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Alcohol-Induced Persistent Mild Cognitive Impairment with Successful Withdrawal from Alcohol Dependence - A Case Report

Toshihide HARADA[1,*], Fumiko ISHIZAKI[1], Nobuko HORIE[2], Hiroyuki KATSUOKA[3], Yumiko NITTA[4], Tohru YAMADA[5], Kohsaku NITTA[6] and Makoto ITO[6]

1) Faculty of Health and Welfare, Prefectural University of Hiroshima, 1-1 Gakuen-machi, Mihara,Hiroshima 723-0053, Japan
2) Department of Neurology, Hiroshima-Nishi Medical Center, Ohtake, Hiroshima 739-0696, Japan
3) Faculty of Health Services Management, Hiroshima International University, Hiroshima 730-0016, Japan
4) Suzugamine Women's College, Hiroshima 733-8623, Japan
5) Yamada Memorial Hospital, 6-2-1, Miyaura, Mihara, Hiroshima 723-0051, Japan
6) Shiraki-no-sato, 230 Shiraki-Cho Kogoshi, Asakita-ku, Hiroshima 739-1412, Japan

ABSTRACT

An 81-year-old man diagnosed with alcohol-induced persistent mild cognitive impairment consulted our clinic presenting with gait disturbance. Between the ages of 20 and 53 years, his alcohol consumption was 1.8 liters of alcoholic sake per day. However, from the age of 53 years onward, his consumption decreased to 360 ml per day. The patient had alcoholic neuropathy, mild cognitive impairment, and alcoholic cerebellar disorder. His score on the revised version of Hasegawa's Dementia Scale (HDS-R) was 22 and his clinical dementia rating (CDR) was 0.5. His score on the Japanese version of the Mini-Mental State Examination (MMSE) was 22. These scores indicated mild cognitive impairment (MCI). He had delusions and confabulations, without impairment of date and place orientation. Magnetic resonance imaging (MRI) demonstrated enlarged ventricles, sulcal widening, and brain atrophy. He was provided with medication and counseling to treat his alcohol abuse. He accepted our treatment and is presently doing well after 1 year 2 months of treatment.

Key words: *Alcoholism, Mild cognitive impairment (MCI), Mini-Mental State Examination (MMSE)*

Alcoholism is a serious social issue[2-12]. Alcohol abuse induces dementia, liver disease, neurological disorders, and other diseases. Alcohol abuse also leads to health, family and social problems. The main problem with alcohol abuse is that it is very difficult to withdraw. Early-onset alcoholics reported being intoxicated twice as often as late-onset subjects, expected less success from the treatment program, and were less likely to complete treatment[4]. It is very unusual to find a case of an elderly person with alcohol-induced persistent mild cognitive impairment who successfully withdrew from alcoholism. Successful withdrawal from alcohol dependence is rare, especially in the elderly[3,6,8,12]. This case is described herein with a review of the literature.

CASE REPORT

An 81-year-old male consulted the neurology department of our hospital complaining of gait disturbance and cognitive impairment on July 11, 2009. Between the ages of 20 and 53 years, his alcohol consumption was 1.8 liters of alcoholic sake per day, and from the age of 20 to 81 years, he smoked 20 cigarettes/day. He worked as an executive clerk. After retirement, he maintained an active social life. His family history was non-contributory. The patient's clinical history included a nasal allergy and insomnia, but he was mentally stable. In March 1981, at 53 years of age, he consulted a physician because of a difficulty in walking. Cerebral infarction was suspected, and ticlopidine hydrochloride was prescribed. He was introduced to the neurology department and was advised to stop drinking. He reduced the amount of alcohol to approximately 360 ml per day. However, his gait disturbance persisted. He visited the neurology department regularly, and his insomnia was brought under control. However, his

*Address correspondence to: Dr. Toshihide Harada
E-mail: hartoshi@pu-hiroshima.ac.jp

alcohol consumption remained the same until his retirement age. The gait disorder that made his walking unstable and caused him to stumble had appeared in 2004 (age 76 years). Furthermore, an unusual sensation while walking on sand (a sense that the skin on his foot soles had thinned) and scorching of both foot soles developed and persisted. At the beginning of 2009, he began to fall often.

Physical examination combined with a routine general and vascular examination revealed a height of 160 cm, a body weight of 59 kg, a blood pressure of 126/80 mmHg, and a regular heart rate of 78 beats/min. He showed good date and place orientation. He had a short-term memory impairment, but retained older memories. He showed no aphasia, apraxia, agnosia, or depression; he was an optimist. He had optical illusions, delusions, and confabulations. He showed akinesia and a wide-based gait that was ataxic and slow. His eyesight and eyelids were normal, although horizontal and vertical eyeball movement was limited. He had slight nystagmus with a horizontal gaze. He showed no whisper, deglutition trouble, or mask-like facial expression, but his speech was slurred. His pharyngeal reflex was normal, and he had no tongue deviation or shrinkage. He showed slightly uncoordinated movements during the bilateral finger-to-nose test. Adiadochokinesis was slightly positive. He had no dysosmia but experienced a slight loss of hearing. His jaw reflexes were normal, whereas his tendon reflexes were slightly diminished. His pathological reflex was negative. Manual muscle testing was 4/5 in the extremities and body, without muscle atrophy. He showed impaired coordination of limbs but no muscle rigidity. "Gegenhalten" was observed. There was a tremor in both hands. The superficial and vibration sensations decreased in both lower limbs, with an unusual sensation in both foot soles. Bilateral palmar jaw reflexes were positive. He suffered from urinary incontinence and orthostatic hypotension. His blood pressure in the supine position was 152/80 mmHg, which reduced to 112/60 mmHg in the standing position. The hemoglobin A1c level was 5.1%, with normal hematological and biochemical screening otherwise. The post-meal blood sugar level was 113 mg/dl. Thyroid hormone, folic acid, vitamin B1 and B12 levels were normal. The syphilis reaction was negative. His score on the revised version of Hasegagwa's Dementia Scale (HDS-R) was 22, and his clinical dementia rating was 0.5. The score on the Japanese version of the Mini-Mental State Examination (MMSE) was 22, which indicated mild cognitive impairment (MCI). Magnetic resonance image demonstrated moderate atrophy of the cerebral cortex, the white matter and the entire cerebellum. The bilateral ventricles and the fourth ventricle were enlarged, with a high intensity area around the ventricle, as observed in a fluid attenuated inversion recovery (FLAIR) image. Sulcal widening was also found. No major lacunar infarction was found, with normal magnetic resonance angiography. These findings were consistent with long term consumption of a large amount of alcohol. Neurological disorders, such as autonomic and cerebellar disturbances, were thought to be the result of chronic alcohol abuse. Other dementia (neurosyphilis, hypothyroidism, cerebrovascular disorder, depression, brain tumor, hydrocephalus, encephalitis, and Parkinson's disease) were negative from the results of diagnostic imaging and hematological examination. We explained the possibility of an alcoholic neurological disorder to the patient and his family, and suggested that he should abstain from alcohol. Vitamins B1, B12, and E were administered regularly.

Many reports demonstrate amantadine hydrochloride-induced delirium. The usefulness of amantadine hydrochloride as an anti-parkinson agent is somewhat lessened by the need to screen patients for a history of seizures and psychiatric symptoms. Amantadine hydrochloride has many effects on the brain, including the release of dopamine and norepinephrine from nerve endings. It appears to act as a weak N-methyl-D-aspartate (NMDA) receptor antagonist as well as an anticholinergic agent[1]. It has been suggested that the central nervous glutamatergic system, with its NMDA receptors, is involved in toxic neuronal loss due to an increased glutamatergic neurotransmission during repeated alcohol withdrawal, and that it may subsequently contribute to the development of alcohol dementia[8]. When amantadine hydrochloride (100 mg/day) was administered with the hope of mental function activation, the gait disorder ended and there was a concomitant decrease in falls. However, amantadine hydrochloride-induced delirium started appearing and we stopped the amantadine hydrochloride. In May 2010, the patient had an unstable ataxic gait, but no optical illusions. He discontinued drinking alcohol. 10- months after discontinuation of alcohol his functions had not weakened and the HDS-R was still 22. He lives with his family and has chosen to use a nursing service.

DISCUSSION

Alcohol-induced persistent dementia is a rare disease that is sometimes associated with Wernicke-Korsakoff syndrome[2-4,6,8]. The central nervous glutamatergic system, with its NMDA receptors, is suggested to be involved in toxic neuronal loss due to increased glutamatergic neurotransmission during repeated alcohol withdrawal, and may subsequently contribute to the development of alcoholic dementia[8]. Recently, the diagnostic criteria

of Oslin were used to discuss alcohol-induced persistent dementia[6,7].

Despite its clinical importance, few studies have been published on successful alcohol withdrawal in elderly patients who maintain an active lifestyle[8]. A short report was published on a 71-year-old female patient with chronic alcoholism for at least 30 years, who was treated with memantine and showed improvements in MMSE and Consortium to Establish a Registry for Alzheimer's Disease Verbal Fluency tests[8]. We also treated our patient with vitamins B1, B12, and E regularly, but his score on HDS-R did not improve. Preuss's case showed improvement in the MMSE, Consortium to Establish a Registry for Alzheimer's Disease Verbal Fluency scores, and 18F-fluorodeoxyglucose-positron emission tomography of glucose metabolism after memantine treatment[8]. Parallel to the psychological testing, 18F-fluorodeoxyglucose-positron emission tomography was performed 1 week after admission and 5 weeks after starting the memantine treatment, as extensively described elsewhere[8]. Significant changes in cerebral blood flow after 5 weeks of memantine treatment were revealed using 18F-fluorodeoxyglucose-positron emission tomography, and these changes correlated with moderate improvements in neuropsychological testing. The same could not be demonstrated in our case. However, despite these findings and improvements in some intellectual and memory functions, severe memory deficits and disorientation persisted in Preuss's case[8]. Continued inpatient care was recommended for their patient but no deterioration in mental abilities was observed even 1 year after discharge[8]. Our patient showed successful withdrawal from alcohol addiction and performed his day-to-day activities at a day-care center. This is the first report of successful treatment of alcohol-induced persistent mild cognitive impairment in an 81-year-old patient. To our knowledge, this is also the first report on day-care for this type of patient. This is the only study of its kind in Japan but has promising outcomes for further research because there is no evidence for the hypothesis that low-affinity NMDA antagonists have a neuroprotective effect even in patients with alcoholic dementia treated with memantine. Withdrawal from alcohol addiction might yield benefits in patients with alcohol-induced persistent dementia.

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