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Vanishing lesions in thalamus - A case of dialysis disequilibrium syndrome

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Clinical Image Description

48 years female patient presented to the emergency services of Apollo Speciality hospitals, Nellore in November 2015 with shortness of breath. Patient was a diagnosed case of renal failure who had refused hemodialysis earlier. On presentation, she was conscious with tachycardia at 120 beats/minute, respiratory rate of 24 per min and blood pressure 88/50 mm of Hg. Patient oxygen saturation was 90%. Blood parameters showed severe azotemia with blood urea 150 mg/ (dL), creatinine 9.2 mg/ (dL), potassium 8.3 (mEq/L), sodium 138 (mEq/L), bicarbonate level of 11 (mEq/L). She was given emergency hemo-dialysis through internal jugular dialysis catheter. As a protocol to avoid dialysis disequilibrium, hemodialysis was done with low blood flow (150 mL/min) and for short session length (2 hours).

Patient became confused immediately after dialysis. On neurological examination, she was drowsy, highly irritable when aroused. There was no limb weakness and was hemodynamically stable.

MRI Axial images showing bilateral symmetrical abnormal signals in thalami, which was hyperintense on T2 (Figure 1A) and Flair (Figure 1B) not showing typical diffusion restriction on DWI (Figure 1C) and ADC (Figure 1D). The imaging was suggestive of vasogenic edema. MRA and MRV were normal. The differentials considered were Dialysis Disequilibrium Syndrome (DDS) and acute Ischemia. Acute ischemia is excluded by normal MRV and MRA. Patient was given glycerol as antiedema measures and patient gradually improved in her sensorium. Her repeat MRI after one week was normal (Figure 2A-D). Further dialysis was done with sodium modeling and is on regular follow up.

DDS is a metabolic syndrome due to rapid removal of plasma urea during hemodialysis, which can lead to osmotic demyelinating syndrome. There could be diffuse cerebral edema due to fluid shifts into the brain during rapid decrease in serum osmolality during hemodialysis and can effect pons, thalamus, basal

ganglia and subcortical structures. Other differentials like acute stroke, metabolic encephalopathy due to hyperosmolar coma and hyponatremia were excluded. DDS can be prevented by slowing the rate of clearance of urea, by gradual standardized decrease in plasma osmolality and to avoid the bicarbonate-based rapid correction of metabolic acidosis.

We report this case of metabolic encephalopathy of DDS syndrome with the rare imaging findings in the thalamus.

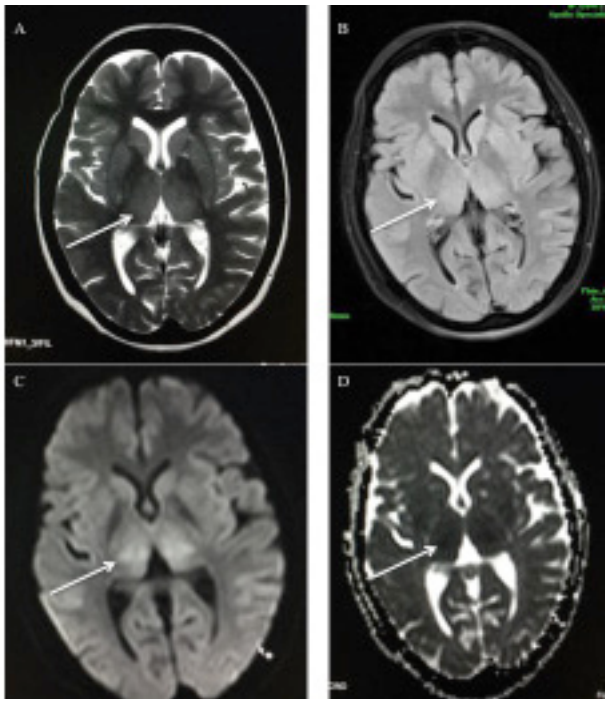

Figure 1 A-D: MRI Axial images showing bilateral symmetrical abnormal signals in thalami, which was hyperintense T2 and Flair not showing typical diffusion restriction on DWI and ADC.

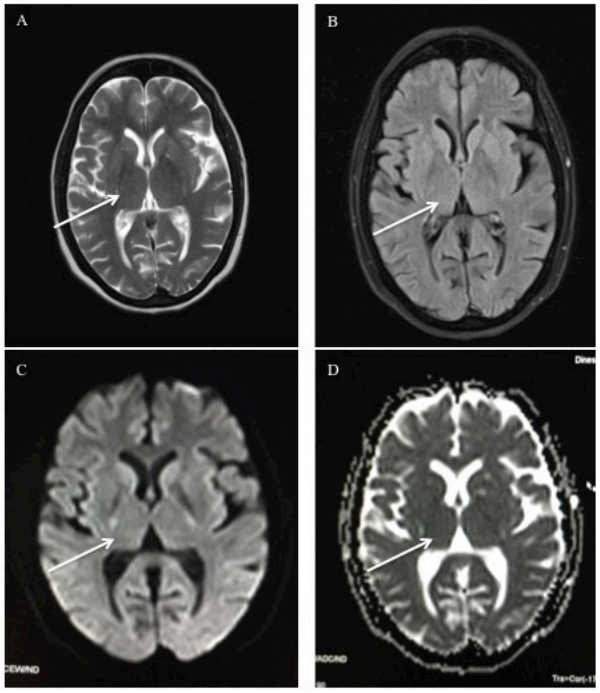

Figure 2 A-D: MRI of brain T2, Flair, DWI and ADC shows normal study.

Conflict of interest: Authors have no conflict of interest.

Author contributions: BM: study design, data acquisition, and writing the manuscript. MD: data acquisition, and reviewing the manuscript. GM: data acquisition, and reviewing the manuscript.

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