Central Pontine Myelinolysis after Alcohol Withdrawal

Isabelle Korn-Lubetzki MD[1], Yelena Virozub MD[1] and Hedi Orbach MD[2]

[1]Neurological Service and [2]Department of Internal Medicine, Bikur Cholim Hospital, Jerusalem, Israel

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Because many drinkers occasionally imbibe to excess, temporary alcohol-related pathology is common in non-alcoholics. We describe a rarely reported complication.

Patient Description

A 31 year old woman, a recent immigrant from Russia, had been episodically drinking huge quantities of vodka for years. The last binge was 2 months before admission. A few days afterwards she suffered from delirium tremens. A month later, right lower lobe pneumonia was diagnosed. Serum sodium level at that time was 141 mEq/L. Shortly after and for several weeks, she progressively developed urinary incontinence and increasing difficulties in walking and speaking. Three days before hospitalization she experienced weakness in her right hand.

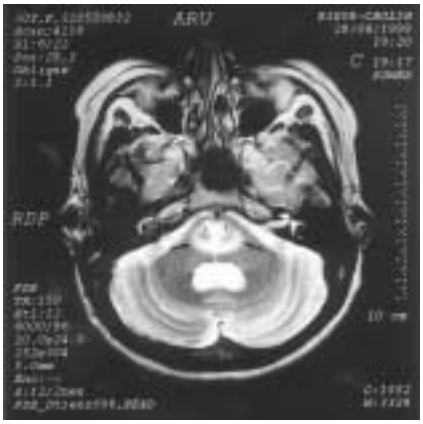
On examination, she was fully oriented and responsive. The neurologic examination revealed dysarthria, jerky nystagmus to the left, pseudobulbar palsy, flaccid quadriparesis that was more pronounced on the right side with bilateral symmetric hyperreflexia, clonus of the patella, positive Tremner and Babinski signs, and impaired vibration sensation over the legs.

Blood (sodium 137 mEq/L), urine and spinal fluid tests including vitamin B12, folic acid, antinuclear factor, antineutrophil cytoplasmic antibodies, rheumatoid factor, C-reactive protein, VDRL, serology for human immunodeficiency and hepatitis virus, were normal. Magnetic resonance imaging demonstrated central pontine myelinolysis [Figure]. The patient's condition improved slowly and spontaneously. At follow-up 2 months later the neurologic examination was normal.

Comment

Central pontine myelinolysis was identified by Victor and Adams in Boston 50 years ago [1], but it remains poorly understood. Although initially considered as rare, its prevalence can be as high as 3/1,000 in an unselected urban hospital population group [2]. In more than half the cases it appeared in the late stages of chronic alcoholism, often in association with Wernicke disease and polyneuropathy. Among other medical conditions conjoined with CPM are dialysis, liver failure and transplantation, advanced lymphoma, carcinoma, cachexia from various causes, severe bacterial infections, dehydration and electrolyte disturbances, acute hemorrhagic pancreatitis, and pellagra [1,2].

Nutritional deficiency is a commonly evoked cause of CPM, often observed in chronic wasting disease and particularly in malnourished alcoholics [1]. Rapid corrections of sodium level, mostly hyponatremia, is a well-recognized factor predisposing to CPM [1,2] and has been demonstrated experimentally in dogs [3]. Conventionally this has been perceived to lead to pontine glial cell swelling through osmosis and eventually to cell death [2]. Extreme serum hyperosmolality, also evoked as a pathophysiologic rationale for CPM development, can explain the CPM cases found in severely burned normonatremic patients [4].



Axial T2 weighted MRI scan reveals asymmetric area of abnormal high signal intensity within the basis pontis (large arrows), with relative preservation of the corticospinal tracts (thin arrows).

Our patient, however, could not be considered as a chronic alcoholic since she had been drinking only episodically. She was not malnourished and did not suffer from a chronic disease. In this case, myelinolysis was a late symptom of alcoholic withdrawal. Asharafian and Davey [2] propose a novel hypothesis for CPM, suggesting that individuals predisposed to CPM have substantial disturbances in one of the regulatory systems of apoptosis, particularly those pertaining to energy provision. Excess production of free radicals and deranged nitric oxide metabolism – recognized metabolic effects in alcohol abuse – may favor apoptosis [2].

Recognition of the CPM syndrome, which relies on a high index of suspicion and on appropriate clinical findings, is supported by computerized tomography but the imaging modality of choice is magnetic resonance imaging [5].

References

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Correspondence: Dr. I. Korn-Lubetzki, Neurological Service, Bikur Cholim Hospital, P.O. Box 492, Jerusalem 91004, Israel.
Phone: (972-2) 646-4220/217
Fax: (972-2) 646-4272
email: ikl@md.huji.ac.il

CPM = central pontine myelinolysis