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Hypoglycemia mimicking ischemic stroke

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Abstract
Acute hemiparesis is often an early presentation of acute stroke, although it can occur in the setting of various disorders such as hypoglycemia. We analyzed the clinical and radiological data of 6 patients initially misdiagnosed with cerebral stroke seen in the emergency department between January 2015 and November 2016. Their ages ranged from 30 to 82 years, they presented to the emergency department for a hemiplegia less than 4.5 hours, with a blood glucose <50 mg/dL. All patients were diabetic on insulin therapy, their NIHSS scores ranged from 4 to 13. Four patients had a brain scan, 3 were normal and the fourth had severe leukoencephalopathy. All received intravenous serum glucose and improved completely thereafter.
On presentation of acute hemiparesis, hypoglycemia mimicking a stroke should be considered to avoid misdiagnosis. Physicians should perform an immediate blood glucose measurement before cerebral imaging, for all patient with mellitus diabetes presenting with acute hemiplegia.

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I. Introduction :

Severe hypoglycemia is one of possible cause of mimic stroke. It can produce a neurological deficit. It can cause hemiplegia, aphasia, confusion, or altered consciousness. These neurological symptoms can be resolved immediately or within a few hours by the intravenous glucose administration. Thus, in patients with acute onset hemiplegia, it is important to differentiate hypoglycemia on arrival by immediate measurement of blood glucose. The pathophysiological mechanisms of this neurological deficit are unclear. Here, we present six cases of acute hemiparesis, hypoglycemia mimicking cerebral stroke.

II. Cases series:

Case 1 :
74-year-old patient, with history of mellitus diabetes treated by insulin, admitted to the emergency department 1 hour and 5 minutes after left hemiplegia, left central facial palsy. NIHSS score was at 10/42, her CT brain scan was normal. Capillary glycemia was less than 50 mg/dL. Few minutes after intravenous glucose administration, the NIHSS score was at 0/42

Case 2:
64-year-old diabetic patient on insulin, admitted to the emergency department 2 hours and 40 minutes after right hemiparesis. NIHSS score was at 4/42, her brain CT scan was normal. Capillary glycemia was less than 50 mg/dL. Few minutes after intravenous glucose administration, the NIHSS score was at 0/42.

Case 3:
82-year-old diabetic patient on insulin, admitted to the emergency department 3 hours and 05 minutes after right hemiplegia with aphasia. Her NIHSS score was at 13/42. Cerebral CT scan showed severe leukoencephalopathy. Capillary glycemia was less than 50 mg/dL. After intravenous glucose administration, the NIHSS score went to 0/42.

Case 4:
30-year-old diabetic patient on insulin, admitted to the emergency department 1 hour and 5 minutes after right hemiplegia with central facial palsy and dysarthria. The NIHSS score was at 9/42. Capillary glycemia was less than 50 mg/dL. After intravenous glucose administration, the NIHSS score went to 0/42. Cerebral CT scan was not done.

Case 5 :
70-year-old diabetic patient on insulin, admitted to the emergency department 1 hour after left hemiplegia and altered consciousness. The NIHSS score was at 12/42. Cerebral CT scan was normal. Capillary glycemia was less than 50 mg/dL. After intravenous glucose administration, the NIHSS score went to 0/42.

Case 6:
60-year-old diabetic patient on insulin, admitted to the emergency department 1 hour and 30 minutes after right hemiplegia and aphasia, with NIHSS score at 9/42. Capillary glycemia was less than 50 mg/dL. After intravenous glucose administration, the NIHSS score went to 0/42. the brain CT scan was not done.

TABLE 1. Demographics of the patients

Cases	Gender	Years	Medical history	Delay	Clinic	Side deficit	NIHSS	CT scan
1	F	74	Diabetic on INSULINE	1h05	Hemiplegia CPF	Left	10	Normal
2	F	64	Diabetic on INSULINE	2h40	Hemiparesis	**Right**	4	Normal
3	F	82	Diabetic on INSULINE	3h05	Hemiplegia and aphasia	**Right**	13	Leucoencephalopathy
4	F	30	Diabetic on INSULINE	1h05	Hemiplegia, CPF Dysarthria	**Right**	9	Not done
5	M	70	Diabetic on INSULINE	1h	Hemiplegia And consciousness disorder	Left	12	Normal
6	M	60	Diabetic on INSULINE	1h30	Hemiplegia And aphasia	**Right**	9	Not done

III. Discussion:

Hypoglycemia, defined as a plasma glucose <45 mg/dL (Claire, et al., 2012) , is the most common endocrinological emergency, and can occur in diabetic patients on insulin therapy, sulfonylurea hypoglycemics, or in patients with an insulin-secreting tumor.

We can distinguish three levels of hypoglycemia, the level 1 (mild hypoglycemia) is defined by blood glucose < 70 mg/dL but ≥ 54 mg/dL, the level 2 (moderate hypoglycemia) is defined by blood glucose < 54 mg/dL, it is considered moderate neuroglycopenia, and the level 3 (severe hypoglycemia) is defined by blood glucose < 30 mg/dL, it is considered severe neuroglycopenia that can cause a state of status epilepticus or disorders of consciousness, in case of prolonged neuroglycopenia we can have cortical necrosis or even brain death. (Choi IY et al, 2021)

Glucose is an essential energy substrate for brain function. Focal deficiency hypoglycemia syndrome is rare (Ashish & Kunjan, 2018). Its physio pathological mechanism is not well known. However, current theories include cerebral vasospasm, cerebral blood flow asymmetry and selective neuronal vulnerability (Claire, et al., 2012). This selective neuronal vulnerability frequently affects the dominant hemisphere explaining more frequent right hemiplegia than left. In our series, 4 of the 6 patients had right hemiplegia. (John W. Foster et al., 1987) Reported that right deficit is far more common than left deficit (72 vs. 28%) and is often accompanied by aphasia, these symptoms may also be due to increased epinephrine and norepinephrine secretion (Ashish & Kunjan, 2018).

Decreased ATP and acetylcholine concentration were found in the brains of hypoglycemic rats, but this theory does not explain the focal neurological deficit (Won, et al., 2012) . Other studies have shown that excitatory amino-acids such as glutamate and aspartate were found elevated in the CSF of hypoglycemic animals. Nevertheless, it is widely accepted that severe hypoglycemia causes brain energy degradation and results in reduced cell membrane ion pump activity and a consequent shift of water from the extracellular to the intracellular space. Acute hypoglycemia can mimic an acute ischemic stroke even on brain imaging, particularly MRI. Approximately 20% of cases have been described in the literature, resulting in either unilateral cortical lesion, hypoattenuation and swelling of subcortical tissues, or incomplete hypersignal (Yong, Morris, Shuler, Smith, & Wardla, 2012). Lesions can be seen on diffusion as early as 45 minutes after the onset of the symptoms but do not appear on the T2 sequence until 12 hours after the onset of symptoms. On the brain CT scan, abnormalities are less frequent and appear later (cerebral edema, or hypoattenuation). When they exist, these lesions disappear a few hours to a few days after intravenous glucose administration, and clinical improvement but can be permanent.

IV. Conclusion:

Hypoglycemia, regardless of cause, severity or duration, can mimic an ischemic stroke. Although rare, hypoglycemia should always be considered in the differential diagnosis of acute focal neurological symptoms. Measurement of capillary blood glucose should be a reflex for any physician examining a patient presenting to the emergency department with a neurological deficit.

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M. Jilla, et. al. "Hypoglycemia mimicking ischemic stroke". *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 22(4), 2023, pp. 09-11.