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A Case Report of Two Rare Diseases Misdiagnosed as Vertigo

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Abstract: ***Diziness is a common symptom that can be seen in various diseases. Mostly diziness is seen in vertigo, vestibular migraine and cerebellar syndromes. So most of rare diseases that could cause diziness can be misdiagnosed as vertigo. This situation could cause major problem especially for treatable diseases. In this case report, a Miller Fisher Syndrome (MFS) and Wernicke Encephalopathy patients presenting with diziness will be discussed.***

Keywords: Diziness; Vertigo; Ataxia

1. Objectives

Dizziness is a common symptom that can be seen in various diseases. Although dizziness is mostly seen in diseases such as vertigo, vestibular migraine and cerebellar syndromes, its rare causes should also be kept in mind. Because, some of rare causes of diziness can be treated if diagnosed early. In this case report, a MFS and Wernicke Encephalopathy (WE) patients presenting with diziness will be discussed. Informed consent was obtained from the patients.

Case 1

A thirty - two year - old male admitted to emergency service with complaints of dizziness and gait instability. Ataxia was detected in his neurological examination. Brain computed tomography (CT) and diffusion - weighted cranial magnetic resonance imaging (MRI) were normal. Considering vertigo, the patient was discharged with betahistine treatment. The patient was admitted to neurology outpatient clinic two days later because of progression in his complaints. He also had diplopia. When questioned in detail, he described numbness in the hands and feet. In his neurological examination he had nystagmus on lateral gaze, glove stocking hypoesthesia, hyporeflexia and ataxia. He had history of upper respiratory tract infection about two weeks ago. Patient admitted to neurology service. Sixth cranial nerve palsy developed one day later. In his electromyographic examination F wave could not be obtained in lower extremities. F wave latency was prolonged in the upper extremities. Protein was high (74, 68 miligram/ decilitre) in his cerebrospinal fluid (CSF) with no cell. His CSF gram/wriht staining and culture was negative. The patient's blood ganglioside panel was studied. The patient was administered 0, 4 milligram/kilogram/day intravenous immunoglobulin (IVIG) for five days. GD2 immunoglobulin G (IgG), GD3 IgG, GT1a IgG and GQ1b IgG antibodies were positive in his blood. On the seventh day of IVIG treatment, his neurological examination was completely normal. He had no complain about numbness. He discharced from hospital with recovery.

MFS is a rare autoimmune disease with ataxia, areflexia and ophthalmoplegia. It is a variant of Guillain Barre syndrome (GBS) that often preceded by viral or bacterial infection. (1). Different type of antiganglioside antibodies can be seen in different variants of GBS. (2). Especially GQ1b IgG antibody positivity commonly found in MFS. İt is present in oculomotor nerves and dorsal ganglia. So opthalmoplegia and sensory ataxia common findings in MFS.

Our patient had typical triad of MFS; ataxia, opthalmoplegia and hyporeflexia. Also, he had typical CSF (albuminocytological dissociation) and serology (antiganglioside antibody positivity) findings. However, without proper anamnesis, the patient may be considered to had vertigo and may not receive the necessary treatment. Since MFS is a disease with a good prognosis with treatment, early diagnosis is important.

Case 2

Fifty-two-year-old women admitted to emergency service with sudden onset diziness and ataxia. Brain CT and diffusion weighted MRI were normal. When she questioned in detail, she had binocular diplopia and history of alcoholism. She had nystagmus on lateral gaze and ataxia. So patient was admitted to neurology service with a preliminary diagnosis of WE. Thiamine 3x500mg/day for two days than 1x500mg/day for five days is started for treatment. Second day of treatment left abducens nerve palsy was developed. She was agitated and her orientation and cooperation was limited. Also she had electrolyte imbalance (hyponatremia, hypokalemia, hypomagnezemia). Lactic acid level was normal. No pathology was observed in patient's brain MRI. Blood thiamine level was low (15, 5 mkirogram/dL). After eight days, she was oriented and cooperated. She had no opthalmoparesis, nystagmus or ataxia. Patient discharced from hospital with thiamine maintenace treatment.

WE is rare disease presented with diplopia, opthalmoparesia, ataxia and encephalopathy. It is caused by thiamine deficiency. Its common ethiology is alcoholism but it can occur in patients with any nutritional deficiency (hyperemesis gravidarum, intestinal obstruction, malignancy, hemodialysis) (3). Only few cases of WE diagnosed before development of Korsakoff Syndrome or death (4). Even the syndrome is mostly recognized at autopsy (3). Many

cognitive changes (apathy, amnesia, disorientation, coma) and different types of ocular symptoms (opthalmoplegia, horizontal nystagmus, sixth cranial nerve palsy, pitosis, anisocoria, miosis, papilledema) could be seen in WE. Also, patients presented with hypothermia, hypotension or coma. Korsakoff syndrome consist of memory loss with confabulation. It could be seen after untreated WE (3).

Ass in our patient classic triad is seen. This triad is more common in alcoholic WE patients than non - alcoholics. (4). In Brain MRI hyperintensity in T2 and hypointense lesions in T1 around aqueduct, third ventricle, medial talamus, dorsal medulla and mamillary bodies. MRI is normal in 1/3 of the patients. So even with normal MRI WE must be taken into consideration.

2. Conclusion

Diziness is common symptom that can be seen in various diseases. Although both diseases in the case report presented with typical clinical symptoms, they were evaluated as vertigo in the emergency department. Therefore, patients presenting with dizziness should be questioned in detail for additional symptoms. Also, rare causes should always be taken into consideration.

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