CASE REPORT

JIACM 2019; 20(2): 134-36

Type 2 Diabetes Mellitus Presenting as Post-Prandial Hypoglycaemia

Ayush Bansal, Jai Bhagwan***

Abstract

Background: Post-prandial hypoglycaemia is an uncommon presenting feature of diabetes mellitus and usually follows a heavy refined carbohydrate meal. More commonly, the adrenergic symptoms of post-prandial hypoglycaemia are attributed to other neuropsychiatric illnesses. The main pathophysiology behind post-prandial hypoglycaemia in diabetes mellitus is the augmented second phase of insulin secretion and impaired glucagon release. This leads to hypoglycaemia, typically after 3 - 4 hours of meals.

Case: Our patient presented for the first time with classical features of hypoglycaemia and was later diagnosed to have overt diabetes mellitus.

Conclusion: It is important to recognise this uncommon presentation of diabetes mellitus, so that early cases are not missed. Thus, documentation of hypoglycaemia is of paramount importance to establish the diagnosis of post-prandial hypoglycaemia.

Key words: Post-prandial hypoglycaemia, insulin, blood glucose, oral glucose tolerance test.

Introduction

The usual presentation of type 2 diabetes mellitus is either asymptomatic individuals detected during routine investigation, or presentation with classical symptoms of polydipsia, polyuria and polyphagia. Few patients may present with recent history of weight loss or recurrent infections. An uncommon presentation may be post-prandial hypoglycaemia. Post-prandial hypoglycaemia, also termed reactive hypoglycaemia, typically manifests after 3 - 4 hours of a refined carbohydrate rich meal.

Case report

A 45-year-old male came to the casualty at 11 pm with symptoms of palpitations, sweating and dizziness. He was fully conscious and well oriented. On examination, he was afebrile with a pulse of 114/min regular, blood pressure 130/80 mmHg, and BMI - 33.91 Kg/m^2. Chest, CVS and CNS were normal. Random blood glucose by glucometer was 42 mg%. No previous history of hypertension, diabetes mellitus or heart disease was available. However, his father was a diabetic. Patient gave history of 2 - 3 similar episodes of palpitations in the last one month which resolved after taking some sweet drinks.

A tentative diagnosis of hypoglycaemia was kept and the patient was given intravenous dextrose and the symptoms subsided. A diagnosis of hypoglycaemia requires the presence of Whipple's triad, i.e., symptoms of hypoglycaemia, low blood glucose level and resolution of symptoms after blood glucose normalises[1]. This patient had all the three parameters and so fitted with the diagnosis of hypoglycaemia.

Investigations

Further investigations of the patient were done which revealed the following results: Hb - 12.5 gm%, TLC - 7,200/mm^3, ESR - 20 mm 1st hour, HbA1c - 7.0%, FBS - 114 mg/dl, PPBS - 162 mg/dl, total cholesterol - 230 mg/dl, HDL cholesterol - 30 mg/dl, LDL cholesterol - 140 mg/dl, VLDL cholesterol - 60 mg/dl, and triglycerides - 450 mg/dl. Liver and kidney function tests were normal. Serum insulin and C-peptide values are summarised in Table I.

Table I: Normal and test values of serum insulin and C-peptide.

	Parameters	Test	Normal values
Serum insulin	Fasting	24	< 25 mIU/L
	Post-prandial	178	16 - 166 mIU/L
C- peptide		2.9	0.51 - 2.72 ng/ml

The patient was subjected to Oral Glucose Tolerance Test (OGTT). It was found that the blood glucose levels increased from 104 mg% (fasting) to a maximum of 182 mg% (90 min post-prandial). However, a reduction of 48% was observed 240 min after having meals, i.e., the blood glucose levels dropped to 54 mg%. Blood glucose levels at different time intervals are shown in Fig. 1.

**Assistant Professor, Department of Medicine, Hamdard Institute of Medical Sciences and Research, Hamdard Nagar, Delhi - 110 062.*
***Consultant Physician and Director, Kanshi Ram Medical Services, Gurugram - 122 001, Haryana.*
Corresponding Author: Dr Ayush Bansal, Department of Medicine, Hamdard Institute of Medical Sciences and Research, Hamdard Nagar, Delhi - 110 062. Phone: 9619583909, E-mail: drayushbansal@gmail.com.

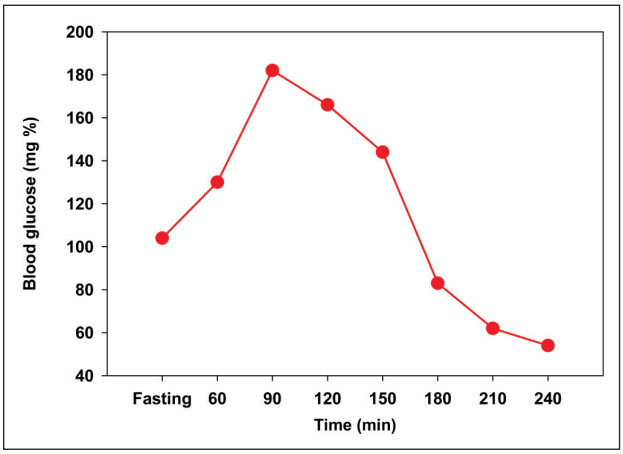

Fig. 1: *Blood glucose levels during oral glucose tolerance test.*

This further narrowed down the diagnosis to hyperinsulinaemic hypoglycaemia, with obesity and diabetes mellitus accompanied, with dyslipidaemia. A differential diagnosis of insulinoma was kept, but excluded because insulinoma leads to fasting hypoglycaemia or at the most, both fasting and post-prandial hypoglycaemia whereas, our patient had only post-prandial hypoglycaemia. Moreover, insulinoma is rare[2] and was ruled-out by a CECT abdomen. Exclusive post-prandial hypoglycaemia is typically seen in post-gastric bypass surgery, early type II diabetes mellitus, or rarely in hereditary fructose intolerance or congenital disorders of glycosylation[1].

Discussion

Reactive hypoglycaemia can occur in individuals either with or without diabetes, and is thought to be more common in overweight individuals or those with a history of gastric bypass surgery. Symptoms of reactive hypoglycaemia can include anxiety, tremors, dizziness, fatigue, palpitations, anorexia, sweating or weakness. These symptoms are however non-specific and may be a consequence of post-prandial syndrome, in which blood glucose levels remain normal.

The pathophysiology of hypoglycaemia is linked to regulation of insulin release. In normal individuals, the rapid increase in blood glucose after food is immediately neutralised by rapid first phase of insulin release. If the blood glucose still remains high, a short second phase of insulin release neutralises it[3]. However, in pre-diabetics or mildly diabetic subjects, the first phase of insulin release is slowed down, giving rise to persistent hyperglycaemia after food[4]. This in turn, stimulates a more robust and prolonged second phase of insulin release which is responsible for post-prandial hypoglycaemia in early diabetic subjects, usually after 3 - 4 hours of food intake.

Similar result was found in a study conducted by Luyckx and Lefebvre[5] on forty-seven patients, in which reactive hypoglycaemia was explained by an exaggerated insulin response in obese individuals with impaired glucose tolerance.

Other mechanisms proposed for reactive hypoglycaemia include renal glycosuria, impaired glucagon secretion and increased insulin sensitivity. Renal glycosuria and increased insulin sensitivity (secondary to rapid weight loss in obese individuals) are not hyperinsulinaemic states[6].

In 1986, the Third International Symposium on Hypoglycaemia in Rome, published a consensus statement indicating that OGTT overestimates the incidence of reactive hypoglycaemia and thus, it should not be used[7]. Moreover, it is evident that using a 75 gm glucose load is a much stronger stimulus to insulin secretion and, therefore, is much more likely to induce reactive hypoglycaemia than any other routine meal. Still, OGTT is the most widely employed tool in this regard. Therefore, ambulatory blood glucose monitoring when the patient is on a routine diet seems to be far more superior for the diagnosis of reactive hypoglycaemia. But, no consensus guidelines have been laid in this context.

Since reactive hypoglycaemia is a feature of early diabetes mellitus, the treatment of choice has to be lifestyle modifications including regular exercise and dietary control. It is the inertia on the part of medical practitioners in laying stress on lifestyle modifications and on the part of patient, that these modifications are under-utilised. Further, a few subset of patients may respond to lifestyle interventions alone and may not need any other treatment. Alpha-glucosidase inhibitors such as acarbose and voglibose may be used which impair carbohydrate absorption and therefore blunt the insulin response to glucose[8]. Biguanides like metformin, also have been shown to reduce reactive hypoglycaemia[9]. Moreover, somatostatin regulates the secretion of gut hormones and lowers insulin levels in both fasting and post-prandial conditions. Further, a dramatic response to a single dose of octreotide was reported in a case of severe post prandial hypoglycaemia[10]. However, the use of somatostatin analogues should be reserved for exceptional cases only, given its cost and its side-effects.

Treatment

Undoubtedly, lifestyle modifications form the cornerstone of treatment. Voglibose with metformin was prescribed alongwith the heaviest meal of the day. Rosuvastatin 10 mg at bedtime was added. The patient improved well and there was no recurrence of hypoglycaemia.

Lifestyle modifications should include:

- Regular exercise, which increases glucose uptake.
- Small frequent low carbohydrate meals which are not more than 3 hours apart.
- Inclusion of fish, poultry, dairy products and fibre-rich food in diet such as whole grains, oats, fruits and vegetables.
- Avoid sugary food and drinks such as doughnuts, frozen desserts, flavoured drinks and soft drinks.

Conclusion

Diabetic patients, especially pre-diabetics or early diabetics, can present for the first time with post-prandial hypoglycaemia. It is important to recognise this uncommon presentation as hypoglycaemia itself is a life-threatening condition. Without laying emphasis on lifestyle interventions, all other efforts, including all anti-diabetic drugs, are unlikely to get desired results.

References

1. Jameson JL, Fauci, Kasper DL *et al*. Hypoglycaemia. *Harrison's Principles of Internal Medicine*. 2. 20 ed: Mc Graw Hill, 2018; p. 2883.
2. Wasada T, Katsumori K, Saeki A *et al*. Lack of C-peptide suppression by exogenous hyperinsulinaemia in subjects with symptoms suggesting reactive hypoglycaemia. *Endocrine J* 1996; 43 (6): 639-44.
3. McCulloch DK, Bingley PJ, Colman PG *et al*. Comparison of bolus and infusion protocols for determining acute insulin response to intravenous glucose in normal humans. *Diabetes Care* 1993; 16 (6): 911-5.
4. Brunzell JD, Robertson RP, Lerner RL *et al*. Relationships between fasting plasma glucose levels and insulin secretion during intravenous glucose tolerance tests. *J Clinical Endocrino Metab* 1976; 42 (2): 222-9.
5. Luyckx A, Lefebvre P. Plasma insulin in reactive hypoglycaemia. *Diabetes* 1971; 20 (6): 435-42.
6. Brun JF, Fédou C, Mercier J. Post-prandial reactive hypoglycaemia. *Diab Metab* 2000; 26 (5): 337-52.
7. Lefèbvre PJ, Andreani D, Marks V *et al*. Statement on post-prandial hypoglycaemia. *Diabetes Care* 1988; 11 (5): 439-40.
8. Lefèbvre PJ, Standl E. New Aspects in Diabetes: Treatment Strategies with Alpha-glucosidase Inhibitors: 3rd International Symposium on Acarbose: de Gruyter; 1992.
9. Hofeldt FD. Reactive hypoglycsemia. *Metabolism* 1975; 24 (10): 1193-208.
10. Pears J, Jung R, Browning M *et al*. Reactive hypoglycaemia in association with disordered islet function and abnormal hepatic glucose 6 phosphatase activity: response to diazoxide. *Diabetic Medicine* 1991; 8 (3): 268-71.