Case Report

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Central Pontine Myelinolysis Induced by Alcohol Withdrawal: A Case Report

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Central pontine myelinolysis (CPM) is a demyelinating disorder characterized by the loss of myelin in the center of the basis pons, and is mainly caused by the rapid correction of hyponatremia. We report the case of a young woman who presented with gait disturbance and alcohol withdrawal, and who was eventually diagnosed with CPM. Generally, the cause and pathogenesis of CPM in chronic alcoholics remain unclear. In this cases, the CPM may be unrelated to hyponatremia or its correction. However, it is possible that the osmotic pressure changes due to refeeding syndrome after alcohol withdrawal was the likely cause in this case. This case illustrates the need for avoiding hasty, and possibly incomplete diagnoses, and performing more intensive test procedures to ensure a correct diagnosis.

Keywords Central pontine myelinolysis, Alcohol withdrawal

INTRODUCTION

Central pontine myelinolysis (CPM) is a demyelinating disorder characterized by the loss of myelin in the center of the basis pons. Myelin loss is primarily caused by the rapid correction of hyponatremia [1]. There are many reports of CPM caused by severe liver disease, burns, and anorexia [2]. However, there are only a few cases of CPM induced by alcohol withdrawal [1,3]. We report a case of CPM due to alcohol withdrawal in a young woman without specific electrolyte corrections.

CASE REPORT

A 34-year-old woman was admitted to the hospital with an abrupt gait disturbance for 4 months. The patient had no history of seizure, head injury or previous hospitalizations for mental illness, drug or alcohol use. However, she had a >10 year history of chronic alcoholism and typically drank two bottles of spirits daily. One week prior to the onset of her symptoms, she went on an alcohol binge, during which she ate very little food. She attributed her binge to anxiety and a depression. Three days

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after stopped drinking, she had a meal. After that she developed gait disturbances.

Upon hospital admission, the patient was alert and well oriented. Her cranial nerve examination was unremarkable. Optokinetic nystagmus was intact, and no other nystagmus was noted. Cerebellar testing revealed intact dysdiadochokinesia, and finger-to-nose performance. However, the patient had bilaterally impaired tandem gait and difficulty with the heel-to-shin task, bilaterally. Her deep tendon reflexes were symmetric and normoactive. The Babinski response was absent bilaterally. Her gait was wide-based with unsteady, irregular steps. Sensory examination revealed intact pain, temperature and vibratory senses. However, proprioception was impaired. The patient also had hyperalgesia in both lower limbs and tingling sensations on the distal upper limbs bilaterally. A manual motor examination revealed grade 3/5 muscle strength in both lower limbs. There were no adventitious movements or tremors.

The laboratory tests on admission revealed a normal blood cell count and normal serum electrolytes. The renal function was within the normal range. The laboratory tests for alcohol-induced brain injury, such as Wernicke encephalopathy or hepatic encephalopathy, were performed. All of these test results, including vitamins B_{12}, B_1, and B_6, ammonia and albumin, were within normal limits. The liver enzymes were slightly elevated (serum aspartate aminotransferase [AST], 83 IU/L; alanine aminotransferase [ALT], 40 IU/L).

A magnetic resonance imaging (MRI) scan of the brain revealed an ill-defined, patchy, trident shaped and mild high signal intensity lesion in the pons. This was highly suggestive of CPM (Fig. 1). Except for this lesion, the MRI scan revealed no other significant focal lesions.

A nerve conduction study (NCV) revealed an absence of sensory nerve action potentials (SNAPs) in both the superficial peroneal and left sural nerves. In addition, the SNAP amplitude in the right sural nerve was markedly reduced. Other forms of motor nerve conduction and needle electromyography (EMG) were within normal limits. These findings were compatible with mild axonal sensory peripheral polyneuropathy (Table 1).

In summary, a patient with chronic alcoholism presented with ataxic gait, impaired proprioception, hyperalgesia in both lower limbs, and tingling sensations on the distal parts of both upper limbs. An MRI of her brain revealed a trident-shaped median pontine lesion. The NCV-EMG study demonstrated mild axonal sensory peripheral polyneuropathy. The patient's overall findings were compatible with CPM induced by alcoholism.

The patient was treated with conventional medical treatments, including vitamin supplementation and formal nutritional support. In addition, she underwent rehabilitation, including Frenkel exercises, proprioceptive neuromuscular facilitation and gait balance training.

After 1 month, the patient's functionality improved. The muscle power in the both of her lower limbs increased to grade 4 (good). She began to ambulate with a cane and perform all activities of daily living independently. The patient was discharged with outpatient follow-up.

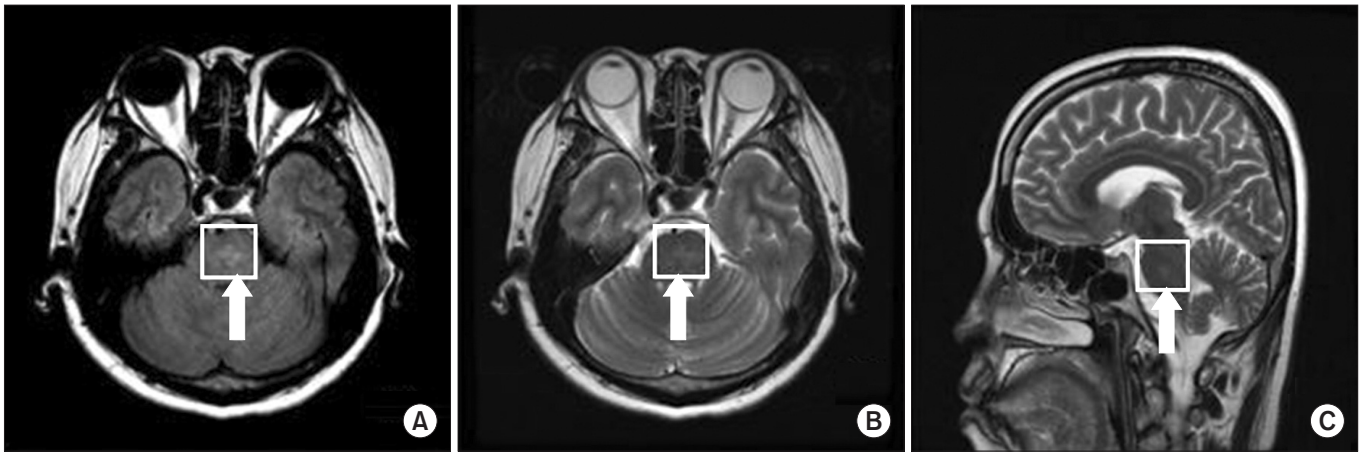

Fig. 1. Brain magnetic resonance imaging (MRI) scan at the level of the pons: T2 FLAIR image (A), T2-weighted image (B), and T2-weighted sagittal image (C). MRI scan demonstrates an ill-defined, patchy, trident-shaped, mild high signal intensity lesion in the pons (arrow). Otherwise, no significant focal lesions were noted.

Table 1. Nerve conduction studies in the upper and lower extremities bilaterally

Nerve stimulation (record)	Amplitude[a]		Conduction velocity (ms)	Distance (cm)	Latency	
	Distal	Proximal			Distal	Proximal
Motor						
Rt. median (APB)	15	10.1	51	23.0	3.2	7.7
Rt. ulnar (ADQ)	8.2	8.5	58	25.0	2.3	6.6
Lt. median (APB)	8	7.8	53	23.0	3.3	7.6
Lt. ulnar (ADQ)	7	6.3	56	24.0	3.7	7.4
Rt. tibial (AH)	8.3	5.9	40	39.0	3.5	13.3
Rt. peroneal (EDB)	2.9	2.7	44	34.0	4.1	11.9
Lt. tibial (AH)	8.4	5.4	46	39.0	4.6	13.0
Lt. peroneal (EDB)	3.4	3.3	43	33.0	3.9	11.6
Sensory						
Rt. median (2nd digit)	22		63	14.0	2.2	2.9
Rt. ulnar (5th digit)	38		61	11.0	1.8	2.5
Lt. median (2nd digit)	35		59	14.0	2.4	3.0
Lt. ulnar (5th digit)	36		54	12.0	2.2	2.8
Rt. superficial peroneal (ankle)	Not evoked					
Lt. superficial peroneal (ankle)	Not evoked					
Rt. sural (ankle)	4		47	9.0	1.9	2.3
Lt. sural (ankle)	Not evoked					

APB, abductor pollicis brevis; ADQ, abductor digiti quinti; AH, abductor hallucis; EDB, extensor digitorum brevis.
[a]Amplitudes are measured in millivolt (mV, motor) and in microvolt (μV, sensory).

DISCUSSION

The most common cause of CPM is a too rapid correction of low sodium levels (hyponatremia). During prolonged hyponatremia, the brain can remain relatively isotonic with its surroundings by decreasing the intracellular levels of these osmolytes. When hyponatremia is corrected with intravenous delivery of fluids, the extracellular tonicity increases, and this is followed by an increase in intracellular tonicity. However, if the serum sodium levels rise too rapidly, and insufficient time is allowed for the brain to adjust to the new tonicity, the increased extracellular tonicity will cause osmolytes to continue moving out of brain cells into the extracellular space. This can lead to the condition of CPM, where the myelin sheath surrounding the nerve axons becomes damaged in the part of the brain called the pons [4]. However, a few cases of CPM have been reported in patients suffering from alcohol withdrawal. In these cases, the CPM may be unrelated to hyponatremia or its correction. Previous reports have suggested that CPM from alcohol withdrawal has a better prognosis than CPM from a too rapid correction of hyponatremia [5].

The diagnosis of CPM is initially determined from clinical symptoms and is confirmed by magnetic resonance imaging of the brain [6]. The neurologic sign associated with CPM in this patient was gait disturbance. This gait disturbance was quite similar to the ataxic gait associated with alcohol withdrawal syndrome in chronic alcoholism. Alcohol withdrawal syndrome can last for up to 1 week after having the last alcoholic beverage. In alcohol withdrawal syndrome, patients exhibit various symptoms like hand tremor, insomnia, nausea, transient hallucinations, psychomotor agitation, anxiety, tonic-clonic seizures, and autonomic instability consistent with a response of the broad central nervous system [7]. However, this patient was young and only showed gait disturbance with loss of smoothly coordinated voluntary movement. Thus it can be distinguished from alcohol withdrawal syndrome in view of the intact orientation and the absence of visual hallucinations and other alcohol withdrawal symptoms. Additionally MRI findings of a focal, trident-shaped median pontine lesion indicated a likely diagnosis of CPM rather than alcohol withdrawal syndrome.

Although CPM may not be associated with an acute correction of hyponatremia, in chronic alcoholics, CPM is often associated with severe liver disease and Wernicke encephalopathy [8]. However, these were ruled out in this case. The patient presented here was too young to have developed chronic alcoholic brain injury. It has been suggested that chronic alcoholics may not be able to maintain protective cerebral mechanisms against osmotic stress, in addition to the effects of the direct toxicity of alcohol during the course of alcohol withdrawal [9]. In addition, women are more sensitive to the harmful effects of alcohol abuse than men are. According to the National Institute on Alcohol Abuse and Alcoholism, women are more vulnerable than men to alcohol-related brain damage. Women have more adipose tissue, but typically weigh less than men. Therefore, alcohol tends to be absorbed more slowly in woman than in men, allowing its effects to last longer [10]. Women also have less water in their bodies to dilute the alcohol, and lower concentrations of the enzymes that metabolize it compared to men.

Although CPM is commonly attributed to the rapid correction of hyponatremia, there are case reports of central pontine myelinolysis without associated hyponatremia [3]. Some cases of CPM without rapid correction of hyponatremia were associated with osmotic pressure changes due to refeeding syndrome [3]. Refeeding syndrome is a metabolic disturbance that occurs as a result of reinstitution of nutrition to patients who are starved, or severely malnourished, and show electrolyte disorders, especially hypophosphatemia, along with neurologic complications. In these instances, a rapid increase in extracellular osmotic pressure leads to pons damage [3]. The patient described here binged on alcohol and fasted for an entire week prior to her presentation. And 3 days after stopped drinking, she had a meal. After that she developed gait disturbances. This was similar to a refeeding syndrome occurring within 10 days of starting to feed. If there was no evidence of electronic disorder at admission, we could not rule out the possibility of a refeeding syndrome. There was a limitation in diagnosing an electronic disorder because the first laboratory study was done 4 months after symptoms began. During a period of prolonged starvation, several intracellular minerals become severely depleted. During refeeding, glycaemia leads to increased insulin stimulating of glycogen, fat, and protein synthesis. These processes cause a decrease in the serum levels of several minerals As a result of electrolyte deficits, a rapid change in basal metabolic rate occurs. In this patient, after feeding resumed, a rapid osmotic pressure change may have been caused by the rapid change in basal metabolic rate. The rapid osmotic stress may cause brain shrinkage and shearing of oligodendrocyte processes, initiating myelin damage. Therefore the myelin sheath surrounding the nerve axons becomes damaged in the part of the brain called the pons. This damage was confirmed by brain MRI imaging.

This case described a patient with gait disturbance which could have been associated simply with alcohol withdrawal. However, it ultimately was diagnosed as CPM by MRI examination. The case demonstrates the importance of intensive diagnostic studies to make a correct diagnosis in such cases. Moreover, a correct diagnosis of CPM would lead to proper rehabilitation programs which are critical for patients with CPM secondary to chronic alcoholism.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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