

Progressive Brain Atrophy due to Chronic Alcohol Abuse

Gleb Slobodin MD and Majed Odeh MD

Department of Internal Medicine A, Bnai Zion Medical Center, affiliated with Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

KEY WORDS: alcohol abuse, neurotoxicity, brain atrophy, dementia

IMAJ 2015; 17: 659

A 43 year-old man was brought to the emergency room with head trauma following a fall. His medical history was significant for heavy alcoholism since age 35 and related falls and injuries. Neurological examination demonstrated significant memory impairment, dysphasia, apraxia, agnosia and disturbance in executive functioning, leading to the diagnosis of alcoholic dementia. Computed tomography (CT) scan of his head repeated during 6 years demonstrated progressive central and peripheral brain atrophy with sulcal widening, ventricular enlargement, and cortical white matter shrinkage characteristic of chronic alcohol abuse [Figure 1].

Long-standing heavy alcohol abuse leads to disproportionate loss of white matter with reduction in brain volume and brain weight.

The most commonly reported findings in the brains of alcoholics are sulcal widening and ventricular enlargement as well as cortical white matter shrinkage. The frontal lobes appear to be more vulnerable to alcohol-related brain damage than other cerebral regions. The cerebellum, cortical-limbic circuits, skeletal muscle, and peripheral nerves are also important targets of chronic alcohol-related metabolic injury and degeneration [1-3]. Although all cell types within the nervous system are vulnerable to the toxic, metabolic and degenerative effects of alcohol, astrocytes, oligodendrocytes and synaptic terminals are major targets, accounting for the white matter atrophy [4].

A heavy drinking history is a cardinal feature in the criteria for alcoholic dementia, which include memory impairment in addition to one or more other cognitive symptoms. Among the cognitive symptoms are dysphasia, apraxia, agnosia, and disturbance in executive functioning [3]. All these features were found in our patient and were persistent for at least 6 months of follow-up.

Alcohol abuse is a very serious health, social and economic problem worldwide. The present case conveys a clear message regarding the brain damage that results from chronic alcohol abuse.

Correspondence

Dr. M. Odeh

Dept. of Internal Medicine A, Bnai Zion Medical Center, Haifa 33394, Israel

Phone: (972-4) 835-9781

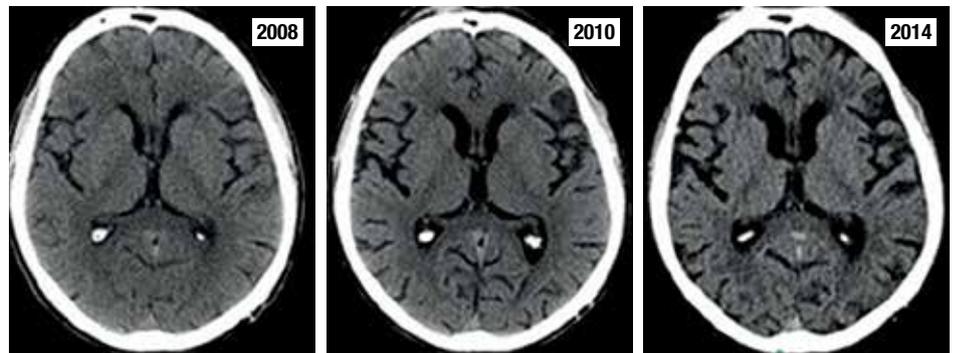
Fax: (972-4) 835-9790

email: majed.odeh@b-zion.org.il

References

1. Paul CA, Au R, Fredman L, et al. Association of alcohol consumption with brain volume in the Framingham study. *Arch Neurol* 2008; 65: 1363-7.
2. García-Valdecasas-Campelo E, González-Reimers E, Santolaria-Fernández F, et al. Brain atrophy in alcoholics: relationship with alcohol intake; liver disease; nutritional status, and inflammation. *Alcohol Alcohol* 2007; 42: 533-8.
3. Vetreno RP, Hall JM, Savage LM. Alcohol-related amnesia and dementia: animal models have revealed the contributions of different etiological factors on neuropathology, neurochemical dysfunction and cognitive impairment. *Neurobiol Learn Mem* 2011; 96: 596-608.
4. De La Monte SM, Kril JJ. Human alcohol-related neuropathology. *Acta Neuropathol* 2014; 127: 71-90.

Figure 1. CT scan of the brain, repeated during 6 years, showing progressive central and peripheral brain atrophy



“I’d rather see a sermon than hear one any day; I’d rather one should walk with me than merely tell the way”

Edgar Guest (1881-1959), prolific English-born American poet who was popular in the first half of the 20th century and became known as the ‘People’s Poet’