



He who Sleeps with a Blind Man Will Wake Up Cross-Eyed: Wernicke's Encephalopathy

Körle Yatan Şaşı Kalkar: Wernicke Ensefalopatisi

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Dear Editor,

Wernicke's encephalopathy is characterized by nystagmus, abducens and conjugate gaze palsy, gait ataxia and mental confusion. Stupor and coma occur rarely. These symptoms may develop acutely or chronically. Although the disease is most commonly seen on the basis of chronic alcoholism, it may also be seen in hyperemesis gravidarum, systemic malignancy, gastrointestinal surgery, hemodialysis or peritoneal dialysis, longstanding intravenous nutrition, anorexia nervosa, dieting or hunger strike, and acquired immunodeficiency (1,2). Whether it is the result of inadequate intake, malabsorption or increased metabolic need, the main cause is thiamine deficiency due to nutritional status.

Pathologic changes developing due to Wernicke's encephalopathy are prominent particularly in the brainstem and hypothalamus, and the characteristic change is subtotal tissue necrosis including neuron, axon and myelin (3). While hyperintensity is seen in medial thalamic and periaqueductal gray matter on T2 and FLAIR sequences of cranial Magnetic Resonance Imaging (MRI) in acute cases, atrophy in mamillary bodies, enhanced signaling in the base of fourth ventricle, midline of cerebellum may be seen in chronic cases and contrast enhancement may occur in mamillary bodies and inferior quadrigeminal region (4,5). Hyperintensity either completely resolves or regresses with replacement therapy (6). However atrophy of mamillary bodies, superior cerebellar vermis and cortex may be permanent.

Only 20% of the cases which were diagnosed on autopsy could be diagnosed when alive (7). This is a worrisome condition.

The reasons for this may be that it doesn't show the classical triad composed of ataxia, ophthalmoparesis and encephalopathy, and the assumption of alcoholism as the cause. Wernicke's encephalopathy is progressive unless treated. Mortality varies between 10-20% despite thiamine treatment (1,3,6). While most ocular findings resolve with treatment within hours, nystagmus may heal with mild sequela. The course of healing of mental state cannot be predicted. Confusion and delirium may resolve within a couple of weeks. Memory failure, which is the most prominent symptom of Korsakoff syndrome, appears during this recovery period. While gait disturbance recovers slower, it may continue even after months in one-third or more of patients.

The 63-year-old male patient who was admitted to the emergency room with complaints of gait disturbance and altered consciousness had been using alcohol and cigarettes for 45 years. His symptoms started as severe nausea and vomiting one week ago. Upon examination, he was confused and somnolent. His pupils were myotic. The patient had bilateral nervus abducens paralysis and a prominent bilateral vertical nystagmus. He had dysarthria and could not walk without aid due to truncal ataxia. His hemogram, thyroid function tests, and vitamin B12 level were normal. Electroencephalography revealed encephalopathic changes, cranial MRI revealed signal enhancement in dorsal midbrain and periaqueductal area (Figure 1). The patient was diagnosed with Wernicke's encephalopathy and was given thiamine infusion. Ocular findings recovered in a short amount of time. A significant reduction was detected in the severity of vertical nystagmus. Cranial MRI findings also resolved (Figure 2).

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We think that it is important for both neurologists and psychiatrists, to show clinically and radiologically the effectiveness of thiamine treatment and to remember the typical cranial MRI findings of this disease with of high mortality rate and low recognition rates.

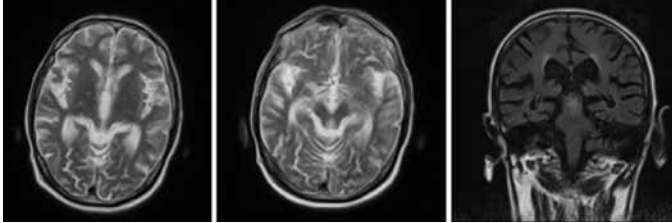


Figure 1. Cortical atrophy and signal enhancement in dorsal midbrain and periaqueductal area on cranial MRI

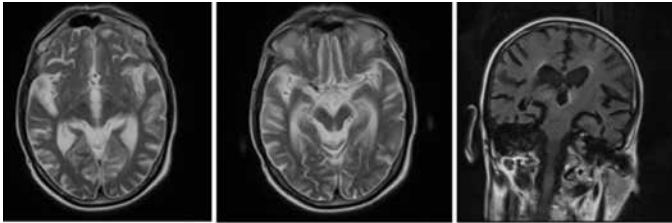


Figure 2. Enhanced signal disappeared on cranial MRI (FLAIR coronal and T2 axial series) following thiamine infusion (2 weeks later)

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