Case Report

The Journal of the Association of Physicians of Tamil Nadu,
Vol. 1, Issue 3, English Quarterly, July – September 2022

A Case of Reversible Hemiplegia

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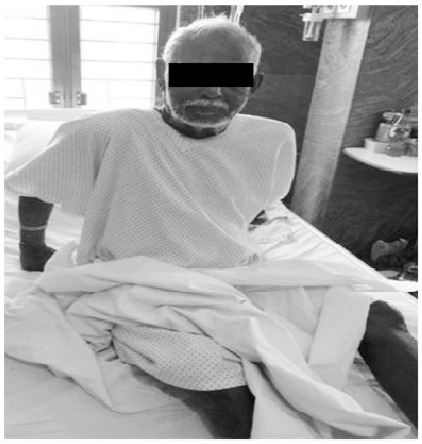
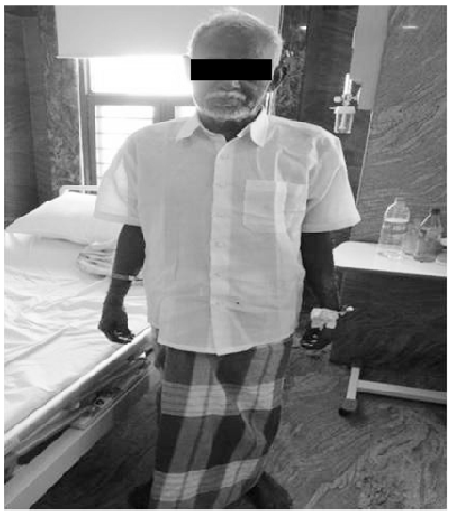
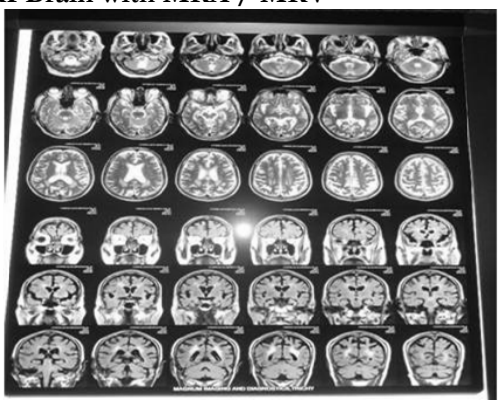
Mr. Kathirvel, 84 Yrs male a known case of T2DM/SHT/CAD admitted with complaints of acute onset of weakness of right upper and lower limb for four days. H/o decreased food intake+. No history of seizures, fever, headache, vomiting, or diarrhea. O/E– Patient conscious, oriented, dehydration+ vitals stable. CNS-right hemiplegia (2/5). Sensory examination normal. The other system is examination-clinically normal. Clinically patient was diagnosed with Acute CVA-right hemiplegia and proceeded with investigations.

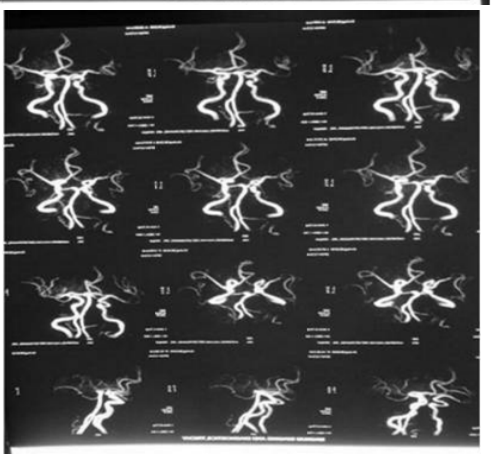




GRBS was high. Random venous blood sugar -720 mg%. Urine ketone was negative. Urea-46 mg %, creatinine - 1.2mg %. ABG analysis – pH - 7.38, Na-129meq/l, K-3.4meq/L, Hco3-18meq/L and cl-91mmol/L. Serum Osmolality 330m Osmol/kg. ECG–Normal sinus rhythm with LAHB. CT brain showed multiple infarcts in the periventricular region, left lateral intraventricular mass, and meningioma. The echocardiogram showed CAD–LAD territory with an EF of 39%. USG abdomen– Bilateral chronic medical renal disease and Grade1 prostatomegaly. Bilateral carotid and vertebral Doppler-2mm thick mixed plaques were seen in both CCA, and no evidence of significant stenosis. MRI/MRA/MRV brain showed age-related atropic changes without any evidence of infarct, hemorrhage, and space-occupying changes

MRI Brain with MRA / MRV





The patient was diagnosed with nonketotic hyperosmolar syndrome and treated

with Insulin infusion, IV fluids, antihypertensives, antiplatelets, statins, and other supportive measures. After lowering blood sugar with insulin infusion, weakness improved completely. After ruling out other causes of weakness, the patient was finally diagnosed with Nonketotic hyperosmolar syndrome (NKHS) induced Reversible hemiplegia.

NKHS is less common than DKA, with an incidence of <1/1000 person Per year HHS is characterized by hyperglycemia, hyperosmolarity, and dehydration in the absence of ketoacidosis. HHS is attributed to deficient insulin function and counter-regulatory hormone increase. A relative deficiency of insulin concentration characterizes HHS to maintain normoglycemia but adequate levels to prevent lipolysis and ketogenesis and increase in counterregulatory hormones. Neurological manifestations in HHS are diverse and include encephalopathy, focal seizures, hyperkinetic movement disorders, and acute stroke-like syndrome.

References

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