

**INTERESTING CASE PRESENTATION:**

**Central Pontine Myelinolysis (CPM)**

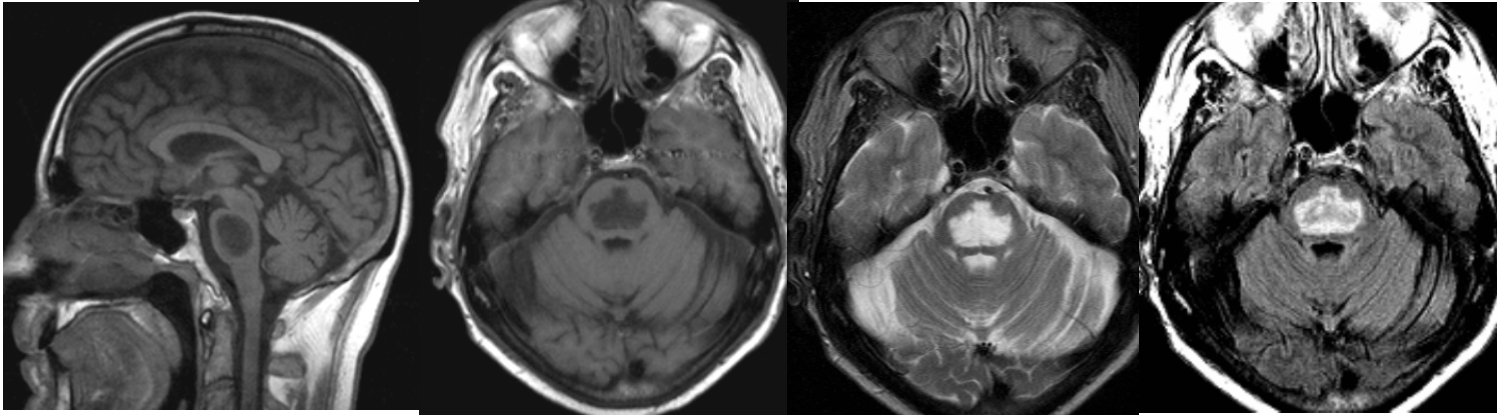


Fig. 1

Fig. 2

Fig. 3

Fig. 4

**CLINICAL PRESENTATION:** This 50-year-old male patient presented to the office of Dr. Jensen with numbness in the left aspect of the body. A brain MRI was ordered by **Dr. David Jensen** and **Dr. Janumpally**. Further history from the patient revealed history of chronic alcohol use.\*

**MRI FINDINGS:** A brain MRI with and without gadolinium contrast was performed on AIC's high-field 1.5 Tesla Siemens Symphony scanner. **Fig. 1** shows a midline sagittal T1 weighted image. **Fig. 2-4** show the axial T1 weighted, turbo T2 weighted, and turbo FLAIR images, respectively, through the pons. There is a 2 x 2.5 cm abnormality occupying the majority of the pons characterized by low T1 and high T2 and FLAIR signal intensity. The pons is not enlarged. There is no mass effect on the 4<sup>th</sup> ventricle and no midline shift. No abnormal enhancement was observed on post-contrast images (not shown). MR **diffusion** and **perfusion** studies (not shown) were also negative for any acute infarct.

**DIFFERENTIAL DIAGNOSIS:** The MRI findings, together with the history of chronic alcohol use, make **Central Pontine Myelinolysis (CPM)** the most likely diagnosis. Lack of mass effect or enhancement makes a neoplasm such as a glioma unlikely. An old pontine infarct may also have this appearance. An old MS plaque could have the same signal characteristics, although the central location and symmetry would be unusual.

**DISCUSSION:** Central pontine myelinolysis (CPM) is a disorder characterized pathologically by dissolution of the myelin sheaths of fibers within the central aspect of the basis pontis. In extreme cases, there may be extension to the pontine tegmentum, midbrain, thalamus, internal capsule and cerebral cortex. The myelinolysis occurs with relative sparing of the nerve cells and axon cylinders. **Many patients are asymptomatic**, and at the *other extreme* are patients whose symptoms are masked by *coma*. Most clinically diagnosed cases present with *spastic quadriparesis*, *pseudobulbar palsy*, and *acute changes in mental status* with progression possible to *altered levels of consciousness* and *death*. Survival is possible with varying residual neurologic deficits. Although initial reports were largely confined to **chronic alcoholics**, CPM has also been seen in patients with electrolyte disturbances, particularly due to **rapid correction of hyponatremia**.

For more information, you may call me at (661) 949-8111, Dr. Jensen at 273-2556, or Dr. Janumpally at 945-6931.

*Ray Hashemi, MD*

Ray H. Hashemi, M.D., Ph.D., Director

\*Clinical information provided by Dr. David Jensen, MD, a GP in Palmdale, and Dr. Janumpally, a neurologist in Lancaster.