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A case of hypoglycemic coma in 62 years old male patient

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Abstract

Hypoglycemia is low blood glucose or low blood sugar, occurs when blood glucose drops below normal levels. Hypoglycemic coma is an internal medicine emergency caused by a serious reduction in blood glucose. We recorded a case of hypoglycemic coma in 62 years old male.

Keywords: Blood glucose, Coma, Hypoglycemia

Introduction

Hypoglycemia, also called low blood glucose or low blood sugar, occurs when blood glucose drops below normal levels. Glucose, an important source of energy for the body, comes from food. Carbohydrates are the main dietary source of glucose. Rice, potatoes, bread, tortillas, cereal, milk, fruit, and sweets are all carbohydrate-rich foods ^[1].

When blood glucose begins to fall, glucagon- another hormone made by the pancreas- signals the liver to break down glycogen and release glucose into the bloodstream. Blood glucose will then rise toward a normal level ^[2]. In some people with diabetes, this glucagon response to hypoglycemia is impaired and other hormones such as epinephrine, also called adrenaline, may raise the blood glucose level. But with diabetes treated with insulin or pills that increase insulin production, glucose levels can't easily return to the normal range. Hypoglycemia can happen suddenly. It is usually mild and can be treated quickly and easily by eating or drinking a small amount of glucose-rich food. If left untreated, hypoglycemia can get worse and cause confusion, clumsiness, or fainting. Severe hypoglycemia can lead to seizures, coma, and even death ^[3].

Hypoglycemic coma is an internal medicine emergency caused by a serious reduction in blood glucose. Transient hypoglycemic episodes are common, especially in diabetic patients, and are usually not life-threatening or associated with persistent neurological deficits or organ damage. Severe persistent hypoglycemic states, however, may cause long-lasting coma, seizures, and a myriad of other global and focal neurologic deficits, some of which may be mistaken for stroke or other acute neurological disorders. In analogy to hypoxic encephalopathy, this syndrome has been named hypoglycemic encephalopathy. However, the underlying pathophysiologies in both entities are markedly distinct ^[4]. We recorded a case of hypoglycemic coma in 62 years old male.

Case Report

A 62 years old male presented with abnormal behavior, sweating, staring at one point since four hours. Patient was a known diabetic and was on glimepiride 1 mg and extended release metformin 500 mg. On clinical examination, the patient was unconscious, unresponsive, breathing spontaneously. Vitals were within normal limits. Glasgow coma score was 3/15. Pupils, mid dilated, reacting to light. Other systems were normal. Immediate capillary glucometer random blood sugar (GRBS) was 11 mg/dl. Immediately resuscitation was started with 200 ml of 25% dextrose (D) intravenously (IV) followed by 5% D (75 ml/hour) infusion. The next four GRBS readings showed declining trend. So, 5% D was replaced with 10% D (50 ml/hour). After six hours, again the patient had severe hypoglycemia (GRBS 32 mg/dl). So, 25% D (20-40 ml/hour) was added along with 10% Dextrose infusion. The patient regained consciousness after four hours of meticulous IV dextrose resuscitation, but remained lethargic and disoriented for the next 18 to 20 hours. Patient became fully oriented

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after 24 hours. The patient remained hemodynamically stable throughout. His laboratory investigations including liver function tests were normal. Oral feeds were started and IV dextrose was tapered down slowly. His routine medications were continued in the hospital. The patient was discharged from the hospital after 72 hours in an otherwise healthy euglycemic condition.

Discussion

Hypoglycemia, a syndrome characterized by adrenergic and neuroglycopenic symptoms induced by an abnormal level of plasma glucose (glycemia < 50 mg/dl), is a common, potentially fatal, yet preventable problem. Drug-induced hypoglycemia remains the commonest cause. Groups of drugs that may potentiate hypoglycemia in non-diabetics are: insulin, sulphonylureas, beta-blockers, salicylates etc [5]. The brain cannot store or synthesize glucose. Glucose depletion to the brain rapidly causes impaired neuronal function, manifested by cognitive impairment or depression of level of consciousness, exhibited as obtundation, stupor or coma. A health care provider can explain which diabetes medications can cause hypoglycemia and explain how and when to take medications. For good diabetes management, people with diabetes should take diabetes medications in the recommended doses at the recommended times. In some cases, health care providers may suggest that patients learn how to adjust medications to match changes in their schedule or routine [6].

Oral hypoglycemic agents involved in drug induced hypoglycemic coma (DIHC) are: gliclazide, glibenclamide, metformin, repaglinide, glimepiride and buformin. Life-threatening hypoglycemia may develop in sulphonylurea-naïve non-diabetics after ingestion of a single therapeutic dose [7]. The duration of hypoglycemia may range from 12 to 24 hours or even longer in cases of large overdose, extended-release preparations as in this case, or long-acting preparations. Prolonged hypoglycemia may also be observed when a relatively low dose of sulphonylurea is taken by patients with advanced age, or with impaired renal or hepatic function as in this case. In general, the hypoglycemic effect of metformin will only be seen in diabetics unless one simultaneously ingests another hypoglycemic agent or if he has co-existing severe hepatic insufficiency [8]. We recorded a case of hypoglycemic coma in 62 years old male.

Lu Z *et al.* [9] found that among the patients, 82 were male and 112 were female (mean age, 66.88±10.62 years). In addition, 72 patients lived in urban areas and 122 lived in rural areas. Occurrence of hypoglycemic coma was correlated with difference between urban and rural residence, glycosylated hemoglobin (HbA1c) level, combined hypertension, and combined neural complications. Self-purchased drugs resulted in significantly lower blood glucose level at the onset of hypoglycemic coma than insulin, secretagogue, or non-secretagogue drugs. Blood glucose level at onset was correlated with season. Patients living in rural areas or with combined macrovascular or microvascular complications had prolonged hospital stay and poor prognosis.

In treating diabetes, continuous monitoring of blood sugar, administration of drugs, and monitoring complications are needed. The pathophysiology of hypoglycemic encephalopathy is poorly understood. Glucose transport into neuronal cells fails when blood concentration is below 2

mM, leading to glucose concentration close to zero in neurons followed by a drop in ATP levels. During the initial phase of hypoglycemia, ketone bodies can be used as an energy substrate for a short period of time, however, afterwards neuronal function is exclusively dependent on glucose supply from the blood. The exact mechanism of neuronal damage in hypoglycemia is unknown. Interestingly, a decrease in ATP levels and isoelectricity on the electroencephalogram coincide. However, clinical symptoms occur before ATP levels reach a critical threshold [10].

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