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ATypical Uremic Encephalopathy

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Abstract

A 20 years old male presented with unconsciousness. On clinical examination and detailed evaluation etiology was not apparent. Patient was suspected to have ncephalopathy on imaging. Patient had history of ayurvedic drug intake. Patient was found to have elevated blood urea nitrogen , creatinine , and parathyroid hormone levels, and a mild decrease in the serum calcium level. Uremic encephalopathy is typically seen in patients with diabetes. It shows typical findings on MRI including bilateral vasogenic or cytotoxic edema at cerebral cortex or basal ganglia. Involvement of basal ganglia is rare, typically occurring in uremic-diabetic patient. Atypical type of uremic encephalopathy is rarest and involves WM. It typically occurs in non-diabetic uremic patient with very high serum urea levels and a variety of neurologic symptoms. Moreover non- diabetic uremic encephalopathy shows unique and atypical imaging findings.

Keywords: NIL

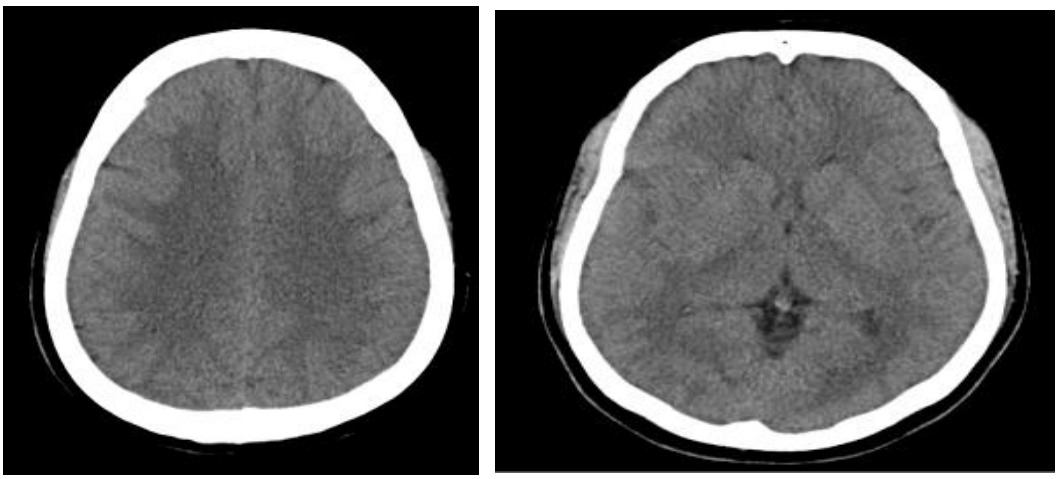
Introduction

History:

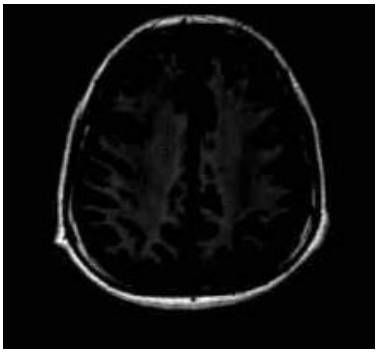
A 20 years old male patient presented with loss of consciousness. No history of Diabetes mellitus or hypertension. Patient was advised CT Brain which revealed no significant abnormality. This was followed by MRI of Brain as patient's condition showed no improvement.

Imaging:

On Ct Scan: No significant abnormality is noted.



ON MRI:



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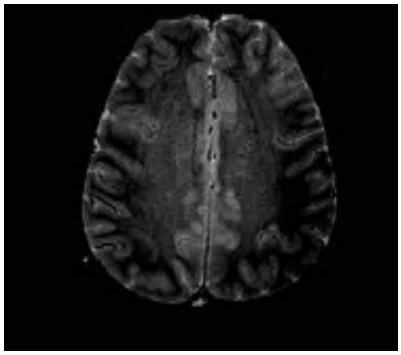

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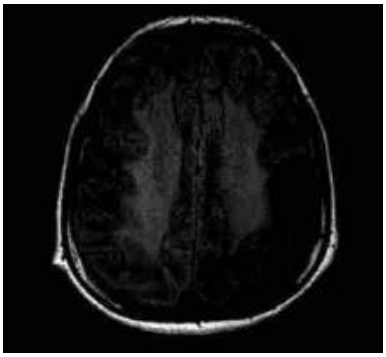
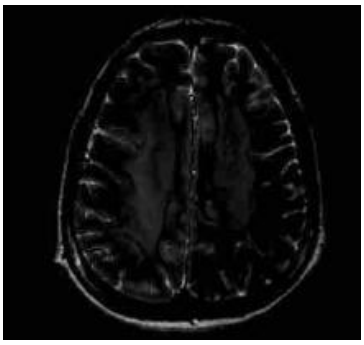
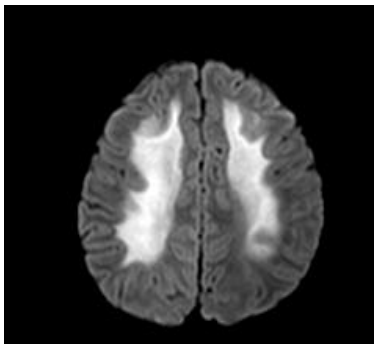


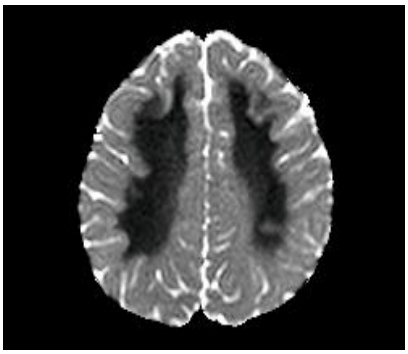
FLAIR



GRE

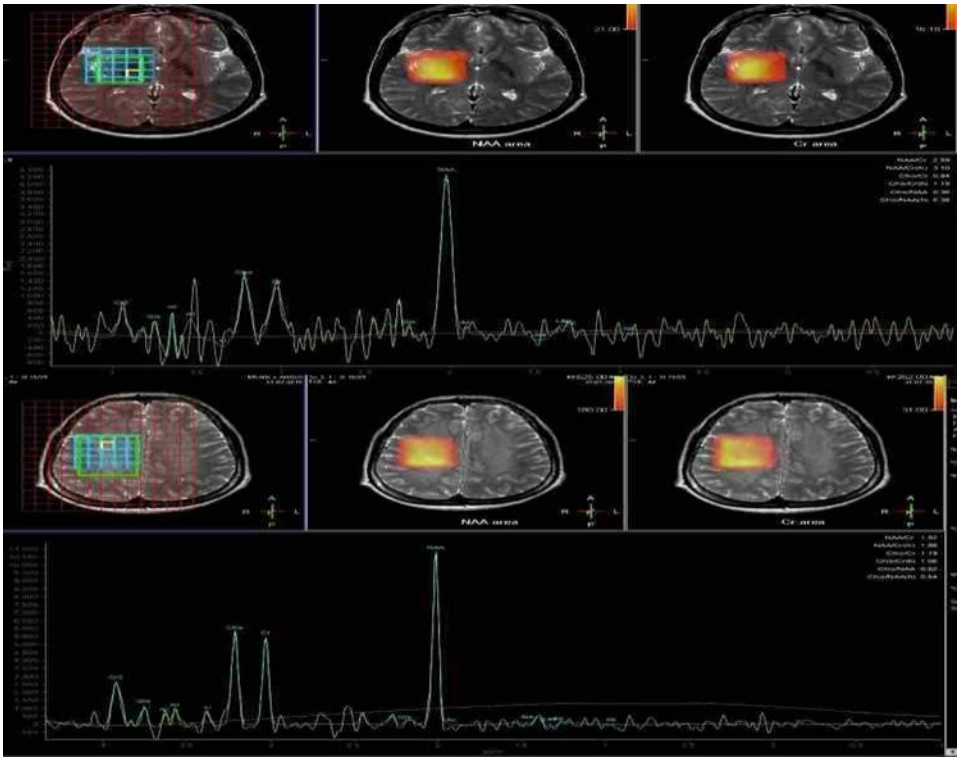


DWI



ADC

Spectroscopy



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Findings:

Laboratory examinations revealed, elevated blood urea nitrogen (BUN) (59.2 mg/dL), creatinine (5.3 mg/dL).

MRI study revealed, bilateral symmetrical restricting confluent hyperintensities on fluid sensitive in images in cerebral white mater, sparing cortex and basal ganglia.

Spectroscopy revealed no significant abnormality.

Diagnosis:

Atypical uremic encephalopathy.

Discussion:

Uremic encephalopathy (UE) is characterized by various neurological symptoms secondary to uremia.

1. UE is usually seen in patients with diabetes. Involvement of basal ganglia is rare, typically occurring in uremic-diabetic patient.
2. Atypical type of UE is rarest and involves white matter. It typically occurs in non-diabetic uremic patient.
3. Factors such as uremia, methylguanidine, aluminum, neurotransmitters, brain osmolality, metabolic acidosis, hypo- or hyper-glycemia, changes in cerebral blood flow, and metabolic acidosis have been cited as causes of UE.
4. Increased levels of urea and toxins may inflict toxic and metabolic injury on brain tissue that has been weakened by microangiopathic changes, energy utilization failures, or vascular autoregulation collapse. The cerebral oxygen consumption is significantly reduced in uremia, and this may affect focal cellular metabolism and result in cellular edema.
5. In most reported cases, lesions on neuroimaiging and symptoms disappeared when uremia was treated.
6. Non-diabetic UE can show unique imaging findings. Even these lesions are considered cytotoxic edema. However, if adequate treatment, including removal of uremic toxins and correction of metabolic acidosis, is provided, complete neurological and radiological recovery can be achieved.

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