VOLUME - 12, ISSUE - 11, NOVEMBER - 2023 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Medical Biochemistry

A RARE CASE OF UREMIC ENCEPHALOPATHY WITH VERY HIGH LIVER ENZYMES IN A KNOWN CASE OF T2DM & HYPERTENSION- CASE REPORT

Dr. Supratim Acharjee* Junior Resident, Academic, Dept. Of Biochemistry, AGMC *Corresponding Author

Prof. (Dr.) Chinmoy Biswas Professor & HOD, Dept. Of Biochemistry, AGMC

Dr. Izora T. R. Marak Assistant Professor, Dept. Of Biochemistry, AGMC

ABSTRACT Uremic encephalopathy (UE) is defined as cerebral dysfunction due to the accumulation of toxins resulting from acute or chronic renal failure. The clinical presentation can vary from fatigue, anorexia to restlessness, drowsiness to delirium, seizures and coma. A Male patient in his 70s, a Known case of CKD, HTN & T2DM presented with Sudden onset shortness of breath for last one day. The patient was unconscious. At admission RBS was High. Blood Urea, Sr. Creatinine & Uric acid were significantly High. Liver Enzymes were very much Elevated. Sr. Potassium, Bilirubin were elevated. The case was diagnosed as CKD with Uremic Encephalopathy with CCF in a known patient of T2DM & HTN. Severe complications of UE can lead to death. Early recognition of neurological manifestation is critical to prevent morbidity or mortality. Cases of UE with this much High ALT, AST levels in thousands figure is not usually seen. coexistence of Congestive Cardiac Failure in this patient causing Ischemia may have caused this condition.

KEYWORDS : Uremic encephalopathy, chronic renal failure, rare case report, Congestive Cardiac Failure, liver enzymes, ALT, AST, complication.

INTRODUCTION

Uremic encephalopathy (UE) is defined as cerebral dysfunction due to the accumulation of toxins resulting from acute or chronic renal failure.[1] It usually develops in patients with acute or chronic renal failure when their estimated glomerular filtration rate (eGFR) decreases and stays below 15 mL/min.[1] The syndrome is likely caused by retention of uremic solutes, alterations in hormonal metabolism, changes in electrolyte and acid-base homeostasis, as well as changes in vascular reactivity, blood–brain barrier transport, and inflammation.[2] The syndrome likely results from alterations in hormonal metabolism, retention of uremic solutes, changes in electrolyte and acid-base homeostasis, blood-brain barrier transport, changes in vascular reactivity, and inflammation.[1] There are no defining clinical, laboratory, or imaging findings, and the diagnosis is often made retrospectively when symptoms improve after dialysis or transplantation.[2] They may occur as a manifestation of acute metabolic derangements such as hypocalcemia, hyperphosphatemia, hypomagnesemia and hyperkalemia, acidosis, hypertensive encephalopathy, brain edema and renal hyper parathyroidism. [3] UE presents with symptoms ranging from mild inattention to coma, and can also accompanied by sleep disorders, headache, dysarthria, gait disorders, and less frequently by extrapyramidal movements such as involuntary movement, chorea and bradykinesia [4] Severe encephalopathy is an uncommon occurrence in patients with progressive CKD because most patients will start planned KRT before severe central nervous system (CNS) manifestations can occur. [2] Upon initiation of renal replacement therapy, UE may be reversed. [1,5,6] So early detection of neurological manifestation can avoid the progression to UE & can mortality & morbidity can be decreased caused by this complication.

Clinical Case Report

A Male patient in his 70s, a Known case of Chronic Kidney Disease, Hypertension & Type 2 Diabetes Mellitus presented to the Emergency Department with Sudden onset Shortness of breath for last one day. Patient was not on Hemodialysis.

On Examination the patient was Unconscious. Afebrile, Heart rate was 86 bpm & Blood pressure 90/70 mmHg. Random Blood Sugar (RBS) – 300 mg/dL on the time of Admission.

METHODOLOGY

Blood samples were collected by Venepuncture for testing of Biochemical parameters. Biochemical parameters were tested by XL 640 Full Automated Analyzer by Erba. Serum Electrolytes by EasyLyte automated electrolyte analyzer by ISE Method.

The Biochemical, Haematological, Microbiological & Serological Tests are given below:

Biochemical Tests

Table 1 : Biochemical parameters

	Renal Profile			Normal Range Values
	07.08.2023	08.08.2023	09.08.2023	
Blood Urea	156	160	182	15-40 mg/dl
Sr. Creatinine	9.3	9.9	13.17	0.6-1.5 mg/dl
Sr. Uric Acid	-	21.2	-	2-7 mg/dl
	Liver Profile			
	07.08.2023	08.08.2023	09.08.2023	
Bilirubin (Total)	2.2	2.1	2.73	0.2 – 1.2 mg/dl
Bilirubin (Direct)	1.1	1.0	1.29	0.1 – 0.5 mg/dl
Bilirubin (Indirect)	1.1	1.1	1.44	0.2-0.8 mg/dl
ALT	6763	3239	2011	5-40 IU/L
AST	4495	4829	1780	5-40 IU/L
Alkaline Phosphatase (ALP)	133	135	135	50-280 IU/L
Total Protein	6.2	5.6	5.5	5.5-8.5 gm/dl
Albumin	3.1	3.0	2.7	3-5 gm/dl
Globulin	3.1	2.6	2.8	2-4 gm/dl
A:G Ratio	1	1.15	0.96	1.2-2.5
	Lipid Profile			
	07.08.2023	08.08.2023	09.08.2023	

Sr. Total Cholesterol	120	103	108	120-200 mg/dl
Sr. Triglyceride	182	170	192	60-175 mg/dl
Sr. HDL	43	38	40	30-70 mg/dl
Sr. VLDL	36	34	38	12-35 mg/dl
Sr. LDL	41	31	30	70-130 mg/dl
	Electrolyte Profile			
	07.08.2023	08.08.2023	09.08.2023	
Sr. Ca2+	8.1	7.8	7.8	9-11 mg/dl
Sr. Na+	136	138	138	135-145 mEq/L
Sr. K+	6.0	5.8	6.31	3.5-5.5 mEq/L

Haematological Parameters:
Hemoglobin - 10 gm%
PCV – 38 %
Total Leucocyte Count- 17,300
Differential Leucocyte Count
- Neutrophil - 92
- Lymphocye-05
- Monocyte-03
- Eosinophil-00
- Basophil- 00

RBC Morphology: Normocytic Normochromic

Serology
HIV 1&2 – Non Reactive
HBsAg - Non Reactive
Anti HCV- Non Reactive

Microbiological Culture
Urine Culture & Sensitivity Test – No Growth seen after 48 Hours.

Differential Diagnosis
Possible Differential Diagnosis of this clinical condition
1. Hyperosmolar Hyperglycemic State (HHS)
2. Wernicke-Korsakoff encephalopathy
3. Hypertensive encephalopathy
4. Hepatic encephalopathy
5. Sepsis
6. Fluid and electrolyte disturbances
7. Hypoglycemia

DISCUSSION
At admission RBS was High. On Day 2, ALT & AST were markedly raised & was showing 6763 & 4495 respectively. This much high values in a known patient of Chronic Kidney Disease is rare. In a study by Rai L et al found serum AST and ALT levels were significantly lower in CKD patients both without and with ESRD compared to controls. [7] This much High ALT & AST level may suggest liver involvement , possibly due to Ischaemia due to coexistence of Congestive Cardiac Failure in this patient. Here our patient was Unconscious with Significantly High Blood Urea & Serum Creatinine level & High Total Leucocyte count which supports the Diagnosis of Uremic Encephalopathy. High Uric Acid level supports the diagnosis CKD. Electrolyte imbalance like Elevated Potassium (Hyperkalemia) could be result from Kidney dysfunction & it can have serious cardiac implication.

CONCLUSIONS
The case was diagnosed as CKD with Uremic Ence phalopathy with CCF in a known patient of T2DM & HTN. Severe complications of UE can lead to death. Early recognition of neurological manifestation is critical to prevent morbidity or mortality. Renal Replacement Therapy has shown significant improvement in this condition. The patient should be considered for treatment options like Hemodialysis & Kidney Transplantation.

REFERENCES:

1. Olano CG, Akram SM, Bhatt H. Uremic Encephalopathy. In: StatPearls. StatPearls Publishing, Treasure Island (FL); 2022. PMID: 33231997.
2. Rosner MH, Husain-Syed F, Reis T, Ronco C, Vanholder R. Uremic encephalopathy. Kidney International. 2022 Feb 1;101(2):227-41.
3. Hamed SA, Abdulhamid SK, Elhadad AF. 2020. Uremic Seizures with Chronic Kidney Disease: Clinical Types, Possible Mechanisms and Response to Treatments. J Neurol Exp Neurosci 7(1): 1-8.
4. Gong, WY., Li, SS., Yu, ZC. *et al.* Syndrome of uremic encephalopathy and bilateral basal ganglia lesions in non-diabetic hemodialysis patient: a case report. *BMC Nephrol* **19**, 370 (2018). https://doi.org/10.1186/s12882-018-1174-0
5. Yanai, A., Uchiyama, K. & Ishibashi, Y. Uremic encephalopathy in patients undergoing assisted peritoneal dialysis: a case series and literature review. *CEN Case Rep* **8**, 271–279 (2019). https://doi.org/10.1007/s13730-019-00406-3
6. 허덕현, 김도의, 이경복, 노학재, 안무영. 급성파킨슨증상으로발현한가역성요독뇌병증. 대한신경집중치료학회지. 2010;3(2):45-8.
7. Ray L, Nanda SK, Chatterjee A, Sarangi R, Ganguly S. A comparative study of serum aminotransferases in chronic kidney disease with and without end-stage renal disease: Need for new reference ranges. International Journal of Applied and Basic Medical Research. 2015 Jan;5(1):31.