How to Cite:
Chandrakaladar, K., Priyanka, A. M. V., Puja, B. B., Prasanthi, K. P., Begum, S. A., & Kosanam, S. (2022). A case report on metabolic encephalopathy. *International Journal of Health Sciences*, *6*(S3), 3027–3031. https://doi.org/10.53730/ijhs.v6nS3.6252

A case report on metabolic encephalopathy

K. Chandrakaladar
Pharm D students, Department of Pharmacology and Pharmacy Practice, Nirmala College of Pharmacy, Mangalagiri, Andhra Pradesh, India 522503

A. M. V. Priyanka
Pharm D students, Department of Pharmacology and Pharmacy Practice, Nirmala College of Pharmacy, Mangalagiri, Andhra Pradesh, India 522503

L. Bhavana Bhavani Puja
Pharm D students, Department of Pharmacology and Pharmacy Practice, Nirmala College of Pharmacy, Mangalagiri, Andhra Pradesh, India 522503

K. Priya Prasanthi
Pharm D students, Department of Pharmacology and Pharmacy Practice, Nirmala College of Pharmacy, Mangalagiri, Andhra Pradesh, India 522503

Shaik Asha Begum
Assistant Professor, Department of Pharmacology and Pharmacy Practice, Nirmala College of Pharmacy, Mangalagiri, Andhra Pradesh, India 522503

Sreya Kosanam
Assistant Professor, Department of Pharmacology and Pharmacy Practice, Nirmala College of Pharmacy, Mangalagiri, Andhra Pradesh, India 522503

***Abstract*---**A 70 years male patient was admitted in hospital with complaints of sudden onset of drowsiness, confusion, fatigue, and generalised weakness since few hours, anxiety, palpitations, paraesthesia all over the body. He is having a poor stream of urine and he is having type-2 DM, hypertension and on medication. He is also having coronary artery disease and have undergone PTCA stunt.

***Keywords*---**encephalopathy, diabetes, hypertension, ketoacidosis.

Introduction

A metabolic encephalopathy is a change in consciousness caused by diffuse or global brain dysfunction caused by impaired cerebral metabolism. Metabolic encephalopathies, which are usually multifactorial in nature, can be serious

***Corresponding author**: Chandrakaladar, K.; Email: kchandrakaladar9@gmail.com*
Manuscript submitted: 18 Dec 2021, Manuscript revised: 27 March 2022, Accepted for publication: 09 April 2022

complications of many diseases in patients in the intensive care unit. As a result of a specific enzyme deficiency, a number of specific metabolites are overproduced; in excess, these metabolites act as an endogenous toxin, impairing other metabolic or physiological processes. Metabolic encephalopathy is a diffuse but potentially reversible disorder of cerebral function caused by a metabolic or toxic cause that frequently impairs arousal and cognitive function [1].

The metabolic encephalopathies are a group of neurological disorders that are caused by systemic illness, such as diabetes, liver disease, renal failure, and heart failure, rather than by primary structural abnormalities. Metabolic encephalopathies are usually acute or subacute in onset and are reversible if the underlying systemic disorder is treated. However, if untreated, metabolic encephalopathies can cause secondary structural damage to the brain. There are two types of metabolic encephalopathies: those caused by a lack of glucose, oxygen, or metabolic cofactors (typically vitamin-derived), and those caused by peripheral organ dysfunction. Metabolic encephalopathy is caused by many factors such as diabetes, liver disease, kidney failure, or heart failure, makes it hard for the brain to work. For example, if blood sugar gets too high in diabetes, it can lead to confusion and even a coma [2].

Signs and symptoms

- Delirium is considered the most common symptom of metabolic encephalopathy.
- Dementia (loss of memory)
- Ataxia (difficulty coordinating with motor tasks such as walking, eating, writing, or some daily activities)
- Decreased consciousness.
- Coma.
- Insomnia
- Depression

Case report

A 70 years male patient was admitted in hospital with complaints of sudden onset of drowsiness, confusion, fatigue, and generalised weakness since few hours, anxiety, palpitations, paraesthesia all over the body. He is having a poor stream of urine and he is having type-2 DM and he is on medication tab. Glimi-M- 1/500 mg and also, he is having past history of hypertension and on medication tab. Metop-AM- 50/5mg. He is also having coronary artery disease and have undergone PTCA stunt his appetite is normal, sleep-normal, bowels- regular.

Lab investigations

Haematology	Abnormal values	Normal values
WBC	17700c/cu.mm	4400-11000
RBC	3.96m/cu.mm	4.50-6.50
HAEMOGLOBIN	7.6g/dl	14.0-18.0
PCV	24.8%	42.0-54.0

MCV	62.7FL		80.0-96.0	
MCH	19.2 pg		27.0-31.0	
MCHC	30.6g/dl		32.0-37.0	
RDW	20.6%		11.6-14.0	
DIFFERENTIAL COUNT				
NEUTROPHILS	76.0%		40.0-75.0	
LYMPHOCYTES	9.0%		20.0-40.0	

Parameters	Day-1	Day-2	Day-3	Normal values
Serum sodium	123.0mmol/l	129mmol/l	132.0mmol/l	134.0-145.0
Serum chloride	90.1mmol/l	-	-	95.0-105.0

Other investigations

- Plain ct scan of chest- no consolidations/ no ground glass opacities calcified granuloma along right major fissure degenerative changes in thoracic spine cholelithiasis.
- MRI brain (plain) – chronic lunular infarcts in right corona radiate and in right frontal lobe. Small vessel changes in bilateral corona radiata and centrum semiovale. Age related cerebral entropy.
- Real time ultrasonography of the abdomen– grade iii prostatomegaly, cholelithiasis, and grade i fatty liver.

Treatment

The treatment as follows:
DAY – 1 to 3
Inj. PIPTAZ – 4.5gm – BD,
Inj. PAN – 4omg – OD,
Inj. DOXYAEQ – 100mg – BD,
Tab. Glimy-M1 – 1/500mg – OD
Day 2 to 3:
Tab. STORVATRIO – 75/75/20 – OD
Tab. METOP- AM – 50/5mg – OD

Discussion

The literature describes encephalopathy caused by hyperglycemic emergencies, but the underlying mechanisms are unknown. 1 The metabolic disturbances that occur during diabetic ketoacidosis/hyperosmolar hyperglycemia can result in a very similar clinical picture. 1 Furthermore, there are a number of potential confounding factors, such as drug and alcohol use prior to presentation, variability in protocols and practise for the treatment of adult hyperglycemic emergencies, and coincidental presentation of encephalopathy due to another cause in a diabetic patient with secondary metabolic disturbance.

Glucose is an essential substrate for brain metabolism, and it must be available at all times for the brain to function. As a result, maintaining stable plasma glucose levels is critical for survival. Significant changes in plasma glucose levels, such as hyperglycemia, can mimic the symptoms of an acute myocardial

infarction. Diabetic ketoacidosis is more common, and this patient is at risk of developing renal injury due to decreased urine output and altered serum electrolytes (Table 1). Metformin use can result in result in drug accumulation in the brain, resulting in neurological abnormalities, speech and walking difficulties, worsening sensory disturbance, tiredness, drowsiness, and weakness. Metformin is a first-line treatment for type 2 diabetes. However, because of an increased risk of lactic acidosis, it is contraindicated in patients with an estimated glomerular filtration rate of 30 mL/min/1.73 m2[3].

Inadequately controlled primary hypertension is the most common cause of hypertensive encephalopathy. Secondary hypertension causes can also predispose patients to this condition. Normally, the brain maintains blood flow within a narrow perfusion pressure range unaffected by changes in systemic arterial pressure. In chronic hypertension patients, the cerebral vasculature adapts, such as arteriolar hypertrophy, to allow for a greater autoregulatory range. In these patients, lowering blood pressure too quickly can result in cerebral ischemia at a higher MAP than in normotensive patients[4].

Usually, patients in this condition will have thamin deficiency, but hospital have not advised to check thamin levels and also, they have given only symptomatic treatment. Malnutrition, osmotic diuresis, insulin deficiency, and even insulin administration can all deplete the body's thiamine stores. Neuronal damage caused by thiamine deficiency affects specific vulnerable regions in animal models, including the thalamus, inferior colliculus, mammillary body, medial geniculate nucleus, and medial vestibular nucleus. Pathophysiological mechanisms underlying this neuronal insult include cellular endothelial dysfunction caused directly by thiamine deficiency and low levels of brain neurotransmitters such as GABA, glutamate, and serotonin, all of which rely on thiamine for production [5].

As a result, we believe that our patient suffered from a non-specific and reversible encephalopathy caused by diabetis. To this day, despite careful investigation into the events preceding his presentation and in patient management, as well as extensive searches of the medical literature, we have been unable to identify a clear cause of encephalopathy other than diabetic ketoacidosis. We present this case to raise medical professional awareness of the importance of early detection and treatment of reversible causes of an encephalopathic state in future patients [6].

References

1. Berisavac II, Jovanović DR, Padjen VV, Ercegovac MD, Stanarčević PD, Budimkić-Stefanović MS, Radović MM, Beslać-Bumbaširević LG. How to recognize and treat metabolic encephalopathy in Neurology intensive care unit. Neurology India. 2017 Jan 1;65(1):123.
2. Ishizaki Y, Nishizono R, Kikuchi M, Inagaki H, Sato Y, Fujimoto S. Case Report: A Case of Encephalopathy Presenting the Lentiform Fork Sign on MRI in a Diabetic Dialysis Patient. F1000Research. 2020;9.
3. Jeon SJ, Choi SS, Kim HY, Yu IK. Acute acquired metabolic encephalopathy based on diffusion MRI. Korean Journal of Radiology. 2021 Dec;22(12):2034.

4. Berisavac II, Jovanović DR, Padjen VV, Ercegovac MD, Stanarčević PD, Budimkić-Stefanović MS, Radović MM, Beslać-Bumbaširević LG. How to recognize and treat metabolic encephalopathy in Neurology intensive care unit. Neurology India. 2017 Jan 1;65(1):123.
5. Potter T, Schaefer TJ. Hypertensive Encephalopathy.
6. Tomkins M, McCormack R, O'Connell K, Agha A, Merwick Á. Metabolic encephalopathy secondary to diabetic ketoacidosis: a case report. BMC Endocrine Disorders. 2019 Dec;19(1):1-8.