International Journal of Scientific Research & Engineering Trends
Volume 7, Issue 4, July-Aug-2021, ISSN (Online): 2395-566X

Hypoglycemic Encephalopathy

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Abstract- We report the case of 23-year-old man without medical history who intentionally ingested a high dose of biguanide. He was found lying in a coma state with low blood glucose, transferred to the emergency department for non resolved biguanide intoxication.

Keywords- Hypoglycemia, Encephalopathy, Biguanide, MRI.

I. INTRODUCTION

1. History:
We report the case of 23-year-old man without medical history who intentionally ingested a high dose of biguanide. He was found lying in a coma state with low blood glucose at the emergency department non resolved after resuscitation.

This is a 23-year-old young man with no medical or surgical history who was found in a coma state in his room by his family members, after which he was transferred to the emergency department intensive care unit. During questioning, the family reported the notion of excessive consumption of narcotics which turned out to be biguanides. Clinical examination revealed a comatose patient with a Glasgow score of 3, tachycardia, profuse sweating, tachypnea and a body temperature of 37 ° with a retained urine output.

Blood glucose examination revealed a profound hypoglycemia measured at 0.2g / l in the absence of ketone bodies on the urine dipstick, elevated plasma metforminemia and metabolic acidosis. To overcome severe hypoglycemia, resuscitation with 30% hypertonic glucose serum intravenously was done, in addition to the correction of metabolic acidosis, and the purification of metformin by plasma exchange.

Despite the monitoring and in the face of the prolongation of the coma state, a brain MRI was set in emergency that revealed diffuse cortical and subcortical lesions as well as basal ganglia on the supratentorial level, T2 and diffusion hypersignals (Figure 1), (Figure 2)), without contrast enhancement after injection of gadolinium (Figure 3).

II. DIAGNOSTIC

Faced with the picture of hypoglycemic coma due to biguanide intoxication, the lack of recovery of consciousness after treatment and the brain lesions on MRI we concluded to the diagnosis of hypoglycemic encephalopathy.

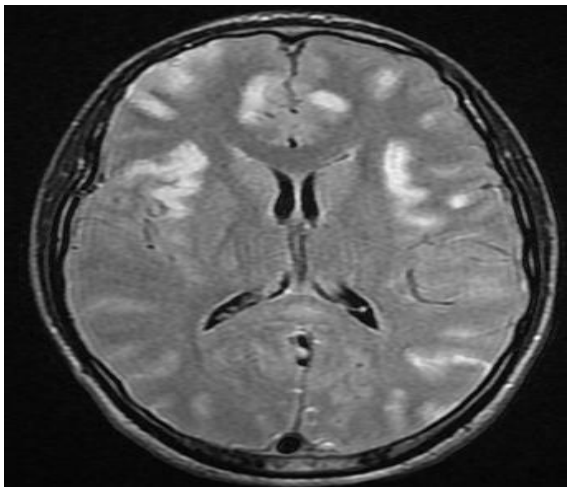

Fig 1. Axial T2-weighted FLAIR slices showing diffuse cortical and subcortical hyper intensity lesions of the island, frontal and left occipital lobes.

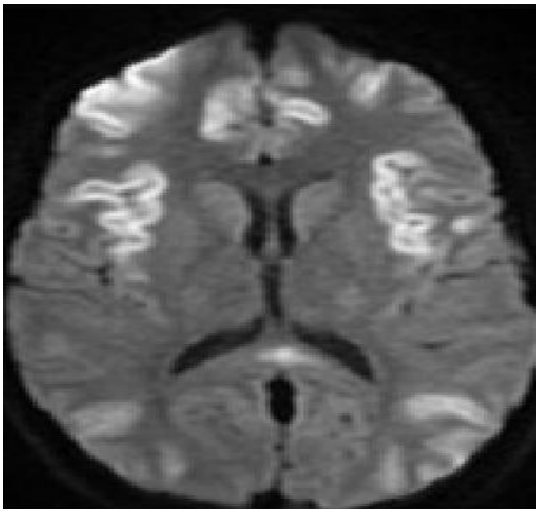

Fig 2. Axial section in diffusion sequence with a value of B to 1000 showing a restriction of the diffusion of lesions.

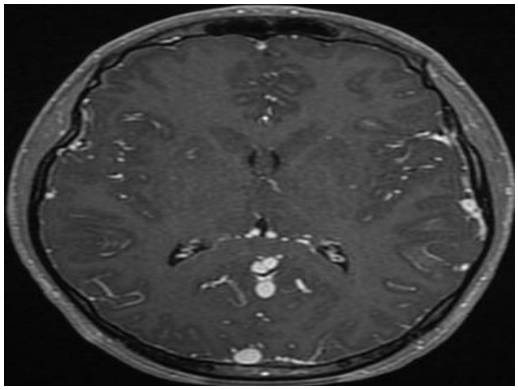

Fig 3. T1-weighted axial slice with fat saturation and gadolinium injection showing the absence of contrast enhancement of the lesions.

III. COMMENTS

Hypoglycemic encephalopathy (HE) is defined by brain damage secondary to hypoglycemic coma or stupor with blood glucose levels <50 mg / dl on admission, or a persistence of coma for ≥ 24 hours despite normalization of the blood sugar, after ruling out any other cause. Clinically severe signs of hypoglycemia are present, such as altered consciousness, coma state, seizures, or signs of focusing [1, 2].

The main source of energy in the brain is the glucose oxidation. The cerebral cortex, hippocampus, cerebellum, caudate nucleus and pallidum exhibit higher energy consumption with greater sensitivity to hypoglycemia.

As a result, when blood sugar levels are low, these areas are affected; significant damage is caused first, followed by extensive neuron denaturation and necrosis of, as well as infiltrates made up of glial cells. Therefore, brain damage secondary to hypoglycemia is highly selective [2]. Radiologically, the key examination remains MRI, which finds typical signal abnormalities T1 hyposignals without contrast enhancement, T2 and Flair hypersignals with restriction of diffusion in the acute phase, which are bilateral and roughly symmetrical.

These anomalies mainly concern the posterior aspect of the limbus, internal capsules, cerebral cortex (more specifically the parietal, occipital and insular lobes), hippocampus and basal ganglia. The corpus callosum splenium can also be affected [1, 3].

The differential diagnosis arises mainly with hepatic encephalopathy, osmotic myelinolysis, CO poisoning, methanol intoxication, ethylene glycol intoxication, metronidazole intoxication, cyclosporine intoxication, and cocaine encephalopathy [1, 4]. The treatment lies in the correction of hypoglycemia and the control of systemic parameters of secondary cerebral aggression and the etiological treatment in our case is based on the correction of metabolic acidosis, the purification of metformin and the treatment of a possible intercurrent pathology. Extra-renal epuration is the gold standard in metabolic acidosis due to drug intoxication, especially if it is associated with renal failure and volume inflation.

The prognosis is directly related to the severity, duration, and depth of hypoglycaemia. In the most severe cases this can lead to death, irreversible coma or hemiplegia or hemiparesis-like sequelae [4].Declaration of conflict of interest: The authors declare that there is no conflict of interest.

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