

The Importance of Early Radiological Diagnosis in the Prevention of Cerebral Parenchymal Damage

Akut Pankreatit sonrası Wernicke Ensefalopatisi; Serebral Parankim Hasarın Önlenmesinde Erken Radyolojik Tanımın Önemi
Radyoloji

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Bünyamin Güney¹, Murat Yunus Özdemir¹, Önder Yeniçeri¹, Neşat Çullu¹

¹ Muğla Sıtkı Koçman Üniversitesi

Özet

Wernicke ensefalopatisi (WE), tiamin eksikliği sonucu gelişen, oftalmopleji, bilinç değişikliği ve ataksi ile kendini gösteren klinik bir tablodur. Klasik klinik tablonun, hastaların küçük bir bölümünde görülmesi ve erken tiamin replasman tedavisinin hastalığın prognozunda önemli olması nedeniyle, hastalığın tanısı hızlı ve erken konulmalıdır. Erken tanıda kranial manyetik rezonans görüntüleme (MRG) altın standarttır. Biz bu olgu sunumunda, pankreatit atağından sonra WE gelişen bir hastanın, medikal tedavi ile serebral parankimal hasar gelişmeden tamamen düzelmesinde erken radyolojik tanımın önemini vurgulamaktayız.

Anahtar kelimeler: *Wernicke ensefalopatisi, Tiamin eksikliği, MRG*

Abstract

Wernicke encephalopathy (WE) is a clinical manifestation of thiamine deficiency that develops with ophthalmoplegia, change of consciousness and ataxia. The diagnosis of the disease should be made quickly and early because the classical clinical picture is seen in a small percentage of patients and early thiamine replacement therapy is important in the prognosis of the disease. Cranial magnetic resonance imaging (MRI) is the gold standard for early diagnosis. In this case report, we emphasize the importance of early radiological diagnosis in a patient who developed WE after the attack of pancreatitis and completely recovered without medical cerebral parenchymal injury.

Keywords: *Wernicke encephalopathy, Thiamine deficiency, MRI*

Introduction

Wernicke encephalopathy (WE) is an acute neuropsychiatric syndrome caused by thiamine deficiency. The classic triad is ophthalmoplegia, ataxia and change of consciousness¹. It is often associated with alcoholism, but conditions including long-term parenteral nutrition therapy, after gastrointestinal system surgery, bariatric surgery, chemotherapy-related long-term vomiting, and rarely after acute pancreatitis are also associated with WE. Most cases of WE are missed by clinicians because patients with malabsorption due to different reasons do not present the classical signs associated with WE. As acute pancreatitis can lead to a variety of metabolic alterations and thus may cause changes in consciousness in these patients, physicians should keep in mind that they must be suspicious for patients who are at high risk and prompt thiamine replacement therapy must be applied. In this article, we presented the clinical and radiologic findings of a patient with thiamine deficiency following acute pancreatitis. Our prompt treatment resulted in a full recovery.

Case Report

A 47-year-old woman presented to the emergency department with sudden onset of abdominal pain, nausea and vomiting. She had epigastric tenderness and abdominal pain. She had no known disease, smoking and alcohol use, and her systemic examinations were normal. Her biochemical tests revealed that she had neutrophilic leukocytosis in the hemogram count; and her glucose level in blood was: 335 mg/dl, urea: 84.9 mg/dl, creatinine: 1.29 mg / dl,

Corresponding Author: Bünyamin Güney,
bunyaminguney@mu.edu.tr

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AST: 235 U/L, ALT: 99 U/L, LDH: 611 U/L, GGT: 158 U/L, alkaline phosphatase: 207 U/L, amylase:739 U/L, lipase: 978 U/L and CRP: 55.9 m/L. Abdominal-contrast computed tomography (CT) was performed to the patient with the diagnosis of acute pancreatitis. The patient who was diagnosed as acute necrotizing pancreatitis was admitted to the internal medicine service.

The patient's general condition deteriorated and blurred consciousness developed while she was in the internal medicine unit and she was admitted to the intensive care unit and intubated. She was given fluid electrolyte and antibiotic treatment for 6 days in the intensive care unit, and her general condition improved and she was extubated. When the patient was planned to be discharged from the intensive care unit, neurology consultation was requested due to sudden confusion and limited eye movements. In neurological examination performed in intensive care unit, in addition to confusion, there was no bilateral inward and outward motion in the eye movements and there was a limitation in bilateral up and down motion. As a result of the neurology consultation, emergency contrast cranial magnetic resonance imaging (MRI) and cranial diffusion MRI were performed. Cranial MRI revealed symmetrical pathological signal fields in the medial part of bilateral thalamus, in the periaqueductal area of the mesencephalon and in the dorsal sensory area of the brainstem, showing extensive T2W hyperintense signaling and moderate diffusion restriction (Figure 1a-d).

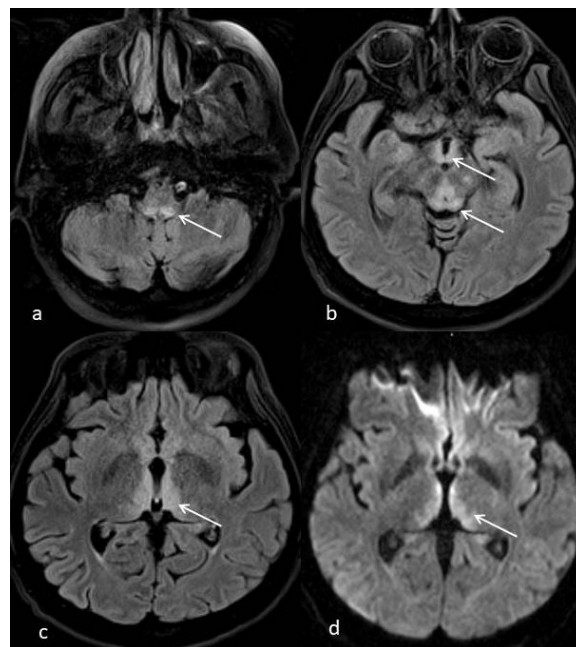


Figure 1

Figure a-b-c axial fluid suppression T2 weighted (FLAIR) images; hyperintense pathological signaling sites in the brain stem dorsal tract (1a), mesencephalon level and hypothalamic region (1b), bilateral thalamus in the medial section (1c), and moderate diffusion restriction in the section passing through the thalamus (1d).

The patient diagnosed with acute WE radiologically, was asked for a neurology consultation again. The neurology consultation result was also WE. Thiamine replacement therapy was administered 3x200 mg during the first three days and the following three days 3x100 mg intravenously. The treatment was continued as 100 mg/day by oral thiamine. After a total of 10 days of treatment, the patient's clinical findings improved. The cranial control MRI revealed that the radiologic findings returned completely to normal (Figure 2a-d).

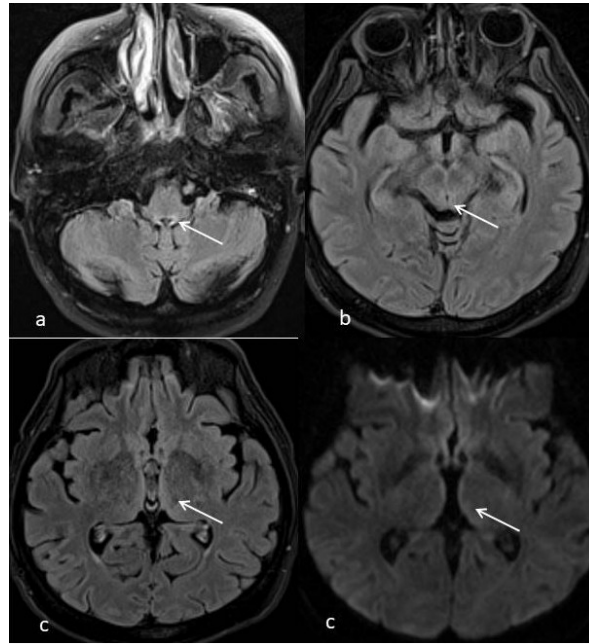


Figure 2

After thiamine replacement therapy, the pathological signaling sites in the same patient's FLAIR (2a-c) and diffusion (2d) images, which have passed through the same levels, are completely regressed without parenchymal destruction.

Written informed consent was obtained from the patient for publication of this case report.

Case Discussion

Wernicke encephalopathy is an acute neuropsychiatric syndrome associated with thiamine deficiency. Its classical clinical manifestations are ophthalmoplegia, ataxia and confusion¹. However, all of these findings are observed in only 10-20% of patients². Mental confusion is the most common finding. While clinically its incidence is 0.6-1.3 thousandth in the population, it is seen in the autopsy series at a rate of 8-28 thousandth³. The most common cause in the etiology of the disease is alcoholism. Other etiologic factors include gastrointestinal system surgery, bariatric surgery, long-term parenteral therapy, HIV, long-term vomiting associated with chemotherapy and frequent dialysis. The most common cause in pediatric WE cases has been reported as malignancy in the literature⁴. In our patient, there were signs of mental confusion and ophthalmoplegia, due to thiamine deficiency after pancreatitis.

Thiamine is a vitamin that is soluble in water and is able to be storable. Thiamine is also an important cofactor (cell energy metabolism) of the Krebs cycle and pentose phosphate pathway enzymes. Since, it is used in the metabolism of carbohydrates and many amino acids, cardiovascular and gastrointestinal disorders may develop alongside neuromuscular dysfunction in case of deficiency. Due to the deterioration in the metabolism of cerebral energy as a result of thiamine deficiency, brain damage may develop in the medial thalamus, periaqueductal grey matter, corpus mamillare and necrosis of the hypothalamus⁴. In the early stage of WE, findings are hyperintense lesions in the FLAIR and T2-weighted images seen on MRI at the level of bilateral medial thalamus, the periaqueductal grey matter, corpus mamillare and hypothalamus⁵.

Since, pancreatitis can lead to neuropsychiatric disorders caused by many changes in metabolites, clinicians should bear in mind that WE may develop in patients with confusion. Pancreatic encephalopathy is characterized

by disorientation, confusion and hallucinations in patients with acute pancreatitis.

In the literature, a wide demyelination of cerebral gray and white matter is described in acute hemorrhagic pancreatitis, but the pathogenesis is not clearly clarified⁶. To identify WE from acute pancreatic encephalopathy is possible with cranial MRI. In our patient, the cranial MRI, which was taken to determine the cause of the sudden confusion and ophthalmoplegia, had typical radiologic findings for WE, which provided early diagnosis. Our patient did not develop cerebral parenchymal necrosis and had a quick recovery as she received early diagnosis and treatment. Fei et al., has detected 9 of the 12 nonalcoholic WE patients who were diagnosed with pancreatitis between 1999 and 2006⁷. In another series, Sun et al., reported pancreatitis in 0.7% of patients diagnosed with WE⁸. Chen et al., reported that pancreatic encephalopathy should be considered in the clinical diagnosis in case of early-onset encephalopathy that develops in the first two weeks after the attack of pancreatitis, and WE should be considered in the clinical diagnosis in case of late encephalopathy⁹. In our case, the clinically occurring confusion and ophthalmoplegic findings emerged approximately sixteen days after the attack of the pancreatitis.

The diagnosis of WE is made based on clinical presentation. However, since clinical findings are not always typical, cranial MRI in non-alcoholic patients has a critical role for early diagnosis. In cranial MRI examination, the classical areas of involvement of WE are the corpus mamillare, 3rd and 4th ventricle circumference, tegmentum, dorsal-medial thalamic region, periaqueductal gray matter and hypothalamus. T1W images show decreased signal, T2W and FLAIR images show increased signal while diffusion-weighted images show diffusion restriction⁴. With early thiamine replacement therapy, abnormal signal changes completely improve but otherwise the atrophy of the corpus mamillare, superior cerebellar vermis and cortex may be permanent. In our case, typical radiologic involvement areas were present and with early radiological diagnosis and early medical treatment (thiamine replacement therapy), all pathological signal changes in the brain stem and mesencephalon disappeared.

WE has high mortality and morbidity rates. However, WE is a reversible condition if the diagnosis can be made early and the treatment is started early. Our patients' condition rapidly improved as the diagnosis was made early by the typical radiological findings and thiamine replacement therapy was started early. In WE, clinical findings are not always typical, therefore, if clinically there is suspicion of WE in postpancreatic cases, MRI should be performed. The role of MRI is important for early diagnosis as it often leads to prompt treatment for WE that could prevent brain damage.

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