SVOA Neurology

ISSN: 2753-9180

ScienceVolks Open Access

Case Report

Cytotoxic Edema of Bilateral Semicentrum Ovale in Uremic Encephalopathy: A Rare Radiological Entity

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https://doi.org/10.58624/SVOANE.2025.06.009

Received: January 24, 2025

Published: March 21, 2025

Citation: Manorenj S, Kumar SS, Shashanka C. Cytotoxic Edema of Bilateral Semicentrum Ovale in Uremic Encephalopathy: A Rare Radiological Entity. *SVOA Neurology* 2025, 6:2, 47-49. doi. 10.58624/SVOANE.2025.06.009

Abstract

Uremic encephalopathy can mimic clinically and radiologically like a stroke. The presence of cytotoxic edema in bilateral semicentrum ovale without involvement of basal ganglion is uncommon in uremic encephalopathy in a diabetic patient. Here we demonstrate the rare white matter radiological subtype of uremic encephalopathy in a 48-year-old woman with chronic kidney disease.

Keywords: *Cytotoxic Edema; Semi Centrum Ovale; Azotaemia*

Introduction

Uremic encephalopathy (UE) is a neurological disorder resulting from the buildup of toxins in the blood due to either acute or chronic kidney failure. Symptoms include confusion, seizures, and difficulties in motor function. UE can be divided into various types, including one that impacts the white matter. This case describes an uncommon occurrence of bilateral cytotoxic edema in the semiovale centrum (SCO), which is generally linked to stroke but can also manifest in UE. [1-4]

Case Presentation

A 48-year-old woman presented with a sudden onset of confusion, weakness in the left side of her body, and difficulty breathing, all of which lasted for one day. She has a history of diabetes, hypertension, and chronic kidney disease, which are being managed medically. On examination, she had general swelling throughout the body, with a blood pressure of 150/90, pulse rate of 86, random blood sugar level of 98mg/dL, and 90% saturation. Her central nervous system examination revealed a confusional state (E4V2M6) , grade 3/5 weakness in the left upper limb, and grade 4/5 weakness in the left lower limb. Basic blood tests indicated elevated levels of urea and creatinine [Table 1].

MRI of the brain showed diffusion restriction in the bilateral semi-centrumovale consistent with cytotoxic edema, mimicking an acute stroke affecting both sides of the middle cerebral artery territory [Figure 1]. However, MR angiogram, echocardiogram, and neck vessel Doppler test all returned normal. The patient received symptomatic treatment for azotemia, and after correcting the metabolic parameters, she showed improvement in weakness, speech, and awareness within 48 hours.

Table 1. Basic lab parameters.

Tests	Value	Normal value
RBS	112mg/dl	80-150mg/dl
Serum calcium	8.7mg/dl	8.8-10.8mg/dl
Serum phosphorus	4.96mg/dl	2.5-4.5mg/dl
Intact parathormone	117pg/ml	11.1-79.5pg/ml
Serum Magnesium	1.61mg/dl	1.6-2.3mg/dl
Triiodothyronine (T3)	0.675ng/ml	0.56-2.05ng/ml
Thyroxine (T4)	6.14µg/dl	3.4-12 µg/dl
Thyroid stimulating hormone (TSH)	3.17µIU/ml	0.27-4.6µIU/ml
Procalcitonin	<0.5µg/L	2-10 µg/L sepsis
Serum sodium	144.2mmol/L	136-145mmol/L
Potassium	**5.37mmol/L**	3.5-5mmol/L
Urea	**96 mg/dl**	14-45mg/dl
Creatinine	**4.9 mg/dl**	0.6-1.2mg/dl
Uric acid	6.1mg/dl	3.5-7mg/dl
Liver function tests	Normal range	

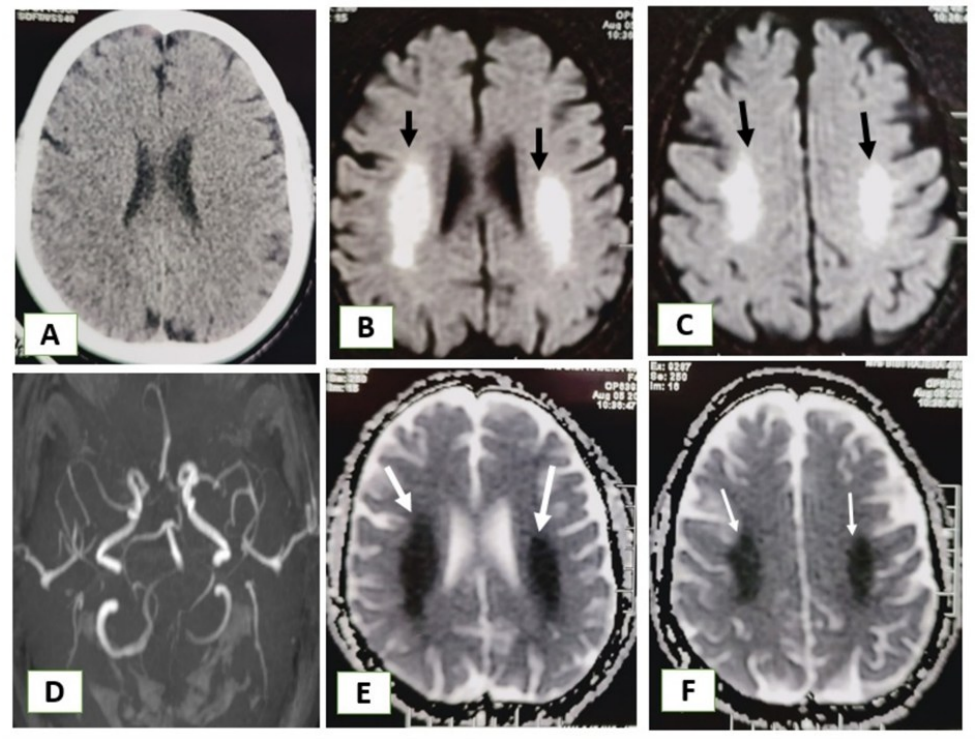

Figure 1. *Computed tomography brain (A) showing normal brain parenchyma at the level of body of lateral ventricle, there are hyperintensity involving bilateral semicentrum ovale in diffusion weighted images (B, C), with corresponding hypointensities on ADC (adsorption diffusion coefficient) images (E, F) consistent with symmetrical cytotoxic edema in semicentrum ovale. MRA (Magnetic resonance angiography) showed normal angiography (D) except attenuated left vertebral artery.*

Discussion

Uremic encephalopathy (UE) occurs when toxic substances such as urea and other protein metabolism products accumulate in the bloodstream due to acute or chronic severe renal failure, leading to an acute neurological condition. It presents with diverse symptoms, typically manifesting as a sudden or gradual onset of neurological symptoms such as headaches, seizures, confusion, asterixis, ataxia, or motor dysfunction. [1] Based on the MRI findings and clinical presentation, three variants of UE have been identified: a cortical or subcortical type closely associated with posterior reversible encephalopathy syndrome (PRES), a basal ganglia (BG) type with classical lentiform fork sign commonly seen in individuals with diabetes, and a white matter type affecting the internal capsule and periventricular white matter. [2,3]

Our patient exhibited bilateral cytotoxic edema in the semicentrum ovale (SCO), a paired mass of white matter above the lateral ventricles and corpus callosum in each cerebral hemisphere. Hyperintensities on diffusion weighted image (DWI) accompanied by reduced ADC (adsorption diffusion coefficient) values in the subcortical white matter, indicating cytotoxic edema, may be observed in watershed infarctions, myelinolysis, diffuse axonal injury, multiple sclerosis, extra-pontine myelinolysis, hypoglycemia, and hypoxic-anoxic encephalopathy, which were excluded based on an incompatible history.

Our case displayed radiological features indicative of white matter involvement in the SCO, resembling a stroke clinically and radiologically. Isolated symmetrical cytotoxic edema in the SCO is very rare in UE. [4] A strong clinical suspicion for UE and correction of renal parameters are more effective in reversing the condition.

Conclusion

This case highlights the unusual manifestation of UE with cytotoxic edema in the SCO, stressing the importance of maintaining a high degree of clinical awareness in patients suffering from kidney failure. Timely identification and management of renal impairment can lead to a reversal of neurological symptoms, reinforcing the significance of early diagnosis and treatment in UE.

Declaration of Informed Consent

The authors confirm that they have acquired all necessary consent forms from the patients. The patient(s) have provided consent for the publication of their images and clinical information in the journal. The patients are aware that their names and initials will not be disclosed, and every attempt will be made to protect their anonymity, although complete anonymity cannot be guaranteed.

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