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The "Collar Tab Sign"–An Atypical MRI Feature in Uremic Encephalopathy

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Dear Editor,

This 56-year-old male patient with hypertension and chronic kidney disease presented with fever, altered sensorium, and generalized tonic–clonic seizures for 2 days. His Glasgow coma scale score was E1V1M1 at admission. Investigations revealed severe metabolic acidosis (pH=7.30), and azotemia (urea=118 mg/dL, creatinine=6.7 mg/dL) with evidence of sepsis (TLC=12.78×10^3/μL, procalcitonin=0.45 ng/mL). Other metabolic parameters including electrolytes, serum glucose and ammonia levels, and thyroid profile were normal. EEG suggested diffuse neurophysiological dysfunction with background activity in the theta range and intermittent activity in the diffuse delta range (3–4 Hz). MRI revealed extensive T2-weighted/FLAIR hyperintensities (HI) that involved the internal capsule, middle cerebellar peduncle, and supratentorial periventricular deep white matter including in the centrum semiovale, brainstem, and cerebellum. We suspected uremic encephalopathy, and so the patient underwent dialysis. This remarkably improved his condition, to regaining complete consciousness without any focal neurological deficit. However, he subsequently succumbed to septic shock due to worsening sepsis.

Various MRI findings have been noted in uremic encephalopathy with "lentiform fork sign" being the most characteristic. This sign is described as a T2-weighted/FLAIR hyperintense rim delineating the putamen border, the external capsule laterally, and the internal capsule medially, which resembles a fork.[1] Apart from the basal ganglia involvement, cortical–subcortical involvement akin to posterior reversible encephalopathy syndrome (PRES) and rarely white matter abnormalities have also been described.[2,3] The MR alterations and neurological symptoms in uremic encephalopathy are considered to occur secondary to accumulation of uremic toxins such as the guanidine compounds leading to excitotoxicity, mitochondrial and endothelial dysfunction. The uremic toxins alters the excitatory- inhibitory amino acid balance by enhancing the neurotoxic effect of N-Methyl-D-aspartate receptors and the inhibition of inhibitory aminobutyric acid receptors simultaneously. PRES like features are supposedly secondary to disruption of the blood brain barrier and endothelial dysfunction.[4]

In our patient, MRI brain showed lesions involving the internal capsule apart from the extensive T2/FLAIR HI. The lateral and medial borders of the internal capsule indicated T2-weighted/FLAIR HI that looked like tabs on the collars of Indian army officers (Fig. 1). Such internal capsule involvement in the absence of basal ganglia involvement has not been described previously in uremic encephalopathy. Basal ganglia, and especially the putamen and ventral pallidum, are well known to be susceptible to cytotoxic changes. The involvement of the internal capsule in the absence of basal ganglia involvement in our patient may be due to an individual variation in susceptibility to uremic toxins.

We propose naming this new sign as the "collar tab sign."

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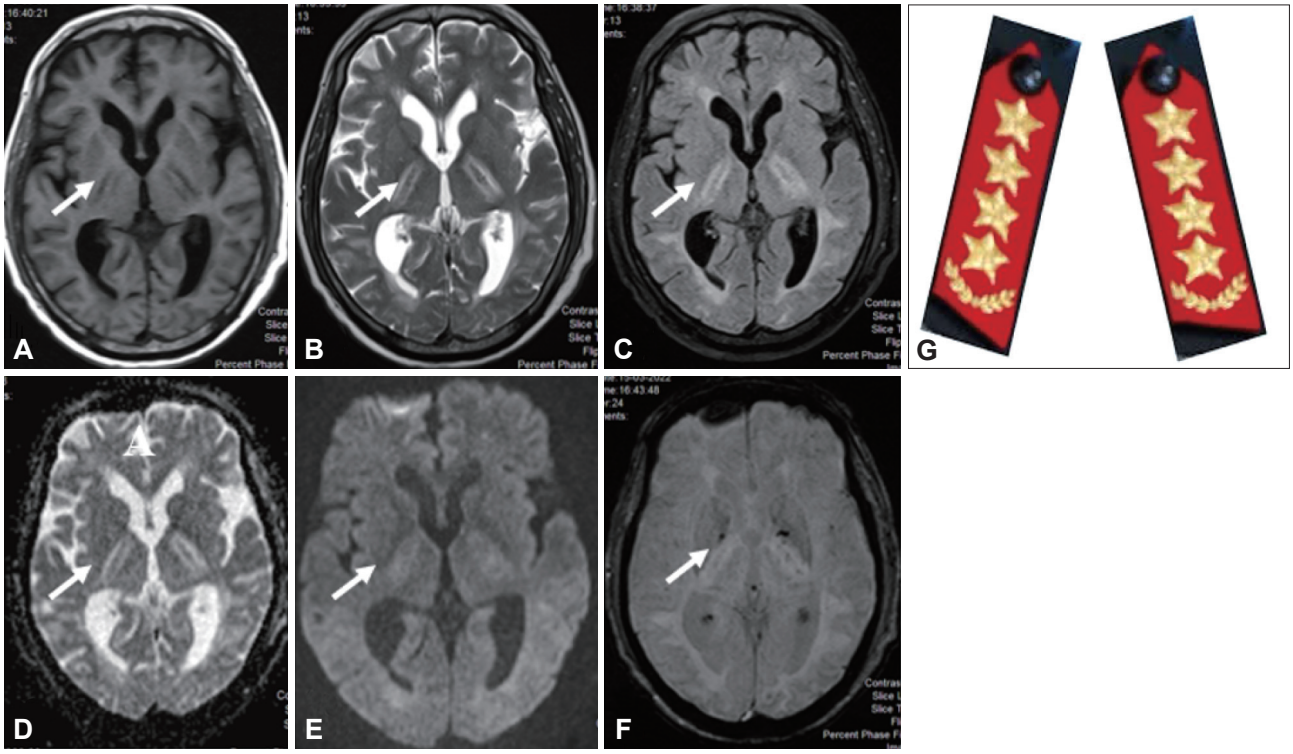

Fig. 1. MRI images showing the "collar tab sign" (B,C), comprising T2-weighted/FLAIR HI that involve the bilateral internal capsules with corresponding hypointensities on a T1-weighted image (A), but not indicating diffusion restriction on diffusion-weighted images (D and E), withevidence of microbleeds in susceptibility-weighted imaging (arrows) (F). The HI have the appearance of the collar tabs worn on the collars of Indian army generals (G).

Ethics Statement

The written informed consent was obtained from the deceased family member

Availability of Data and Material

Data sharing not applicable to this article as no datasets were generated or analyzed during the study.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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