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Case Report on Metabolic Encephalopathy.

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Abstract:

Background: Encephalopathy means disorder or damage of brain . In modern use the term encephalopathy doesn't refer to a single disease bit rather to a syndrome of overall brain dysfunction.. So Metabolic Encephalopathy is a symptom of organ dysfunction caused by pathological conditions such as diabetes, cardiovascular disease, liver disease, and renal failure. The most common cause of consciousness abnormalities in systemic disease is metabolic encephalopathy, which is defined as a change in consciousness induced by distributed or global brain dysfunction due to decreased cerebral metabolism. Mostly occurs around 65 years of age. In this conditions damage the brain and patient have unconscious status. Diabetic brain ketoacidosis can cause metabolic encephalopathy, which is a rare but potentially fatal consequence. This case illustrates a young man's rapid cognitive impairment as a result of a metabolic encephalopathy complication. The goal of these case studies is to illustrate metabolic issues.

Main symptoms and /or important clinical finding : Altered behavior in 15days , relevant talk in 15days. Generalised weaknesses 15days ,patient lead H/O failure episode in hospital the blood test are done in which total WBC count are 11400, HB level 8.4. , total RBC count 3.41. Sodium 125, albumin 2.9, lactate 2.1, total protein 4.9.

The main diagnose therapeutic interventions and outcomes: Inj. Piptaz 4.5mg tds, Inj. Pantoprazole 40mg od , Inj. Emset 4mg tds, Inj. Livepsy 500mg BD , as per doctor's advices.

Conclusion : Metabolic encephalopathy is a serious condition and can be controlled with timely initiation of treatment and care.

Keywords : Diabetic Ketoacidosis, Devastating, Irrelevant, Encephalopathy, Metabolic, Behaviour.

Introduction :

The most common cause of discorded consciousness in systemic disorders is metabolic enecephalopathy.[1] which is described as a change in consciousness induced by diffuse or global brain dysfunction due to decreased cerebral metabolism.[2] Mostly occur in 65 years age. This conditions damage the brain and patient have unconscious status .Metabolic encephalopathy is a very uncommon but potentially fatal condition. The significant cognitive impairment of a young guy due to metabolic encephalopathy worsening DKA is highlighted in this instance the case report purpose is to raise awareness of encephalopathy of metabolic as a consequence of Diabetic ketoacidosis examine current research in DM

brain injury.[3] It is also demonstrated the importance of investigating and treating reversible causes of enceephalopathy.[4]

Patient Information: This 67 years old case was admitted to AVBRH, Wardha.

Relevant Past History: Present case visited in MGIMS Sevagram, for the Management of altered behaviour

Clinical findings: Physical and mental status examination was done and irrelevant talk and unconscious is present.

Timeline: Present case has history of altered behavior, irrelevant talk, weakness, since from 15 days. And he visit in MGIMS hospital in 07 January for management. Blood, spinal fluid and urine test was done. And detect of metabolic encephalopathy. And then follow up to AVBRH for further treatment, patient was visited in AVBRH hospital on opd based with Chief compliant of altered behavior, irrelevant talk, altered , dementia and jaundice since from days.

Diagnostic assessment: In physical examination fear and anxiety on face, all Routine blood test was done, sodium, platelets and WBC was decreased.

Diagnosis: After the mental status examination and dignostic procedure like a arterial blood gas analysis. laboratory analysis, electroencephalography doctor diagnosed a case of metabolic encephalopathy.

Prognosis: Present case of prognosis was fair.

Therapeutic interventions: Medical management provided to the patient. Tab. Temain 20mg , antibiotics etc.

Follow up and out comes: Patient had a history of irritability, loss of memory, weakness and they visit to Sevagram hospital all investigations was done and detect the case of metabolic encephalopathy. Patient was refered to AVBR hospital,

Discussion:

DKA-induced brain damage has been thoroughly documented in both populations, according to a review of the literature, but The processes of brain injury, on the other, are unknown. This uncertainty is exacerbated by the numerous metabolic abnormalities that occur with DKA. [6]. In the adult population, There are few cases of metabolic encephalopathy documented caused by DKA. Miras et al. documented a 44-year-old male who had a very similar condition[5]. He has a background as well as of excessive intake of alcohol consumption and poorly controlled type 1 DM, with recurring severe hypoglycemic episodes. The client in this example had substantial behavioural issues, including violence and bewilderment. The neuroimaging was normal, the CSF was tasteless, and the EEG showed generalised slowing, all of which are signs of encephalopathy. During the next six months, this patient's irritation and slow speech improved steadily with neuropsychiatric therapy, although irritation and sluggish speech persisted. [5].

Although diabetic ketoacidosis is showed cause brain injury, there specific pathophysiology this in injury is still unknown. Subclinical cerebral oedema is prevalent in children, with oedema in the brain occurring

in 0.5–1% of children with diabetic acidosis [7]. Previously, edema in cerebral caused by Diabetic acidosis was assumed to be caused by excessive fluid resuscitation and a lack of brain osmotic equilibrium, however this hypothesis has since been debunked. Hypoperfusion of the brain followed by reperfusion damage coincides with the wide range of cytotoxic and vasogenic oedema that can be detected in individuals with diabetic acidosis-induced cerebral oedema, and is currently thought to be the cause of brain injury in diabetes acidosis patients[7]. Cerebral oedema in adults with DKA or hyperglycemic hyperosmolar syndrome (HHS), on the other hand, is extremely uncommon with a 0.03 percent incidence rate reported in a large population-based study in the United States[8]. Because No clinical or radiological evidence was found of cerebral oedema in the case described above, other causes of brain injury must be at work. Jessup et al., for example, evaluated a sample of young patients with new-onset type 1 diabetes and discovered that patients with DKA performed worse on visual cognitive tests than age-matched patients without DKA. 8–12 weeks after discharge, the cognitive gap between the two groups maintained. According to the scientists, metabolic imbalance during DKA causes neuroinflammation and oxidative stress in the brain, resulting in neuronal damage. [9]. This is backed by histology research, which demonstrated signs of oxidative stress in brain tissue from individuals with DKA and cerebral oedema, with higher oxidative damage products found in sensitive brain locations as compared to healthy controls [10]. Hoffman et al have discovered that T1DM can affect brain function in both acute and chronic metabolic dysregulation, promoting neuroinflammation, cerebral insulin resistance, and reduced insulin signaling, all of which lead to increased oxidative and inflammatory cerebral stress. [10].DKA, which is caused by chronic hyperglycemia, ketoacidosis, and dehydration with abrupt shocks, can lead to diabetic encephalopathy [10]. The patient's cognitive decline may have been exacerbated by the patient's long term inconsistent glycemic management in situation described above.

The research into brain metabolites and the application of magnetic resonance spectroscopy in diabetic brain illness is still in its early phases. More research and development could reveal the specific mechanism of diabetic brain injury, as well as produce a new diagnostic tool to assess early disease and allow intervention. [11]. Magnetic resonance spectroscopy (MRS) analysis of brain metabolites indicates significant changes in levels of brain metabolites in DM brains, which are consistent with and connected to specific diabetic problems. There have been changes in these metabolites. Neurotransmission is likely to be reduced, as well as demyelination and brain shrinkage. There are further nutritional factors that may have elevated the risk of brain injury in the situation described above. In young adults, folic acid insufficiency has been associated to cognitive impairment and behavioral instability[12]. Seizures, hemichorea, and hemianopia are some of the neurological repercussions of non-ketonic hyperglycemia (NKH). Hyperglycemia-induced permeability of the brain of blood in barrier contributes to epileptogenesis in NKH [13]. As a result, we can speculate that recurrent hyperglycemia and consequent brain- blood barrier permeability may have played a role in epileptogenesis and, in this case, brain damage. Furthermore, whereas permeability of the blood-brain barrier is not the major disease in MS, It could have resulted in a reduction in neuroprotection and a higher risk of encephalopathy.[14]. Other related studies were reviewed[15-20].

Conclusion : Metabolic encephalopathy is a serious condition and can be controlled with timely initiation of treatment and care.

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