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CASE REPORT

Metabolic Encephalopathy in a Hypertensive Patient with Hyponatremia: A Case Report

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ABSTRACT

Metabolic encephalopathy is a reversible neurological disorder resulting from systemic metabolic derangements. Hyponatremia is among the most common electrolyte abnormalities implicated in altered sensorium and when compounded by chronic hypertension it can significantly exacerbate cerebral function. We report a case of an 80-year-old female, also a known hypertensive who came to the casualty with complaints of sudden loss of hearing and sudden loss of vision. The patient was conscious but disoriented for 4 hours with confusion, restlessness, nausea, headache. Blood pressure was measured to be 220/104 mmHg, was administered 20 mg Labetalol iv stat.

The patient had no h/o chest pain, palpitations. No h/o Cerebrovascular accident. The patient had h/o myocardial infarction 4 years back. She was not on any medications, which progressively led to worsening conditions. Timely intervention was critical to prevent rapid descent into neurological and metabolic damage.

This case highlights the importance of controlling hypertension, and early intervention, to prevent destabilization of metabolic equilibrium.

KEYWORDS

Disoriented, Drowsy, Hypertension, Hyponatremia, Encephalopathy.

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INTRODUCTION:

The metabolic encephalopathies comprise a series of neurological disorders not caused by primary structural abnormalities; rather, they result from systemic illness, such as diabetes, liver disease, renal failure, and heart failure (1) .In one study there were 251 patients who had encephalopathy out of these patients 110 were women. Most of these patients 186 to be exact were between 60 and 75 years of age. Their average age was 70.78 years. 112 of them went to the hospital within 6 hours of altered mental status. Encephalopathy can result due to a magnitude of causes like neurological in one third patients, infection, and sepsis in one third and metabolic in one third. The most common cause of metabolic encephalopathy is hyponatremia followed my hypoglycemia. There were 19% deaths out of which 79% had preexisting comorbidities (2). Most of these encephalopathies are due to complications occurring in ICU patients (3) .Early presentation to hospital (within 6 hours of commencement of symptoms), higher GCS and conscious level at presentation are good prognostic markers in elderly patients. Metabolic encephalopathies result from alteration of brain chemistry at both neocortical and brainstem ARAS centers. Clinical features include generalized depression, decreased consciousness, depressed respiratory function. It can also progress to asterixis which is seen in uremia, hepatic disease, and sedative intoxication. Other complications include seizures, Cheyne stokes respiration, disorders in movement and coordination. metabolic encephalopathies are reversible but can lead to secondary brain damage. Some mechanisms by which cerebral dysfunction occurs in metabolic encephalopathies include focal or global cerebral edema, alterations in transmitter function, the accumulation of uncleared toxic metabolites, post capillary venule vasogenic edema and energy failure (4). Such varied mechanisms reflect the heterogenous etiology that produces this condition of altered consciousness. Metabolic encephalopathy is therefore not a diagnosis, but rather a clinical state.

PATIENT INFORMATION:

On 28/08/2025, an 80-year-old female, also a known hypertensive came to casualty with complaints of sudden loss of hearing and sudden loss of vision. The patient was conscious but disoriented for 4 hours with confusion, restlessness, nausea, headache, and breathlessness. Blood pressure was measured to be 220/104 mmHg, was administered 20 mg Labetalol iv stat. The patient was alright 3 days back when she had a fall, following which she was taken to the hospital and diagnosed with a right neck of femur fracture, and was planned for surgery.
The patient had no h/o giddiness, chest pain, palpitations. No h/o cerebrovascular accident.
The patient had h/o myocardial infarction 4 years back. She did not take any regular medications.

CLINICAL FINDINGS:

On examination, blood pressure was raised up to 220/100 mmHg. The patient complained of breathlessness and was hence supported with 4 L oxygen, with spo2 85%. Bilateral rhonchi were heard. The right pupil had a sluggish reaction to light on admission. The left pupil was leucomatous and showed corneal changes. Bilateral plantar reflex was not elicitable. Movement was present in all four limbs.

DIAGNOSTIC ASSESSMENT TIMELINE:

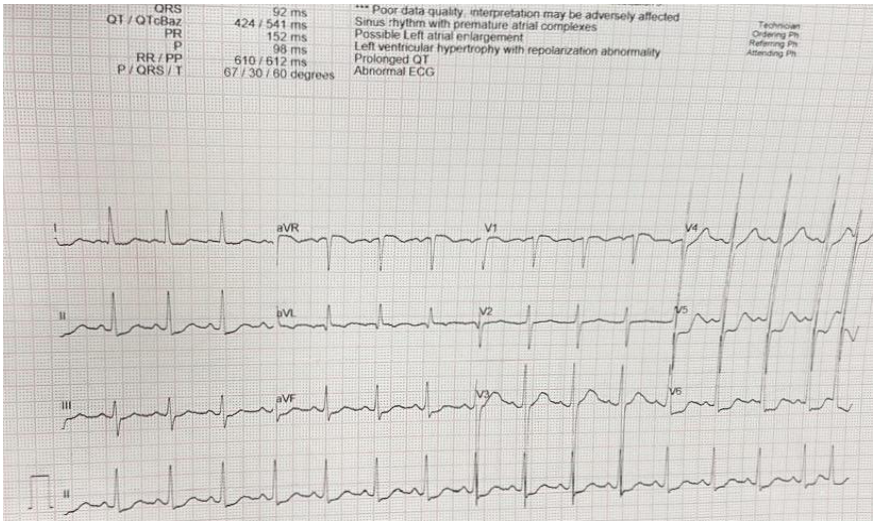
Date	Blood pressure	Changes in investigation	Management
28/08/25	220/100 mmHg	Serum sodium 112.7 mg/dl Trop I 297	3 % NaCl infusion at 18ml/hr Labet 20mg IV stat Inj. Strocit 500 mg stat Nebulisationwith duolin and budecort Tab temson 40 od 1-0-0 Injaugmentin 1.2g TDS 1-1-1 Inj pan 40 od 1-0-0 Injperiset 8mg TDS 1-1-1
29/08/25	200/98 mmHg	Serum sodium 115.58 mg/dl Serum potassium 3 mg/dl	3 % NaCl infusion at 18ml/hr InjCeftriazone 2mg bd 1-0-1(exchanged) Inj mannitol 20% 300ml ½ stat Tab ecospirin 75mg 0-1-0 Tab atorac 40mg hs 0-0-1 Tab temson 40 od 1-0-0 Inj. Kesol 1 ampoule in 500 cc NS
30/08/25	172/98 mmHg	Serum sodium 122.8 mg/dl Serum potassium 2.2 mg/dl	3 % NaCl infusion at 18ml/hr Inj. Strocit 500 mg stat InjCeftriazone 2mg bd 1-0-1(exchanged) Inj mannitol 20% 300ml ½ stat Tab ecospirin 75mg 0-1-0 Tab atorac 40mg hs 0-0-1

			Tab temson 40 od 1-0-0 Inj. Kesol 1 ampoule in 500 cc NS
31/08/25	150/96 mmHg	Serum sodium 133.6 mg/dl Serum potassium 3.1 mg/dl	3 % NaCl infusion at 18ml/hr InjCeftriazone 2mg bd 1-0-1(exchanged) Inj mannitol 20% 300ml ½ stat Tab ecospirin 75mg 0-1-0 Tab atorac 40mg hs 0-0-1 Tab temson 40 od 1-0-0 Inj. Strocit 500 mg stat Inj. Kesol 1 ampoule in 500 cc NS
01/09/25	146 / 90 mmHg	Serum sodium 138.8 mg/dl Serum potassium 3.6 mg/dl	3 % NaCl infusion at 18ml/hr Coconut water sips orally Oral liquids <1.2 1 / day Inj tramadol 1 amp in 100 ml ns 1-0-0 And continued dose of Ramipril Tab temson 40 od 1-0-0 Inj. Strocit 500 mg stat Inj. Kesol 1 ampoule in 500 cc NS

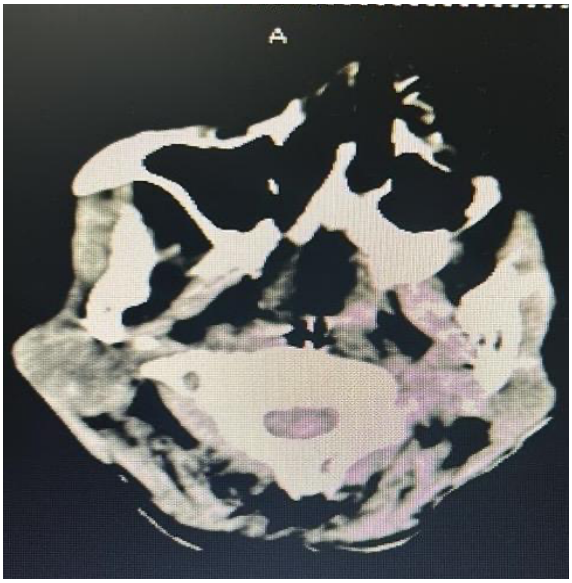
Normal range:
Na - 135-145 meq /l
K- 3.5-5.3 meq /l
Trop I < 0.04 ng /ml

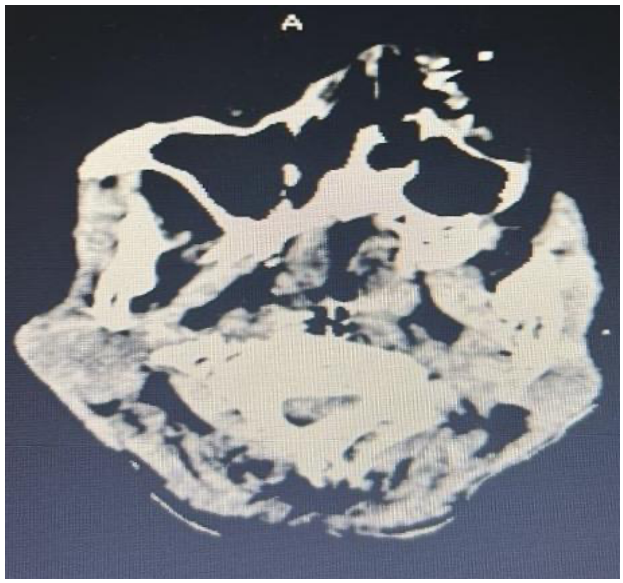
Relevant investigations like BSL, other electrolytes,blood gas analysis, hormone levels like renin were normal.

ECG
Shows possible left ventricular hypertrophy with slight left atrial enlargement
Could be indicative of hypertension



CT
Ill-defined hypodense area noted in left capsuloganglionic region with mean CT attenuation value of 22-25HU
There is prominence of Sulcus spaces, Sylvian fissures, basal cisterns on both sides s/o generalized cerebral atrophy.





MRI
Consistent finding of PRES (posterior reversible encephalopathy syndrome).

DIAGNOSIS

Uncontrolled hypertension and hyponatremia leading to metabolic encephalopathy

THERAPEUTIC INTERVENTION

The patient was given Labetolol 20mg IV stat, and was started on 3% NaCl infusion at a rate of 18 ml/hr to correct hyponatremia. Injection Strocit 500 mg was given stat. Injection Ceftriaxone 2 g was administered twice daily (bd) as the antibiotic of choice, replacing Augmentin. Injection Mannitol 20% (300 ml) was given half stat to manage cerebral edema. For cardio protection and lipid control, Tab Ecosprin 75 mg once daily and Tab Atorac 40 mg at bedtime were prescribed. Tab Temson 40 mg once daily was continued for hypertension. Nebulization with Duolin and Budecort was given for respiratory support in the earlier phase. Injection Kesol (1 ampoule in 500 cc NS) was infused to manage raised intracranial pressure and inflammation. The patient was advised restricted oral intake with total liquids less than 1.2 L/day, with coconut water sips allowed. Pain relief was provided with Injection Tramadol 1 ampoule in 100 ml NS once daily. Serum sodium and potassium were monitored (noted as 138.8 mg/dl and 3.6 mg/dl respectively), and blood pressure recorded as 146/90 mmHg. The ongoing dose of Ramipril was continued for hypertension management.

INFORMED CONSENT

The patient was first informed about the study, and then informed consent was obtained

DISCUSSION

Metabolic encephalopathy represents a diffuse, reversible dysfunction of cerebral function resulting from systemic metabolic disturbances rather than structural brain lesions. Among the various metabolic causes, hyponatremia is one of the most frequent and clinically significant contributors to altered mental status. It produces neurological manifestations due to osmotic imbalance and resultant cerebral edema, leading to impaired neuronal excitability and transmission. In the present case, the patient had chronic hypertension, which may have contributed to impaired cerebral autoregulation.

This case reinforces the importance of considering metabolic causes in any hypertensive patient presenting with altered sensorium, even when neuroimaging is normal. A multidisciplinary approach involving physicians and neurologists is crucial for accurate diagnosis, safe correction of sodium imbalance, and prevention of complications

CONCLUSION

This case of multiple encephalopathy caused by untreated hypertension in elderly80-year-old female without prior trauma or surgical intervention underscores the importance of medical management and intervention in managing hypertension. Prompt medical treatment, including investigation, was crucial in controlling the worsening condition and preventing long term complications. This case highlights the need for continued management of hypertensive patients and ensuring optimal outcomes, especially in elderly, comorbid individuals.

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