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Wernicke's Encephalopathy Associated with Methanol Ingestion

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ABSTRACT

Wernicke's Encephalopathy (WE) is an acute onset disease characterized by ophtalmoplegy, ataxia and confusion. Thiamine (B1) deficiency is responsible essentially from etiology. In this article, we represent a case of WE following the methanol ingestion. A 39 year old right-handed male patient was brought to emergency department with nausea, vomiting, nonsense talking, and imbalance in walking after drinking 300 cc cologne contenting methanol. On admission, the patient was awake but he was apathic and disorientated. He had a global amnesia, ataxia, diplopia and nystagmus. A short time later, he developed decreased sight findings. The patient's cranial MRI relieved the thalamic and cerebellar intensity changes. The patient was treated with a thiamine cure and in the third day he totally recovered. We think that WE may develop following methanol ingestion and early treatment is important in the prognosis.

Key words: *Emergency department, methanol ingestion, Wernicke's Encephalopathy, early thiamine treatment*

ÖZET

Metanol Alımı İle İlişkili Wernicke Ensefalopatisi

Wernicke Ensefalopatisi (WE) oftalmopleji, ataksi ve konfüzyon ile karekterize akut başlayan bir hastalıktır. Hastalığın etyolojisinden esas olarak tiamin eksikliği sorumlu tutulmaktadır. Biz bu makalede metanol alınımını takiben Wernicke Ensefalopati geliştirmiş bir olguyu sunarız. 39 yaşındaki sağ elini kullanan erkek hasta, metanol içeren kolonyadan 300 cc içdikten sonra başlayan bulantı, kusma, konuşma ve yürüme bozukluğu ile acil servise getirildi. Başvuruda hasta uyanıktı, fakat apatik görünümünde ve dezoryante idi. Hastanın global amnezi, ataksi, diplopi ve nistagmusu vardı. Kısa bir sure sonra, hastanın görmesi azaldı. Bunun üzerine elde edilen kraniyal MRI'de talamik ve serebellar intensite değişikliği vardı. Hasta tiamin ile tedavi edildi ve üçüncü günde tam olarak düzeldi. Biz methanol alınımını takiben WE gelişebileceği ve erken tiamin tedavisinin hastalığın prognozunda önemli olduğunu düşünmekteyiz.

Anahtar kelimeler: *Acil servis, metanol alımı, Wernicke Ensefalopatisi, erken tiamin tedavisi*

Wernicke's encephalopathy (WE) is an acute or subacute onset disease characterized by ocular findings, ataxia and altered mental status (1). In the etiology of WE, thiamine (vitamin B-1) deficiency is essential (1-5). Thiamine insufficiency is essentially associated with chronic alcoholism (1,3,5). However, excessive glucose intake may cause thiamine deficiency. Because when 25-50 g of glucose is given in coma patients, at the same time, giving thiamine (intravenous; bolus) also has been recommended to prevent the precipitation of WE (4). In thiamine insufficiency, adenosine triphosphate (ATP) synthesis (carbohydrate metabolism), integrity of myelin sheath (lipid metabolism), and production of GABA are influenced negativelly (3,5). Methanol poisoning is potentially a fatal medical emergency because of its metabolism to formic acid, responsible for the direct toxicity (6). Formic acid inhibits the mitochondrial enzyme cytochrome c oxidase, which is involved in oxidative metabolism leading to the synthesis of ATP (7). On the other hand, methanol may cause thiamine insufficiency (8). Rotenstreich et al. (8) reported a case of methanol blindness recovered with the combination therapy of steroids and thiamine. In our research of the literature data, we couldn't find any cases of WE due to methanol intoxication. We report a case of acute WE recovered with thiamine therapy and following methanol ingestion.

CASE REPORT

A 39 year old right-handed male patient was brought to our emergency department (ED) with nausea, vomiting, nonsense talking, restlessness, and difficulty in walking. From his anamnesis, we learned that he drank 300cc of cologne contenting methanol and had nausea and vomiting three hours after methanol intake. In the past medical history, there was no significant disease. However he was an alcohol addict. On admission, the patient had a global amnesia. A short time later, he developed acute confusional state with attentiveness and disordered orientation. These findings were followed with ataxic walking, diplopia, and isolated horizontal nystagmus. The other cerebellar examination findings were normal. His plantar responses were flexor in the both extremities. Blood laboratory tests were normal with no evidence of metabolic acidaemia. In our hospital, blood methanol level can not be examined. At the 21st hour of ingesting, he developed confabulation and eye blurriness. In the funduscopic examination, the papilla was slightly pale. In the finger counting test, sight was 10 cm at the left eye and 2 m at the right eye. Other systemic examination findings were normal. According to history and findings, we therefore considered the diagnosis of Wernicke's Encephalopathy due to methanol and obtained magnetic resonance imaging (MRI) from the patient. The brain MRI of the patient relieved

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hyperintense focal areas in T2 W images and FLAIR sequences on bilateral the medial thalamus, the white matter regions of the frontal lobe, and the left side of the cerebellum (Figure 1 A, B). Electrocardiographic (ECG) findings were normal. We administered thiamine 100mg and ethyl alcohol 2 mg/kg. via intravenous rout about 22 hours after ingestion of methanol. In the second day of the therapy the confusion was resolved, with normal attention and orientation to person, place, and time. In the third day the patient was discharged from the hospital with a total cure.

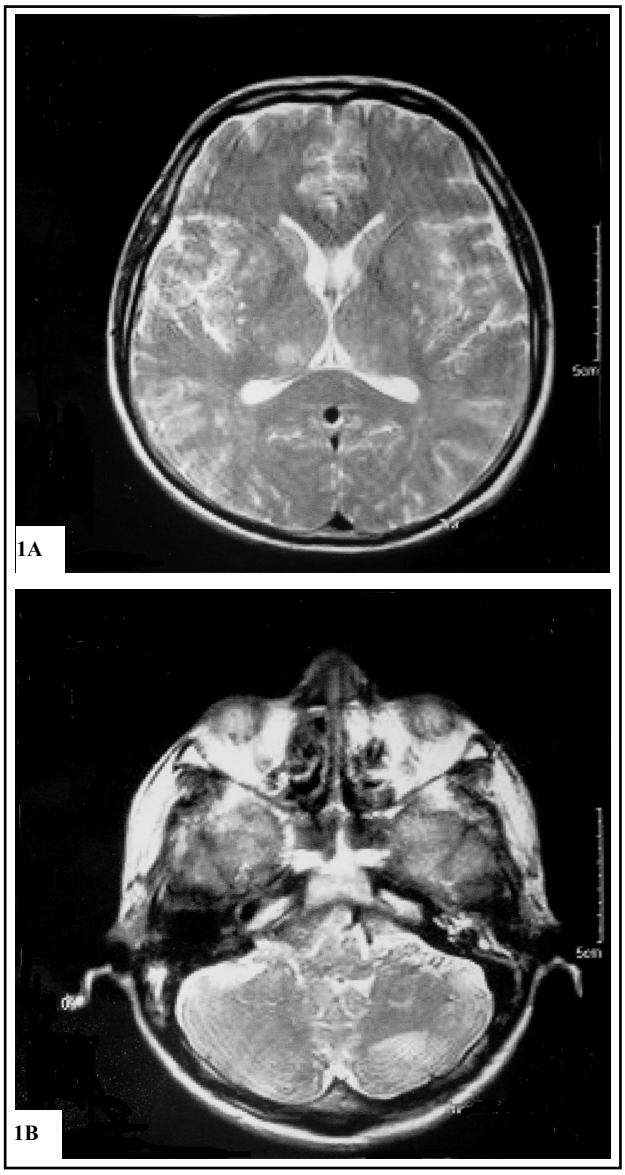

Figure 1: *The T2 W images of brain MRI of the patient show multiple hyperintense foci on bilateral the medial thalamus (A; thick arrows), and within the cerebellar white matter (B; thin arrows).*

DISCUSSION

Methanol is metabolized to formaldehyde and formic acid by alcohol dehydrogenase in the liver. It has been reported that these metabolites are responsible for the direct toxicity of methanol (7). It has been reported that common early symptoms of methanol intoxication are nausea, vomiting, abdominal pain and diminution of vision (7). The present case had nausea and vomiting on admission. The most common neurological sequela is visual impairment due to optic nerve damage from retrolaminar demyelination and probably also axonal damage (7). Our case developed visual impairment 21 hours after ingestion. A loss of consciousness has also been described and it may progress to obtundation, and coma (7). In methanol intoxication, while the optic nerve, the deep white matter such as the putamina, and the occipital and frontal lobes are more commonly involved, the cerebral cortex and cerebellar involvement are rare (7,9).

In WE, pathological changes affect the specific areas of the brain, such as the medial dorsal thalamic nucleus and mammillary bodies which are the strategic regions of memory, the hypothalamus, the superior vermis of the cerebellum, the periaqueductal region, the pontine tegmentum, the reticular formation of the midbrain, the posterior corpora quadrigemina, and the cerebral cortex (2,5). It has been reported that acute lesions caused by extreme rapidity of thiamine deficiency show a symmetrical distribution (5). In WE, histopathologic findings include loss of myelin, followed with axon damage, reactive gliosis, sometimes hemorragies in involved (1,2,5).

Common symptoms or signs at presentation of WE include ocular abnormalities, mental status changes, incoordination of gait and trunk ataxia (2,5). Abnormal eye movements are nystagmus, ophthalmoplegy due to often abducens palsy, and impairment of the conjugated eye movements (1,2,5). The WE patients with altered mental status may exhibite a disordered spontaneous speech, loss of memory, disorientation, loss of attention, and decreased awaking or lethargy (2). Our case was global amnestic at admission. A short time later, he exhibited altered mental status (acute confusional state). At same time, in his examination, we determined diplopia and horizontal nystagmus. Diplopia was related to asymmetric paralysis of lateral rectus muscle. Our case was presented with ataxic walking.

In the etiology of WE inadequate thiamine intake or deficiency underlies. Thiamine dependent enzymes include the α-ketoglutarate-dehydrogenase complex and the pyruvate-dehydrogenase complex in the tricarboxylic acid cycle, and transketolase in the pentose-phosphate pathway (5). It has been reported that thiamine also seems to have a role in acetylcholinergic and serotoninergic synaptic transmission, axonal conduction, and production of GABA (5). It has been reported that changes in serum osmosis of ethanol may lead to acute demyelination (10). Similarly, methanol may also be caused acute demyelination as a result of changes in serum osmosis (11). In the anamnesis of our patient there were no other diseases except chronic alcoholism. The patient had taken metylalcohol because he could not have etylalcohol for a few days. This syndrome may begin acute, subacute or chronic. For example it may begin acutely due to excessive glucose intake (4). Our case had a history of chronic alcoholism which may cause thiamine deficiency; however, before exposure to methanol intoxication, he had no symptoms or complaints relate to WE. Rotenstreich et al. (8) reported that vitamin B-1 might well be effective in methanol intoxication. We observed that vitamin B-1 was highly effective in our case with methanol intoxication. These reasons may explain the developing acutelly of WE in our case and improving rapidly from WE with thiamine of our case.

The diagnosis is clinical and is mainly supported by the dramatic response of neurological signs to parenteral thiamine (5). Magnetic resonance imaging supports diagnosis of WE (5). Because of that acute confusional status, ataxia, nystagmus, optic neuropathy, and amnesia followed methanol ingestion and it improved completely with thiamine therapy. Thus we thought that in our case, diagnosis was WE. Also the brain MRI findings were consistent with WE.

Finally, methanol intoxication may cause WE. Methanol induced WE may also be developed due to thiamine deficiency, direct toxic effects of methanol, or both of them at the same time. In a case of acute confusional status following the ingestion of methanol, WE should also be thought and early thiamine therapy should be started in ED.

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