Obstructive uropathy leading to osmotic demyelination syndrome: an unusual case of extremely high osmotic load leading to central pontine and extrapontine myelinolysis

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Learning Objectives

- Concomitant and sudden increase in serum sodium and urea levels can cause osmotic shift leading to demyelination.
- Mildest form of osmotic demyelination syndrome is characterized by involvement of the corticobulbar tract, presenting as dysphagia and dysarthria.

Case description

A 67-year-old male was brought to the hospital after he was found in a minimally responsive state. He presented with a notable confusion, dysarthria, and dysphagia.

Past medical history: Unknown

Physical examination: Suprapubic tenderness and fullness, foley catheter was placed and drained almost 2 liters of bloody urine. Nodular prostate on digital rectal examination

His altered mental status on presentation was attributed to uremic encephalopathy. However, even though his electrolytes were improving, his dysphagia and dysarthria persisted

In patient work-up:

- **Initial laboratory analysis:** Sodium 166 mmol/L, blood urea nitrogen (BUN) 241 mg/dL, creatinine 35.9 mg/dL from unknown baseline and potassium 6.6 mmol/L.
- **CT CAP:** Multiple metastatic lesions throughout the body
- **Head CT:** Questionable hypodensity in the posterior limb of the left internal capsule extending to the cerebral peduncle.
- **MRI brain:** Confluent T2 signal abnormality in the brainstem extending into the dorsal pons as well as the superior cerebellar peduncles bilaterally, consistent with osmotic demyelination. (see figure 1 and figure 2)

Figures

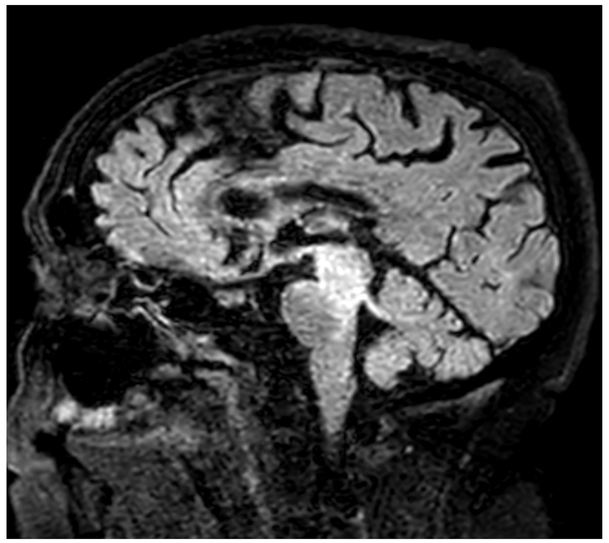

Figure 1. Sagittal T2 FLAIR sequence through the midline of the brain shows confluent T2 hyperintense signal in the brainstem, extending along the dorsal pons (outlined in blue dashed line).

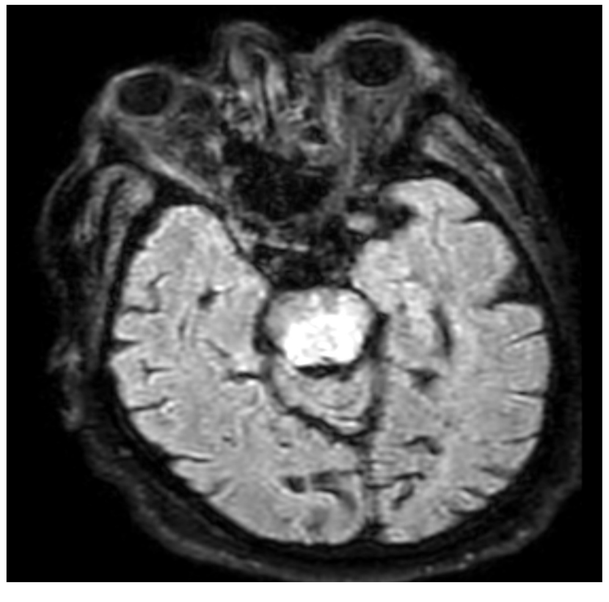

Figure 2.Axial T2 FLAIR images demonstrate extension of the T2 hyperintense signal into the pons, sparing the periphery and the corticospinal tracts, giving the characteristic “trident” appearance (outlined in red dotted line) seen in osmotic demyelination syndrome.

Follow-up

- Our patient presented with the mildest form of osmotic demyelination, confined to the corticobulbar tract presenting with dysarthria and dysphagia.
- The patient was commenced on continuous renal replacement therapy (CRRT). His sodium and BUN were corrected at a recommended rate over a 10-day period.
- Fortunately, it did not progress to the full blown “Locked-in” syndrome as the electrolytes were timely corrected.

Discussion

- This case illustrates the central pontine and extra pontine myelinolysis without iatrogenic overcorrection of hyponatremia.
- Elevated BUN might sometimes have a protective effect on demyelination process while hyponatremia is corrected too fast as it keeps osmotic balance within the safe window.
- However, if there is a simultaneous and significant increase in BUN and sodium within a short period of time, it might lead to osmotic demyelination.
- Although such cases have been reported infrequently, they hold substantial clinical significance and should be considered as a potential diagnosis when a patient presents with an exceptionally high osmotic load.

References

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