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Hypoglycemic Encephalopathy in a 46-Year-Old Patient Following Sulfonylurea Overdose: A Case Report

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Abstract **Case Report**

Hypoglycemic encephalopathy is defined by coma or altered consciousness in patients with a blood glucose level below 50 mg/dl persisting for more than 24 hours despite normalization of glucose levels, without any other obvious cause [1]. It is a rare but serious complication with a mortality rate that can reach 50%, often associated with visible structural anomalies on Magnetic resonance imaging (MRI) [2]. We report the case of a 46-year-old patient admitted to the intensive care unit for altered consciousness secondary to voluntary sulfonylurea overdose. The MRI performed on the seventh day of hospitalization revealed bilateral cortico-subcortical and basal ganglia abnormalities characteristic of hypoglycemic encephalopathy. The clinical course was marked by a progressive improvement in neurological status.

Keywords: Hypoglycemia, Encephalopathy, Basal Ganglia, Hyperintensity.

INTRODUCTION

Prolonged hypoglycemia can lead to severe neuronal damage, particularly in vulnerable regions such as the cortex, hippocampus, and basal ganglia (BG) [3]. MRI plays a crucial role in the evaluation of these anomalies, although the correlation between imaging results and clinical evolution remains complex [4]. Here we present an illustrative case of hypoglycemic encephalopathy.

OBSERVATION

A 46-year-old male patient, homeless and addicted, with a history of severe depressive syndrome, was admitted initially to the emergency department for management of an indefinite-duration, afebrile altered consciousness. Clinical examination revealed a neurological evaluation: GCS 7, no deficits, equal reactive pupils, present brainstem reflexes, and absence of meningeal signs. Respiratory assessment showed bradypnea, auscultation without abnormalities, and absence of cyanosis. Hemodynamic status revealed tachycardia at 115 bpm and blood pressure at 110/65 mmHg. He was found in possession of a blister pack of sulfonylureas with 15 missing tablets. Initial blood glucose reading was 0.4 g/L. A bolus of 30% dextrose was administered, followed by a continuous infusion of 10% dextrose, with persistent deep coma necessitating endotracheal intubation and sedation.

The initial cerebral CT scan showed no specific abnormalities.

The biological assessment (complete blood count, renal and liver function tests, calcium, blood electrolytes, arterial blood gases, toxicological examination) was inconclusive: calcium, sodium, urea, creatinine, and liver function tests were normal.

Toxicology screening demonstrated the presence of a large quantity of sulfonylurea in the gastric fluid.

The patient was placed on symptomatic treatment consisting of a continuous infusion of 10% dextrose, gastric protection, sodium valproate for anti-epileptic prophylaxis, and thromboprophylaxis.

Due to a lack of improvement after sedation was stopped, a brain MRI performed on the seventh day of hospitalization showed discontinuous signal abnormalities in the left insular, hippocampal, and parieto-occipital cortex, with hyperintensities on T2 and FLAIR, restricting diffusion. This was associated with signal abnormalities in the cortico-subcortical areas,

right parieto-occipital regions, bilateral cerebellar areas, external capsules, and lenticular nuclei, with hyperintensities on FLAIR but no diffusion restriction.

Tracheostomy was discussed, but the patient was extubated on day 12 after respiratory weaning, with a Glasgow Coma Scale (GCS) of 10.

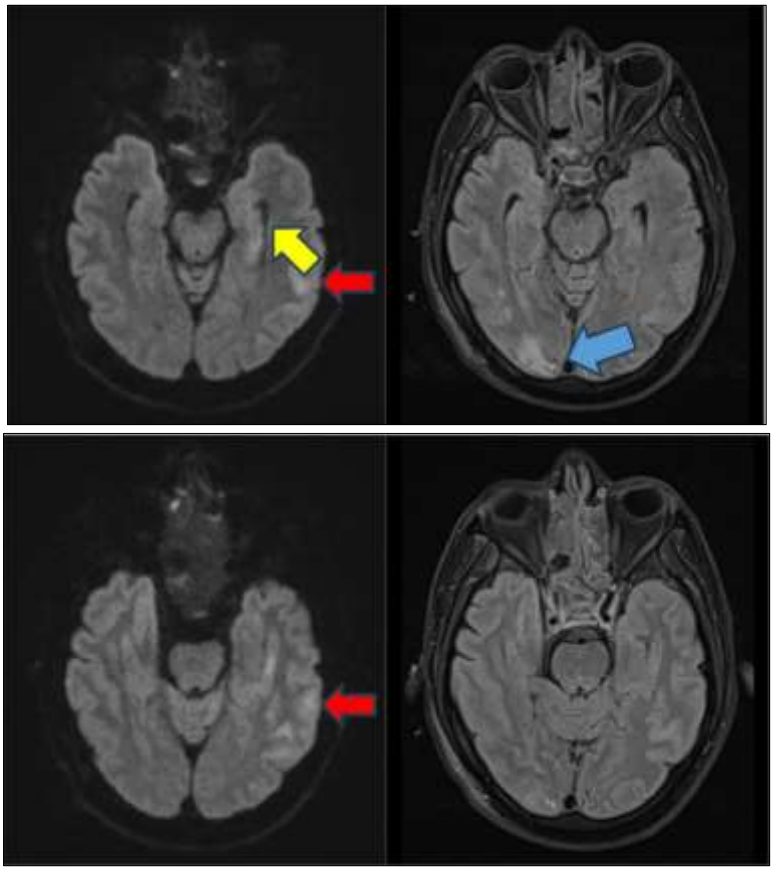
DISCUSSION

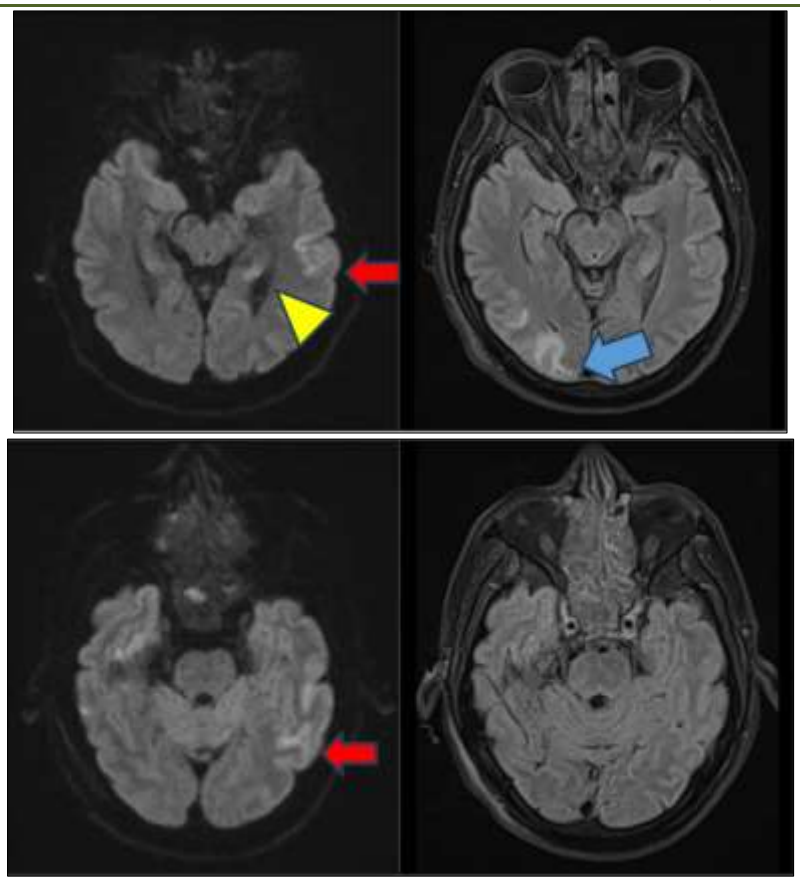
Glucose is the main energy source for the nervous system, and severe hypoglycemia can lead to neuronal death [5]. The clinical signs of hypoglycemia are complex and related to the duration of hypoglycemia and the extent of sequelae [5].

Cerebral energy deficit reduces intracellular protein synthesis, leading to dysfunction of the ion pumps of the cell membrane, causing water to move from the extracellular to the intracellular space [3]. This results in cytotoxic edema affecting the white matter, basal ganglia, hippocampus, and cerebral cortex. The brainstem is generally spared due to increased local glucose transport systems [3].

Changes in neurotransmitter regulation also play an important role in the pathophysiology of hypoglycemic neuronal lesions [3]. Glucose oxidation produces precursors for neurotransmitters, notably glutamate and acetylcholine [3]. Glutamate and glutamine provide another energy source, leading to intracellular alkalosis due to ammonia and accumulation of aspartate in the extracellular space, which can result in selective neuronal necrosis [3]. Moreover, the excitotoxic activity of glutamate has been associated with edema of glial cells and myelin sheaths [3].

The main neuroradiological characteristics reported in cases of hypoglycemia are specific lesions of white matter or alterations affecting both white and gray matter [6]. Selective white matter lesions are typically reported in the corona radiata, corpus callosum, and internal capsule, but rarely in the ponto-mesencephalic region. Lesions associated with hypoglycemia appear as hyperintensities on T2/FLAIR and DWI sequences [6]. These neuroradiological lesions may be reversible after normalization of serum glucose levels [6].





Diffusion (left) and FLAIR (right) axial sequences of a brain MRI showing signs of hypoglycemic encephalopathy in a 47-year-old patient with severe hypoglycemia following sulfonylurea poisoning.

- Discontinuous signal abnormalities in the left insular cortex and left parieto-occipital cortex (red arrow), as well as left hippocampal (yellow arrows) hyperintensities on FLAIR, with restricted diffusion.
- Associated with this are cortico-subcortical signal abnormalities in the right parieto-occipital region with hyperintensities on FLAIR, without diffusion restriction (blue arrows).

Analysis of responsible medications in diabetic patients revealed that the use of sulfonylureas was a major risk factor for severe hypoglycemia, particularly in elderly patients. Patients experiencing severe hypoglycemia induced by sulfonylureas were older than those treated with insulin, in both the general population of patients with severe hypoglycemia and in type 2 diabetic patients [5]. The risk of hypoglycemia under sulfonylureas is especially heightened in the elderly, malnourished, and renal insufficient patients [5]. In our case, the non-diabetic patient ingested a number of sulfonylurea tablets for suicide purposes.

Treatment

Studies indicate that clinically profound and persistent hypoglycemia is associated with poor prognosis. Several studies have sought to find an association between neuroradiological patterns and clinical outcomes [3]. Research findings show that only lesions isolated to the internal capsule are associated with complete recovery, while extensive white matter lesion White matter or basal ganglia lesions are associated with poor short-term prognosis [3].

CONCLUSION

This case illustrates the severity of prolonged hypoglycemia associated with sulfonylurea poisoning. Early management and the use of diagnostic tools such as MRI are crucial to limit complications. A multidisciplinary approach and prolonged neurological follow-up are necessary to optimize prognosis.

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