To the Editor: Central pontine myelinolysis (CPM), a form of osmotic demyelination syndrome (ODS) typically occurring in the central pons, was first described in alcoholic and malnourished individuals by Adams et al.[1] in 1959. ODS is frequently associated with alcohol abuse, liver transplantation, and rapid correction of hyponatremia.[2] The clinical manifestations of CPM are variable, including dysarthria, dysphagia, oculomotor dysfunction, variable degrees of quadripareisis and even locked-in syndrome. In addition, some patients may be asymptomatic. The size of myelinolysis in the central pons on brain magnetic resonance imaging (MRI) is not always matched with the severity of clinical symptoms. Previously, a limited number of case reports focused on the magnetic resonance spectroscopy (MRS) findings of CPM. Here, we report a case of CPM and emphasize the importance of MRS.

A 45-year-old man with known long-standing alcohol abuse (a daily average of 300 mL Chinese liquor for 20 years) presented with slurred speech and gait disturbance for 2 weeks. On admission, his vital signs were a temperature of 36.5°C, a heart rate of 72 beats per minute, and blood pressure of 124/70 mmHg. Neurological examination revealed dysarthria, ataxia of trunk and four limbs, and lower extremity weakness (Medical Examination revealed normal findings. Based on previous long-standing history of alcohol abuse and typical MRI findings, the patient was diagnosed with CPM. He was administrated with high-dose multivitamins as well as nutritional support. His lower limbs weakness improved moderately without improvement of dysarthria and he was discharged on day 22. Unfortunately, the patient did not receive follow-up MRI examination.

Myelinolysis can be found in pons in up to 50% of patient with ODS.[3] Although the exact pathogenesis of CPM is not well understood, a pre-determined susceptibility of oligodendrocytes to oxidative stress and ionic fluctuations plays an integral role. Myelinolysis mainly occurs in central pons because of the inflexible “grid-like” arrangement of oligodendrocytes. The clinical presentation of CPM can vary widely, ranging from asymptomatic myelinolysis to locked-in syndrome. An autopsy study of 3000 brains found the incidence of asymptomatic CPM was estimated to be 0.5%.[4] The clinical presentation of CPM is closely related to brainstem anatomy: if corticospinal tracts are affected, patients may manifest as variable degrees of quadripareisis; with corticobulbar tracts involvement, patients may present with dysarthria and dysphagia; if lesions are located in tegmentum of the pons, ataxia and oculomotor abnormalities may also be documented. Locked-in syndrome due to CPM can occur in some severe cases, but the outcome is not always dismal.

Typical MRI feature of CPM is a trident-shaped hyperintense in the central pons on T2-weighted image. Enhancement scanning is usually normal and occasionally enhanced at the periphery of the area of myelinolysis. A cohort study was performed to identify potential prognostic factors in ODS. There was no correlation between the severity of the clinical features and the size of myelinolysis in the pons.[5] Previously, very few studies have focused on the MRS findings of CPM. Nomoto et al.[6] reported a case...
of CPM with chronic alcoholism and rapidly corrected hyponatremia. MRS showed that the Cho/Cr ratio increased to 2.58 while the NAA/Cr ratio was normal. In addition, Guo et al⁷ described decreased NAA/Cr ratio (1.58) and increased Cho/Cr (1.52) ratio in a patient of CPM after liver transplantation. To our knowledge, the decreased NAA level and increased Cho level may be related to neuronal loss and gliosis, respectively. In those two patients, the NAA peak was not obviously decreased and the motor function improved moderately. For the patient in this study, the severity of clinical presentation is not matched with the extent of myelinolysis in the pons. In addition, MRS scanning showed the NAA peak was decreased, but not significantly, suggesting the majority of neurons did not undergo apoptosis. We, therefore, speculate that NAA peak may play a role in evaluating the extent of neuronal loss and the severity of the disease.

Whether MRS can be identified as a factor predicting a poor outcome indeed requires more large-scale prospective studies to elucidate the robustness of the association.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initial will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Conflicts of interest
None.
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How to cite this article: Shen YY, Nie HB, Xia ZB, Bao B, Wu XB. Magnetic resonance spectroscopy findings of central pontine myelinolysis in an alcohol abuser. Chin Med J 2020;133:874–876. doi: 10.1097/CM9.0000000000000703.