

Central Pontine Myelinolysis: A case report.

Central pontine myelinolysis (CPM) (also known as **osmotic demyelination**) refers to acute demyelination of the white matter tracts traversing the pons. It seen in the setting of osmotic changes, typically with the rapid correction of hyponatraemia. When the extrapontine structures are affected it is known as extrapontine myelinolysis (EPM). Extrapontine structures which are affected include basal ganglia, midbrain and subcortical white matter.

Clinical History, Imaging findings:

62 year old female presented with history of altered sensorium with encephalopathy. MRI performed revealed trident area of altered signal intensity in the central pons. It showed hyperintense signal on T2WI and FLAIR with restriction on DWI. The cortico-spinal tracts were spared. No abnormality was seen in rest of the neuroparenchyma. Blood examination revealed hyponatremia.

Discussion:

The initial description was entirely in chronic alcoholics. Later it has been increasingly recognized in other patient groups, but usually in the setting of rapidly corrected electrolyte disturbance. Clinically CPM presents in a biphasic pattern.

The first phase is usually attributable not to the demyelination but rather to the inciting electrolyte abnormality, with patients being acutely encephalopathic. Following rapid reversal of this abnormality the patient transiently improves before progressing onto the classic CPM features 2 - 3 days later. These consist of spastic quadripareisis, pseudobulbar palsy, changes in levels of consciousness, coma or death.

Pathology:

Although the exact mechanism is still uncertain, it is known that oligodendroglial cells are most susceptible to osmotic stresses, leading to their demise. It is not surprising that the distribution of osmotic myelinolysis therefore parallels the distribution of these cells.

Radiographic features:

CT: CT may demonstrate low attenuation crossing the midline in the lower pons, although this region is frequently degraded by streak artifact and beam hardening phenomenon.

MRI: MR imaging findings of CPM include symmetric signal intensity abnormality in the central pons at T2-weighted and FLAIR imaging. This may progress to classic hyperintense “trident-shaped” central pontine abnormality, with sparing of the ventrolateral pons and corticospinal tracts. The earliest change is seen on DWI with restriction in the lower pons. This is seen within 24 hours of the onset of quadriplegia. This same region demonstrates eventual high T2 signal and later low T1 signal. The T1 and T2 changes may take up to two weeks to develop. Occasionally gadolinium enhancement is also demonstrated. Furthermore, the diagnosis of CPM is not ruled out in the setting of normal imaging.

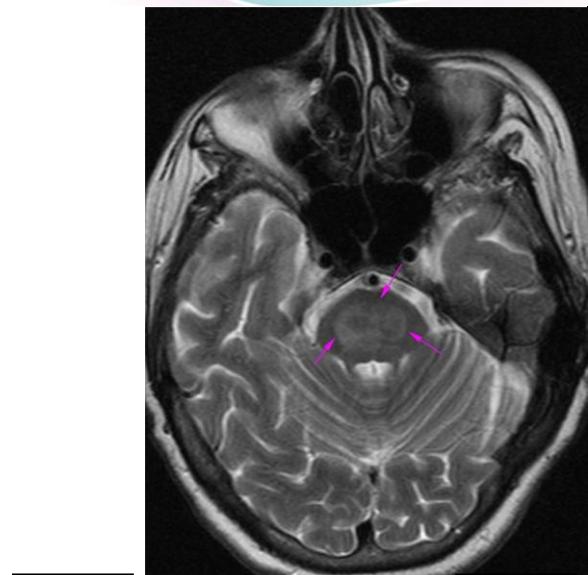


Fig 1

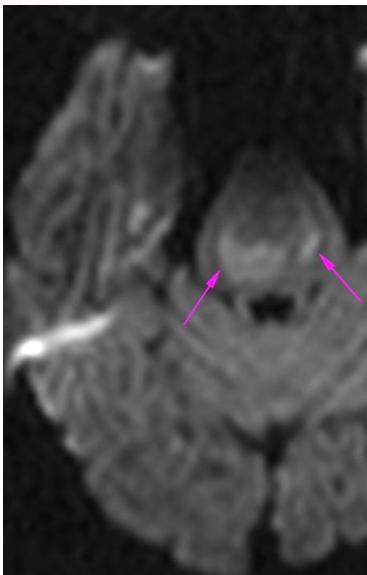


Fig 2

Fig 1: T2Wi axial image show trident area of hyperintense signal in the central pons.

Fig 2: DWi axial image shows restricted diffusion in the affected area of the central pons.

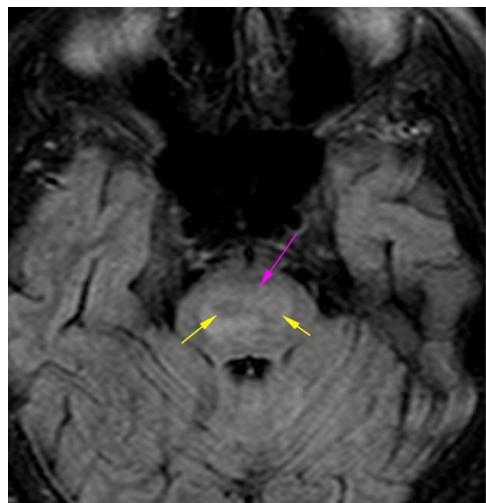


Fig 3

Fig 3: FLAIR axial image show trident area (pink arrow) of hyperintense signal in the central pons. Sparing of corticospinal tracts is seen. (yellow arrows)

Differential diagnosis:

General imaging differential considerations include:

- Demyelination including multiple sclerosis(MS)
- Ischemia / infarcts.
- Pontine neoplasms including astrocytoma.

Regards,

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N.B: These cases are authentic and from the archives of Radiance Diagnostics. For any queries/suggestions/feedback write to us at radiance@radiancediagnostics.in. Case of the month can also be accessed anytime online at **VIEW BOX** at www.radiancediagnostics.in