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Case Report

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The Hypoglycemia Evaluated Mistakenly as Cerebrovascular Disease

Demet Coskun[a, b], Ahmet Mahli[a], Nuray Camgoz[a], Ferda Koksal[a], Belde Tarhan[a], Lale Karabiyik[a]

Abstract

We aimed at emphasizing the significance of monitoring and regulating the blood glucose level of type II diabetic patients by presenting the case of a patient who was initially diagnosed as cerebral ischemia by mistake and later treated and healed via regulating the blood glucose level. A 79-year-old female patient who suffered from type II diabetes mellitus (type II DM) and hypertension was operated on because of left intertrochanteric fracture. On the postoperative 4th day, the overall situation of the patient worsened. She was unconscious, showing no responses to vocal stimulations. Cerebrovascular disease was not eliminated because of clinical findings, and the patient was intubated as mechanical ventilation was applied. The blood glucose level was 12 mg/dl at the time. As 500 ml of dextrose 10% was applied in twenty minutes, the blood glucose level increased to 237 mg/dl and the neurological findings improved. The next day, the patient was extubated. In cases where factors such as stress and starvation are present, the level of blood glucose should be examined frequently for diabetic patients during pre-, per-, and postoperative periods. In our case, our recommendation is to think systematically even from the simplest syndrome to the most complicated one.

Keywords: Hypoglycemia; Cerebrovascular disease; Complication

Introduction

Iatrogenic hypoglycemia causes recurrent morbidity in most cases with type I diabetes mellitus (type I DM) and many cases with type II diabetes mellitus (type II DM), and it is sometimes fatal. Glucose is an obligate metabolic fuel for the brain. Because the brain can neither synthesize glucose nor store more than a few minutes supply as glycogen, it is critically dependent on a continuous supply of glucose from the circulation [1]. Hypoglycemia causes undesirable neuroglycopenic and autonomic symptoms; in daily life, it can cause risks of injury, anxiety, permanent neurological damages and even death [2]. The neuroglycopenic syndrome, which is often due to sulfonylurea-induced hypoglycemia, is frequently overlooked or misinterpreted as cerebral ischemia [3].

We aimed at emphasizing the significance of monitoring and regulating the blood glucose level of type II diabetic patients by presenting the case of a patient who was initially diagnosed as cerebral ischemia by mistake and later treated and healed via regulating the blood glucose level.

Case Report

A 79-year-old female patient with a medical history of hypertension was diagnosed as type II DM. Her blood glucose level was kept under control by use of oral anti-diabetic (sulfonylurea). There was no prior history of cerebrovascular insufficiency. Her family history was noncontributory. She was operated on because of left intertrochanteric fracture. Although the preoperative biochemical values were within the normal range, her blood glucose level was found to be 256 mg/dl. The diabetic protocol was then applied to the patient by ceasing oral anti-diabetics and supplying 1000 ml Dextrose 5% + 8, 4 and 4 IU insulin every 8 hours pre- and perioperatively. Type II DM diet was sustained by oral anti-diabetics postoperatively. Also on the postoperative 3rd day, blood glucose level was 117 mg/dl. On the postoperative 4th day, the overall situation of the patient worsened. She was unconscious, showing no responses to vocal stimulations. There was no history of nausea, vomiting, headache, cranial trauma or seizure. Her blood pressure and heart rate were within the normal range. The cardiovascular examination and ECG findings were normal. Neurological examination

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[a]Department of Anesthesiology and Reanimation, Gazi University Faculty of Medicine, Ankara, Turkey
[b]Corresponding author: Demet Coskun, Gazi University Faculty of Medicine, Department of Anesthesiology and Reanimation, 06500-Ankara, Turkey. Email: dcoskun@gazi.edu.tr

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findings were unconsciousness, lack of response to vocal stimulations, shallow breathing, and minimal extension on the upper extremity with pain stimulus, right facial weakness, and right plantar extension response. The remainder of her cranial nerve examination, and sensorial and cerebellar function tests were within normal limits. Her liver and renal function tests, blood electrolytes, coagulation tests were within the normal range. Cranial CT did not show any acute pathological findings. Cerebrovascular disease was not eliminated because of clinical findings; therefore, mannitol, acetylsalicylic acid and low molecular weight heparin treatment was started. In advancing hours, the patient was taken to intensive care unit (ICU) because of additional breathing problem. She was intubated and mechanical ventilation was applied. The blood glucose level was 12 mg/dl at the time. As 500 ml of dextrose 10% was applied in twenty minutes, the blood glucose level increased to 237 mg/dl and the neurological findings improved. However, as she was still somnolent, mechanical ventilation was continued. Later, in every 4 hours, the blood glucose level was monitored and the need for insulin was adjusted. Next day, her conscious became clear and the patient was extubated and transferred to orthopedics inpatient clinic since her overall situation was good and breathing parameters became normal.

Discussion

Studies on type II DM patients indicate that the appropriate glycemic control is generally ignored. Intensive blood glucose control improves clinical outcomes in diabetes [4, 5], but most treatment regimes increase weight gain and risk of hypoglycemia [6]. The incidence of hypoglycemia seen in type II DM patients is thought to be around 90% [2]. Su [7] found that hypoglycemia mainly occurred due to excessive use of sulfonylurea or insulin (79.83%). Sulfonylureas act by stimulating insulin secretion from the pancreas and augmenting glucose-stimulated insulin secretion. Hypoglycemia is the major risk associated with the use of sulfonylureas, particularly in elderly people [8]. The resultant hypoglycemia can be prolonged and recur for a period of more than 24 hours despite the treatment. Case fatality rates of 4% - 10% are reported and 5% of survivors may have permanent neurological impairment [9]. Diabetes mellitus cause acute or chronic neurological disorders as it contains risk factors for either hypoglycemia, or even cerebral vascular diseases in the long term. The limits of the blood glucose level for neurogenic or neuroglycopenic symptoms are determined as 50 - 55 mg/dl [1].

Hypoglycemia may come out with autonomic and neuroglycopenic symptoms. Glucose counter regulatory mechanisms have generally been found to be intact early in the course of type II DM [10, 11]. However, iatrogenic hypoglycemia becomes progressively more limiting to glycemic control over time [12, 13]. Patients with advanced type II DM, selected for insulin deficiency, were found to have virtually absent glucagon secretory responses to hypoglycemia and their glycemic threshold for autonomic and symptomatic responses were shifted to lower plasma glucose concentration following recent hypoglycemia [11].

As a result of hypoglycemia, due to the stimulation of autonomic neural system, adrenaline and acetylcholine are released. Shivering, stress and anxiety occur due to adrenergic system, also sweating, feeling of hunger and paresthesia occur due to cholinergic system. In elderly people, the classical symptoms and signs of hypoglycemia may not be present and neuroglycopenic features dominate the picture, so the diagnosis can be easily missed [14]. Neuroglycopenic symptoms depend on the inadequate glucose entry to neurons; it may appear with headache, feeling of fever, weakness, nausea, confusion, behavioral changes, emotional labiality, as it may cause fainting, loss of consciousness, even brain damage and death [1, 10]. Type II DM patients are generally elderly; the symptoms of hypoglycemia may appear less frequently due to depression, dementia, earlier strokes and other chronic situations. Particularly, as the use of β-blocker suppresses the autonomic symptoms of hypoglycemia, the neuroglycopenic symptoms may often be mistaken for cerebral ischemia [2]. In order to reduce the risk of hypoglycemia for the patients who use long lasting oral anti-diabetics or insulin, the meals must be taken care of and the patients have to be trained.

Transient hypoglycemic hemiparesis is rare but a dramatic number of cases are frequently misdiagnosed as stroke [15, 16]. A clinical review of 29 documented cases by Forster and Hart [17] indicated that 75% of patients were on insulin and 14% were on oral anti-diabetics for blood glucose control. The mechanism of hypoglycemic hemiplegia has not been well documented. The brain is a highly metabolic organ, using glucose as its exclusive energy source. Under hypoglycemic conditions the energy demand is met by oxidation of cerebral lipids and proteins, and this may result in brain damage. Several theories may explain the pathogenesis of a hypoglycemia associated motor deficit. Structural narrowing of blood vessels that supply a particular portion of the brain could result in even lower levels of glucose. However, angiographic studies have failed to support this theory. A second theory is that hypoglycemia may act on a cerebral vasomotor center to cause vasospasm in the smaller cortical vessels. A third theory attributes local vulnerability to hypoglycemia to differences in cellular metabolism and vascular supply [16].

Conclusion

In cases where factors such as stress and starvation are present, level of blood glucose should be examined frequently for diabetic patients during pre-, per-, and postoperative pe-

riods. Improved glycemic control may limit morbidity, mortality and use of ICU and hospital resources. Metabolic disorders should be excluded in all patients with a diagnosis of suspected stroke, especially if the presentation is associated with the state of confusion or focal seizures. The conventional symptoms and signs of metabolic disorders may be minimal or absent. In our case, our recommendation is to think systematically even from the simplest syndrome to the most complicated one; the possible reasons such as metabolic encephalopathy, cerebrovascular vasospasm, ischemia, and selective neuronal defects should not be ignored. Besides, checking the blood glucose level in the first place should be an obligatory act before going into any further complicated investigations.

Conflict of Interests

There is no conflict of interests related to this manuscript.

References

1. Cryer PE, Davis SN, Shamoon H. Hypoglycemia in diabetes. Diabetes Care 2003;26(6):1902-1912.
2. Murata GH, Duckworth WC, Hoffman RM, Wendel CS, Mohler MJ, Shah JH. Hypoglycemia in type 2 diabetes: a critical review. Biomed Pharmacother 2004;58(10):551-559.
3. Holstein A, Egberts EH. [CNS symptoms caused by hypoglycemia: frequent misdiagnosis: “stroke”. Pitfall of the neuroglycopenia syndrome]. MMW Fortschr Med 1999;141(22):42-44.
4. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group. N Engl J Med 1993;329(14):977-986.
5. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). UK Prospective Diabetes Study (UKPDS) Group. Lancet 1998;352(9131):854-865.
6. Wright AD, Cull CA, Macleod KM, Holman RR. Hypoglycemia in Type 2 diabetic patients randomized to and maintained on monotherapy with diet, sulfonylurea, metformin, or insulin for 6 years from diagnosis: UKPDS73. J Diabetes Complications 2006;20(6):395-401.
7. Su CC. Etiologies of acute hypoglycemia in a Taiwanese hospital emergency department. J Emerg Med 2006;30(3):259-261.
8. Graal MB, Wolffenbuttel BH. The use of sulphonylureas in the elderly. Drugs Aging 1999;15(6):471-481.
9. Salas M, Caro JJ. Are hypoglycaemia and other adverse effects similar among sulphonylureas? Adverse Drug React Toxicol Rev 2002;21(4):205-217.
10. Cryer PE. Hypoglycaemia: the limiting factor in the glycaemic management of Type I and Type II diabetes. Diabetologia 2002;45(7):937-948.
11. Segel SA, Paramore DS, Cryer PE. Hypoglycemia-associated autonomic failure in advanced type 2 diabetes. Diabetes 2002;51(3):724-733.
12. U.K. prospective diabetes study 16. Overview of 6 years’ therapy of type II diabetes: a progressive disease. U.K. Prospective Diabetes Study Group. Diabetes 1995;44(11):1249-1258.
13. United Kingdom Prospective Diabetes Study 24: a 6-year, randomized, controlled trial comparing sulfonylurea, insulin, and metformin therapy in patients with newly diagnosed type 2 diabetes that could not be controlled with diet therapy. United Kingdom Prospective Diabetes Study Group. Ann Intern Med 1998;128(3):165-175.
14. McAulay V, Deary IJ, Frier BM. Symptoms of hypoglycaemia in people with diabetes. Diabet Med 2001;18(9):690-705.
15. Carter F, Taylor C. Transient hypoglycemic hemiparesis. J Natl Med Assoc 2002;94(11):999-1001.
16. Wattoo MA, Liu HH. Alternating transient dense hemiplegia due to episodes of hypoglycemia. West J Med 1999;170(3):170-171.
17. Foster JW, Hart RG. Hypoglycemic hemiplegia: two cases and a clinical review. Stroke 1987;18(5):944-946.