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Case Study

CASE REPORT- ASYMPTOMATIC NEUROGLYCOPENIA WITH COMORBIDITIES

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Abstract

A case of a 65-year-old women with Type 2 diabetes mellitus having comorbidities who developed asymptomatic neuroglycopenia which is also knownas hypoglycaemia unawareness with recurrent episodes of low blood sugar. She presented with confusion and disoriented speech after taking his usual morning dose of medication following a period of poor adherence. The laboratory evaluations confirmed hypoglycemia which leads to neuroglycopenia. Then supportive care was initiated to rule out potential cause after discontinuing oral hypoglycemic agents. This case highlights the increased risk in diabetic patients with other comorbidities like hypertension and hypothyroidism.

Keywords: Diabetes mellitus, neuroglycopenia, hypoglycemia unawareness, hypertension, hypothyroidism.

INTRODUCTION:

Neuroglycopenia is a term that refers to a shortage of glucose in the brain resulting in alteration of neuronal function[1]. One of the major causes of neuroglycopenia is hypoglycemia [2]. In human medicine, hypoglycemia is usuallydefined by a blood glucose concentration below 70mg/dl [3].Neurogenic (autonomic) and neuroglycopenic symptoms occur in humans with hypoglycemic [4,5]Neurogenic symptoms result from the physiologic response to hypoglycemia by the autonomous nervous system which include tremors, palpitations, anxiety, sweating, tachycardia, hunger, and paresthesias [4] Neuroglycopenic symptoms are related to deprivation of low glucose concentration in the brain, which manifest as confusion, sensation of warmth, blurred speech, fatigue, cognitive failure, seizures, coma, and death if untreated [3, 4]. During the early stage of neuroglycopenia, cortical dysfunction might only be recognized through a systematic cognitive testing

[5]. Cerebral cortical dysfunction has been recorded when BG concentration is 36 mg/dl or less in humans and rats.[1, 2] Measurement of glucose concentration in cerebrospinal fluid (CSF) which might aid toinvestigate glucose concentration in the brain [6,7]. Low glucose concentration in CSF is termed hypoglycorrhachia and is most commonly associated with infection (mainly bacterial meningitis) but other causes such as stroke, malignancy, neurosarcoidosis, and severe hypoglycemia are associated with low CSF glucose concentrations [8,9].

CASE PRESENTATION

A 65 years old female patient with a known case of Type – 2 diabetes mellitus; hypertension; hypothyroidism; presented to the emergency department with the chief complaints of confusion, frothing from mouth, decreased responsiveness and asymptomatic from last night prior to arriving to the hospital. Previously, she was on a fixed dose combination of Glimepiride [2mg], and Metformin [500mg] to treat diabetes mellites' Hypertension she was on combination dose of Telmisartan [40mg] and hydrochlorothiazide [12.5mg] with regular dose of Levothyroxine [50mcg] to treat hypothyroidism.
She had a social history of consuming alcohol in every alternative day [whisky]. At the same time, she was allergic to the plantar fasciitis [a pain in the heel and arch of the foot] and she had mild pitting oedema.There is elevated BP, Fluctuation in temperature and pulse rate with normal respiratory sounds which is given clearly in the following table

SYSTEMIC EXAMINATION:

	Day-1	**Day-2**	**Day-3**	**Day-4**	**Day-5**
Temperature[℉]	98	96.5	98	99	99
BP [mmHg]	185/110	190/110	182/100	185/100	186/100
PR [bpm]	110	100	98	100	102
RS	BAE+	BAE+	BAE+	BAE+	BAE+

BP: Bloodpressure, PR: Pulserate, RS: Respiratory system

Detailed laboratory investigations on case report:

Laboratory test	**Day-1**	**Day-2**	**Day-3**	**Normal range**
Hemoglobin	10.2	9.4	9.3	[13-17] g/dl
RBC	3.7	3.48	3.46	[4.0-5.5] million/cumm
WBC	16,200	12,770	11,650	[4000-10000] cells/cumm
ESR	75	70	68	[10-12] mm1st hr.
Platelets	4.8	4.18	4.3	[1-4] lakhs/cum
Glucosuria	Not performed	+	+	<15mg/dl
Hematuria	Not performed	+	+	0-3 RBCs/HPF
FBS	44	50	54	[70-110] mg/dl
HbA1c	6.3	-	-	5.5%

RBC: Red blood cell, WBC: White blood cell, ESR: Erythrocyte sedimentation rate, HPF: High power field

TREATMENT CHART:

S.No.	**Trade name**	**Generic name**	**Dose /ROA**	**Frequency**	**Indication**
1.	T. ROSUVAS	Rosuvastatin	10mg/po	OD	To reduce the risk of heart disease, stroke.
2.	T. TELMA-H	Telmisartan+ hydrochlorthiazide	40+12.5mg/po	OD	To treat hypertension
3.	THYRONORM	Levothyroxine	50mcg/po	OD	To treat hypothyroidism
4.	INJ.LASIX	Furosemide	10mg/IV	STAT	To decrease pitting edema and hypertension
5.	INJ. HAI	Insulin	9.5 u/SC	TID	To treat diabetes

					mellitus
6.	INJ.HYDROCART	Hydrocortisone	10mg/ IV	STAT	To treat adrenal crisis, hypoglycemic in pt
7.	T. DOLO	Paracetamol	650mg/ PO	SOS	To decrease elevated body temperature in middle of therapy
8.	INJ.PAN	Pantoprazole	40mg/ IV	OD	To decrease acid reflux which is caused by other drugs
9.	INJ. THIAMINE	Thiamine hydrochloride	300mg/ IV	TID	To prevent Wernick's encephalopathy in thiamine deficiency patient
10.	INJ. MONOCEF	Ceftriaxone	1gm/ IV	BD	To prevent meningitis and seizures
11.	INJ. ZOFER	Ondansetron	4mg/ IV	SOS	To reduce vomiting during therapy
12.	T. ECOSPRIN	Aspirin	75mg/RT	OD	To prevent myocardial infarction, transient ischemic attack
13.	T. CINOD- T	Clinidipine+Telmisartan	10+40mg/RT	BD	To decrease elevated blood pressure
14.	NEB.DUOLIN	Ipratropium bromide, salbutamol+ budesonide	1 resp/PN	STAT	It helps/ reduce difficulty in breathing as patient is unresponsive
15.	INJ.METROGYL	Metronidazole	100mg/IV	TID	To decrease rate of infection which progress neuroglycopenia
16.	INJ.PIPTAZ With 100ml NS	Piperacillin tazobactam	4.5g/ IV	TID	To treat infection which is caused due to elevated levels of WBC, ESR
17.	1O NS	Normal saline	500ml/IV	Bolus	Given with dextrose to meet with neuroglycopenia [glucose should be monitored]
18.	BECOSULE- Z	B-Complex forte + Vitamin-C+Zinc	1 TAB/PO	OD	To provide enough nutrition to the patient

DISCUSSION

Neuroglycopenia was commonly seen in patients with a history of longstanding Type II diabetes mellitus. But in this case, Hypoglycemia Unawareness was seen in the elderly diabetic patient presenting with neuroglycopenia. Past history of hypertension, hypothyroidism including with type-2 diabetes mellitus are a cause of neuroglycopenia which worsen the current condition. Major diagnoses to identify hypoglycaemia is fasting blood glucose and CT/MRI scan of brain to know the clear investigation, any changes in the brain due to neuroglycopenia which is caused by less amount of glucose in the brain. Pt was unresponsive and had a frothing from mouth. She takes alcohol in every alternative day which may leads to Wernicke's encephalopathy. So, to avoid [or] to prevent this condition thiamine is administered intravenously. This case had a unique clinical feature, comorbidities, management strategies, highlighting the complexity of diagnosing and treating

hypoglycemic events. Despite, variation in presentation, timely recognition and appropriate intervention including adjustment in medication dosage were crucial in achieving favourable outcomes. To minimise drug interactions with current and past medications appropriate measures are taken. Supportive agents like antiplatelets, folic acid supplements should be given to avoid blood clottingand anaemia respectively. Comprehensive management required not only correction of hypoglycemia but also stabilization of comorbid conditions, identification of precipitating factors, and optimization of metabolic control. This case underscores the importance of a multidisciplinary approach, integrating endocrinological, haematological, and systemic evaluation to achieve neurological recovery and prevent recurrence.

CONCLUSION

This is case of asymptomatic neuroglycopenia in a patient with a known history of type 2 diabetes mellitus (T2DM), hypertension (HTN), and hypothyroidism on regular medication therapy underscores the intricate pathophysiological interactions among metabolic, endocrine, hematologic, and lifestyle-related factors. Despite the absence of overt neuroglycopenic symptoms, the biochemical evidence of recurrent hypoglycemia highlights impaired glucose homeostasis likely secondary to antidiabetic therapy, altered hepatic metabolism due to chronic alcohol consumption, and possible blunting of hypoglycemia awareness—a recognized complication in long-standing diabetic patients Thepresence of anaemia (haemoglobin 9–10 g/dL, RBC 3.0–4.5 million/cumm), leucocytosis (WBC >10,000/cumm), and elevated ESR further suggests an underlying chronic inflammatory or infective process, which may exacerbate metabolic instability and contribute totissue hypoxia, thereby potentiating neuroglycopenic risk. Additionally, mild pitting oedema could indicate early nephropathy, fluid retention due to hypothyroidism, or alcohol-related hepatic dysfunction, all of which may compound systemic metabolic stress.

The asymptomatic nature of neuroglycopenia in this patient warrants special attention, as hypoglycemia unawareness increases the risk of severe, unrecognized hypoglycemic episodes that can result in neurocognitive impairment or cardiovascular complications. This emphasizes the necessity of individualized glycaemic target adjustment, cautious pharmacologic management, and regular metabolic monitoring.

In conclusion, this case illustrates the importance of a multidimensional diagnostic and therapeutic approach in patients with overlapping endocrine and systemic disorders. Recognition of subtle metabolic disturbances, even in the absence of clinical symptoms, is essential for preventing serious complications. Integrated management involving optimization of diabetic therapy, correction of anaemia, evaluation of inflammatory markers, and modification of lifestyle factors particularly alcohol cessation plays a pivotal role in improving overall prognosis and quality of life.

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