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Reactive Hypoglycaemia: Diagnosis and Management with Flash Glucose Monitoring Sensor, Extended Glucose Tolerance Tests, and Voglibose

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Abstract

Background: The present case report describes a 39-year-old man who experienced frequent hypoglycemia postprandially. On evaluation, he was diagnosed with reactive hypoglycemia (RH). *Case Report*: The importance of extended glucose tolerance test (e-GTT) and continuous glucose monitoring using flash glucose monitoring sensor (FGMS) in diagnosing RH and subsequent monitoring of glycemic variability is demonstrated. *Conclusion*: The role of e-GTT, FGMS and α-glucosidase inhibitor (voglibose) in managing RH for the last 3 years without any further episode of biochemical and clinical symptoms of hypoglycemia is highlighted.

Keywords: Diabetes, Dizziness, Glucose, Hypoglycemia, Monitoring.

Introduction

Reactive hypoglycemia (RH) is a rare pathophysiologic condition in which hypoglycemic symptoms occur postprandially within 120-300 minutes (2-5 hrs) after taking foods [1]. RH is clinically characterized in three different forms such as idiopathic reactive hypoglycemia (at 3 hrs), alimentary (within 2 hrs), and late RH (at 4-5 hrs). The earliest change seen in the onset of type 2 diabetes is the loss of first-phase insulin release, which emerges with fasting glucose levels of about 110 mg/dL. When there is a decrease in the first-phase insulin response initially, blood glucose begins to rise following the meal, which leads to late but enhances secretion of the second-phase insulin secretion; causing late reactive hypoglycemia to occur [2]. The level of glucose that defines hypoglycemia is debatable but it ranges between 40 mg/dL (2.2 mmol/L) and 72 mg/dl (4.0 mmol/L) [3,4].

We present a patient with possible reactive hypoglycemia; an extended glucose tolerance test (e-GTT) was done to measure the insulin and glucose level to detect the hypoglycemia biochemically; in addition, a flash glucose monitoring sensor (FGMS) marketed by Abbott freestyle Libre Pro system for continuous glucose monitoring (CGM) was used to study the glycemic patterns.

Case Report

A 39 years old teacher presented to us with the repeated episode of hunger, abdominal pain, tremors, dizziness, impending to fall, and anxiety of 6 months duration. This episode usually occurred two to three times after eating. He had documented low blood sugar varying from 56-70 mg/dL on multiple occasions. The patient denied any history of type 2 diabetes or gastric bypass surgery and had no immediate family members with diabetes. He was referred here for evaluation of postprandial hypoglycemia. This disorder is occasionally encountered in patients with other medical illnesses like hyperthyroidism, impaired glucose tolerance, early-stage diabetes, and rarely insulinoma.

The physical and neurological finding was unremarkable. A detailed hormonal and electrolytes were checked which was normal. Laboratory investigation revealed: fasting blood glucose and HbA1c were 92 mg/dL, 6.2% respectively. Renal function test, liver function test along with adrenal function, and thyroid functions were also in normal range. He was planned for 72-hour fasting; during that period his lowest blood sugar was recorded as 72 mg/dL without obvious features sympathoadrenal or neuroglycopenic symptoms. An FGMS was fixed to his arm to measure the diurnal variation of blood sugar; 72 hrs glucose reading **[Fig.1a]**, the interstitial glucose level remained within a normal level (80-126). This ruled out endogenous hyperinsulinism. Hence, he was not further evaluated for insulinoma. He was subsequently planned for e-GTT with 75 gm of glucose. An FGMS was connected to record the variation of glucose at a particular point of time shown in **[Fig.1b]**. His fasting blood sugar was 92 gm%, peaked at 1hr to 218 mg/dL and lowest at 3 hr to 40 mg/dL with development of symptoms of tremors, and dizziness. Likewise, his fasting insulin was 1279 mU/mL (2.6-24.9) and rose to 1367 mU/mL at 3 hrs then remain elevated with a small gradual fall within 2 hrs. The details of blood sugars and serum insulin level are depicted in **Fig.2**. At the baseline serum, insulin was high in addition to fasting C-peptide indicating insulin resistance, followed by insulin peaked at 3 hrs after glucose ingestion with gradual fall thereafter. The results showed that this patient had diabetes, which was induced by high insulin and C-peptide 8.15 ng/mL (1.10-4.40) levels at baseline and highest insulin concentration at 2 hrs suggestive of insulin resistance, and consequently, he experienced hypoglycemia due to the delayed sustained hypersecretion of insulin.

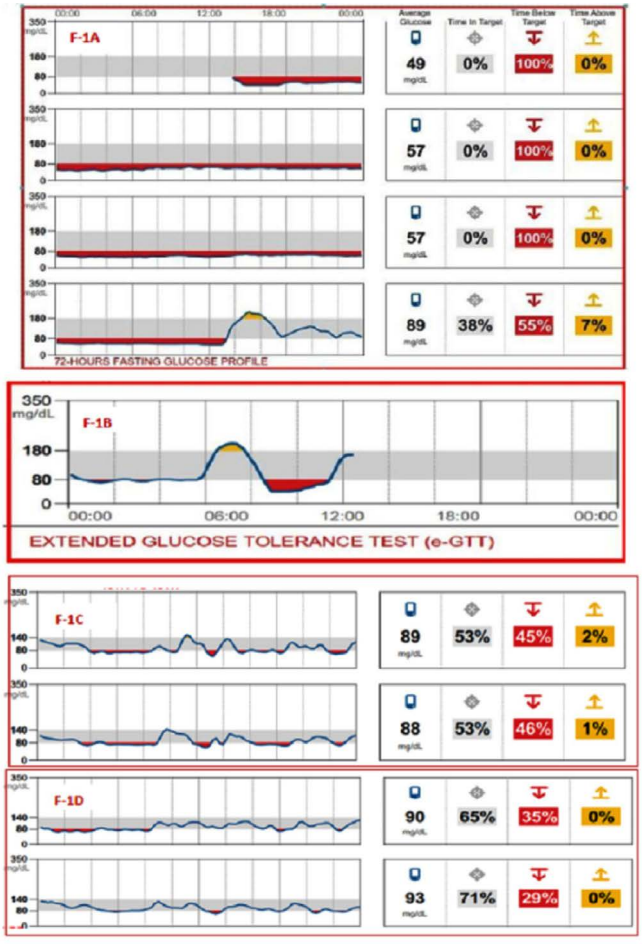

Fig.1: (a) *72 hours glucose profile;* **(b)** *Extended glucose tolerance test;* **(c)** *More glucose variability profile without voglibose;* **(d)** *Stable glucose profile with voglibose twice daily.*

He was started with voglibose 0.3 mg (α-glucosidase inhibitors) twice daily with breakfast and dinner following which he was free from symptoms of hypoglycemia. He had first followed up at 6 months then subsequently once in a year for the last three and half years. During the follow-up, it was decided to fix the glucometer

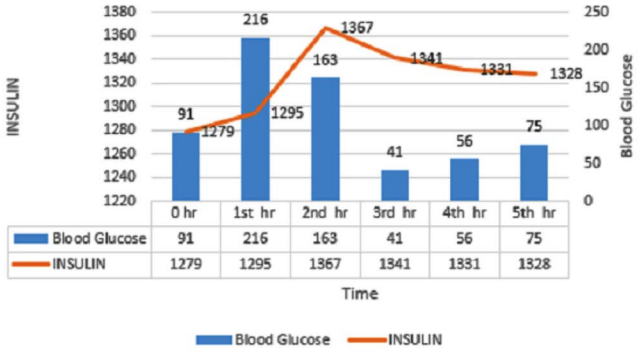

Fig.2: *The details of blood sugars and serum insulin in extended glucose tolerance test.*

to study the glycemic variability with and without voglibose. The CGMS recording revealed a more stable glycemic profile with voglibose twice daily in contrast to without it **[Fig.1c,d]**.

Discussion

We reported a patient with recurrent postprandial hypoglycemia that occurred mostly after breakfast, and after post-lunch. No history of diabetes in past, he has also ruled out insulinoma with the appropriate test, so he was diagnosed with reactive hypoglycemia. The most widely postulated cause for RH is due to exaggerated insulin release and insulin resistance [5]. In clinical practice, e-GTT and continuous glucose monitoring systems using flash glucose monitoring sensors are two diagnostic tools conclusive in successful diagnosis and treatment.

e-GTT is widely used for the diagnosis of reactive hypoglycemia and insulin resistance [3,6], our case showed a peaked blood sugar at one hour and peaked insulin at two hours and lowest documented blood sugar of 41 mg% at three hours of e-GTT, conclusive of RH. Inappropriate secretion of insulin following a meal is the principal causative factor for RH. Several reports are showing that α-glucosidase inhibitors reduced the early onset insulin excursion [7] during OGTT and are effective in treating RH [8].

In our case, as we have shown the role of FGMS in picking up hypoglycemia, its role in monitoring the glycemic variability on treatment with voglibose.

Conclusion

The present case report describes a 39-year-old man who experienced frequent hypoglycemia due to RH. The role of e-GTT and CGMS in diagnosing and subsequent monitoring of glycemic variability on voglibose has been highlighted.

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References

1. ltuntaş Y. Postprandial reactive hypoglycemia. Sisli Etfal Hastan Tip Bul. 2019;53(3):215-220.
2. Mitrakou A, Kelley D, Mokan M, Veneman T, Pangburn T, Reilly J, *et al*. Role of reduced suppression of glucose production and diminished early insulin release in impaired glucose tolerance. N Engl J Med. 1992;326(1):22-29.
3. Stuart K, Field A, Raju J, Ramachandran S. Postprandial reactive hypoglycaemia: varying presentation patterns on extended glucose tolerance tests and possible therapeutic approaches. Case Rep Med. 2013;273957.
4. Ratner RE. Hypoglycemia: New definitions and regulatory implications. Diabetes Technol Ther. 2018;20(S2):S250-S253.
5. McCool C, Luqman W, Schmitt T, Raymundo R, Nolan S, Stephan T, *et al*. Transient insulin increase in reactive hypoglycemia in obese and non-obese subjects. Int J Obes. 1977;1(2):179-183.
6. Koyama H, Ohguchi H, Yagi T, Imaeda K. Nocturnal reactive hypoglycaemia well treated subjectively and objectively with voglibose. BMJ Case Rep. 2017;2017:bcr2017220295.
7. Ozgen AG, Hamulu F, Bayraktar F, Cetínkalp S, Yilmaz C, Túzún M, *et al*. Long-term treatment with acarbose for the treatment of reactive hypoglycemia. Eat Weight Disord. 1998;3(3):136-140.
8. Hirtz D, Berg A, Bettis D, Camfield C, Camfield P, Crumrine P, *et al*. Practice parameter: treatment of the child with a first unprovoked seizure: Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology. 2003;60(2): 166-175.

