wernicke encephalopathy (WE) is an acute neuropsychiatric disorder that develops due to thiamine (vitamin B1) deficiency, presenting with specific symptoms. It is often associated with chronic alcoholism. However, it can also be seen after gastrointestinal surgery, chemotherapy, systemic infections and prolonged vomiting since they all cause thiamine deficiency. Due to the recently increased use of gastric bariatric surgeries, they stand out in the etiology of WE (1,2).

Cases classically present with acute consciousness disorders, ophthalmoplegia and ataxia symptoms. Since the clinical representations are not always typical, neuroimaging findings carry importance in the diagnosis stage. In this article, we present the conventional magnetic resonance imaging (MRI) and diffusion-weighted imaging (DWI) findings in a case who was diagnosed with WE after undergoing gastrointestinal surgery and recovered clinically and radiologically due to early diagnosis.

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Case

Sixty-three year old male patient who had cholecystectomy T-tube drainage due to acute cholecyst came to general surgery department of our hospital for massive lower gastrointestinal bleeding. The patient who had a hemicolectomy operation was being followed at the ICU because his thigh muscles developed collection. After he started having sudden consciousness changes and disorientation, he was examined by the neurology clinic. He did not have a history of chronic alcohol abuse and he had been rapidly losing weight for the past 2 months. His neurological examination did not show any lateralization signs. The patient was in a state of confusion and had lost his location, time and person orientation. He was referred to the radiology department with a cerebrovascular pre-diagnosis. He had an emergency MRI scan with a 1.5 T (Signa hdx GE Healthcare, Milwaukee, WI, USA) magnet without contrast. Coronal FLAIR, axial T2 TSE, axial and sagittal T1 TSE sequences and diffusion-weighted images ($b=1000 \text{ sec/mm}^2$) with ADC maps were used for diagnostic purposes. Sedation was not used upon the request of the clinician. There were motion artifacts present in the diffusion-weighted images which also showed hyperintense regions in the mammillary bodies, bilateral medial parts of thalamus, in the vicinity of 3rd ventricle, periaqueductal regions (Figure 1A). These regions seemed isointense in the ADC map. The areas identified in the T2-weighted and FLAIR (Figure 2A) sequences were seen to be hyperintense (Figure 2). Acutely developing changes in consciousness following gastrointestinal surgery and severe weight loss history, in combination with MRG findings, suggested WE diagnosis. After vitamin B1 treatment, the patient's clinical symptoms improved in 1 week and his 3-month follow-up MRI showed that the previous imaging findings have improved and disappeared (Figure 1B, 2B).

Discussion

The clinical description of Wernicke encephalopathy includes the classical triad described by Wernicke: ocular findings (nystagmus, bilateral lateral rectus paralysis), consciousness changes and ataxia. However, only as little as half of the patients may exhibit this triad which causes under-diagnosis of WE (3).

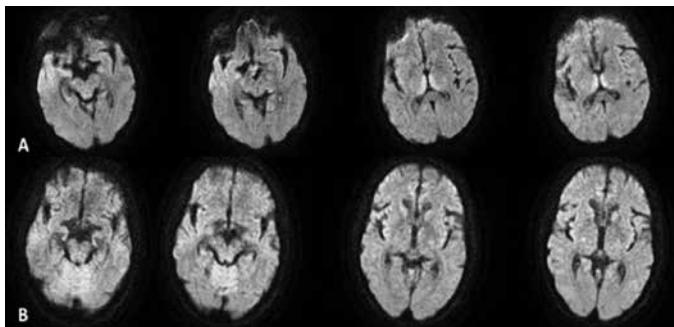


Figure 1. A) Axial DWI from the first contrast-free MRI of the patient shows hyperintense regions in periaqueductal area, mammillary body and bilateral paramedian thalamus. B) 3 months following the thiamine replacement treatment, the signal abnormalities in the first scan had disappeared. Due to the motion artefact, there is an offset in the slice planes.

Thiamine deficiency plays an important role in the etiopathogenesis of the disease. Thiamine is a vitamin necessary for the continuum of osmotic gradient of cell membrane, glucose metabolism and neurotransmitter synthesis. Thiamine absorption is related to carbohydrate intake, and the body's thiamine storage is depleted within 4-6 weeks when the intake is cut off. The most well known case of thiamine deficiency is of chronic alcoholism (4). In addition, gastrointestinal surgery, chemotherapy, systemic infections, dietary deficiencies, and excessive vomiting may also cause thiamine deficiency (1,2). In our case, the patient did not have a history of alcoholism but his rapid weight loss secondary to a recent gastrointestinal surgery suggested nutritional thiamine deficiency.

Since the clinical findings in WE are not always typical, it is of importance to be familiar with the imaging findings seen in patients who do not have a history of alcoholism. Sensitivity of computerized tomography (CT) is significantly lower than that of MRI in the diagnosis of WE (5). Typical MRI findings include symmetrical signal changes in mammillary bodies, medial thalamus, tectum and periaqueductal regions visible in T2-weighted and FLAIR sequences. Atypical lesions can be seen in cerebellum, cerebellar vermis, cranial nerve nuclei, splenium and cerebral cortex. There can be individual differences in the cranial MRIs of the patients in especially in the acute stage depending on whether they use alcohol or not. Atypically located lesions are often seen in patients who do not use alcohol, whereas alcohol users often show lesions in mammillary body and thalamus. In addition, mammillary body atrophy in the acute stage is generally not observed in the non-alcoholic patients (6). Cytotoxic edema can be seen as diffusion constriction in the DWI. For this reason, DWI does not have high specificity in the diagnosis of WE (1). In our patient, we observed signal changes in the regions typical for WE and the lack of mammillary body atrophy was congruent with the fact that the patient was not an alcohol user.

The first MRI volumes of the patient have large amounts of movement artefacts especially in the axial T2 and FLAIR sequences (Figure 1A). Literature suggests that such movement artefacts are common in WE cases but they do not interfere with the validity of diagnosis (8).

Cranial MRI is important also in identifying or ruling out other pathologies that may cause consciousness changes. Signal

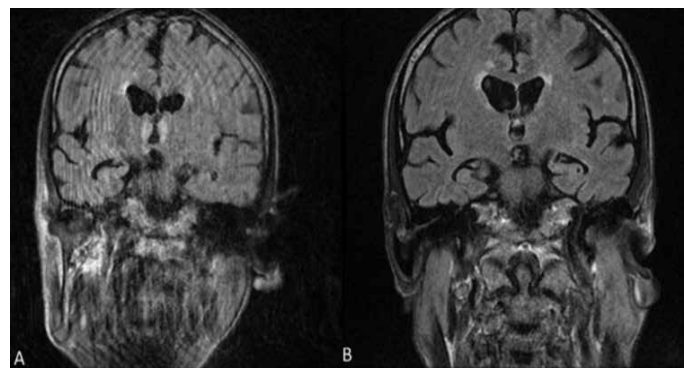


Figure 2. Pre-treatment A) and post-treatment B) coronal FLAIR at approximately the same positions shows the disappearance of the hyperintense signal in bilateral paramedian thalamus.

abnormalities in medial thalamus in cases of isolated consciousness changes may indicate Percheron artery blockage or deep cerebral venous thrombosis. The differential diagnosis table also includes Leigh disease, cytomegalovirus encephalitis and primary cerebral lymphoma (9).

The prognosis of WE is associated with the onset and the course of thiamine replacement. Therefore, early diagnosis and early start of the treatment is crucial for the improvement of the clinical symptoms. Zhong et al. reported in their 6-patient series that the hyperintense signal changes on periaqueductal area, medial thalamus and caudate nucleus in addition to the cortical involvement observed in T2 and FLAIR went back to normal within 2 weeks to a year following the onset of thiamine replacement (10). In our case the clinical improvement was seen within 1 week and the radiological improvement in the pathological signs was observed in the 3-month follow-up MRI. Also the vasogenic edema findings seen in DWI and ADC of our case indicated that it was still very early in the process, which turned out to be reversible (Figure 1A).

The incongruence of the lesions seen in our patient with the vascular blood supply area, the lack of other infectious, neoplastic or metabolic disorders and the typical localizations of the lesions suggested radiological diagnosis of WE. The diagnosis was confirmed by the clinical and radiological improvement of the patient's condition after thiamine replacement treatment.

Even though it is not a rare condition, WE is not very well known. It is not solely caused by chronic alcoholism and it may develop after rapid weight loss. Especially considering the increased number of bariatric surgery operations in the past 10

years, clinicians and radiologists should be alert for possible WE complications. Since the clinical findings associated with WE are not always typical, imaging findings has an important role in the diagnosis stage. Early diagnosis and treatment are important factors determining the prognosis of the disease.

References

1. Zuccoli G, Pipitone N. Neuroimaging Findings in Acute Wernicke's Encephalopathy: Review of the Literature. *AJR* 2009;192:501-508.
2. Chaves LC, Faintuch J, Kahwage S, Alencar FA. A cluster of polyneuropathy and Wernicke-Korsakoff syndrome in a bariatric unit. *Obes Surg* 2002;12:328-334.
3. Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. *J Neurol Neurosurg Psychiatry* 1986;49:341-345.
4. Torvik A, Lindboe CF, Rogde S. Brain lesions in alcoholics: a neuropathological study with clinical correlations. *J Neurol Sci* 1982;56:233-248.
5. Antunez E, Estruch R, Cardenal C, Nicolas JM, Fernandez-Sola J, Urbano-Marquez A. Usefulness of CT and MR imaging in the diagnosis of acute Wernicke's encephalopathy. *AJR* 1998;171:1131-1137.
6. Gascón-Bayarri J, Campdelacreu J, García-Carreira MC, Estela J, Martínez-Yélamos S, Palasí A, Delgado T, Reñé R. Wernicke's encephalopathy in non-alcoholic patients: A series of 8 cases. *Neurologia* 2011;26:540-547.
7. Bergui M, Bradac GB, Zhong JJ, Barbero PA, Durelli L. Diffusion-weighted MR in reversible Wernicke encephalopathy. *Neuroradiology* 2001;43:969-972.
8. Zuccoli G, Gallucci M, Capellades J, Regnicolo L, Tumiatì B, Giadàs TC, Bottari W, Mandrioli J, Bertolini M. Wernicke encephalopathy: MR findings at clinical presentation in twenty-six alcoholic and nonalcoholic patients. *Am J Neuroradiol* 2007;28:1328-1331.
9. Matheus MG, Castillo M. Imaging of acute bilateral paramedian thalamic and mesencephalic infarcts. *Am J Neuroradiol* 2003;24:2005-2008.
10. Zhong C, Jin L, Fei G. MR Imaging of Nonalcoholic Wernicke Encephalopathy: A Follow-Up Study. *Am J Neuroradiol* 2005;26:2301-2305.