Reactive (post prandial) hypoglycaemia in a patient with Type 2 Diabetes Mellitus An interesting paradox.

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Abstract:
Reactive hypoglycaemia is a disorder that is seldom diagnosed and the symptoms of such patients are often passed on as functional. Further the diagnosis of reactive hypoglycaemia should prompt the search of the underlying cause for the same. Here we report the rare case of a patient with early latent Type 2 Diabetes Mellitus who presented with reactive hypoglycaemia.

Keyword: Reactive post prandial hypoglycaemia diabetes mellitus

Reactive or post prandial hypoglycaemia is a diagnosis that is rarely made as the symptoms of such patients are often passed on as functional. We report an interesting case of a patient with Type 2 Diabetes Mellitus, who was found to have reactive hypoglycaemia.

MEDICAL HISTORY:
A 50 year old male with no known medical co morbidity presented to us with a 2 year history of increasing episodes of sweating, palpitation and anxiety typically occurring 2 to 3 hours by delayed meals and exertion. It was associated with a feeling of light headedness and headache. The same symptoms were relieved by intake of food. There was no diurnal pattern to the symptoms. There were no other symptoms such as loss of consciousness, chest pain or dyspnoea. He had never been documented to have hypoglycaemia. On examination he was found to have a BMI of 30.2 kg/m². He was normotensive with a regular pulse rate of 84 beats/min. Other general and systemic examination was within normal limits. The differential diagnosis considered for the symptom complex of light headedness, sweating palpitation and anxiety were as follows - Hypoglycaemia probably secondary to an insulinoma, phaeochromocytoma, cardiac arrhythmias, hyperthyroidism and panic attacks. Hence we investigated the patient as following.

A contrast CT of the abdomen did not reveal any evidence of insulinoma. He underwent an endoscopic USG which again did not show any mass lesion in the pancreatic parenchyma. Fasting C peptide and fasting insulin
assays were also normal in this patient. As mentioned above investigations for hyperthyroidism and phaeochromocytoma were also negative. He also underwent a HOLTER and a Treadmill Test which failed to reveal any cardiac cause for his symptoms. A psychiatric evaluation ruled out any features of psychoneuroses. At this point in time, as his symptoms best corroborated with that of hypoglycaemia and since his symptoms predominantly occurred after intake of meals, a 75gm, 5 hour long extended Oral Glucose Tolerance Test (OGTT) was administered to reproduce the symptoms. Hourly monitoring of serum Insulin levels and blood glucose was done. The results of the OGTT were as follows. Blood glucose values given are laboratory values.

### 5 hour extended OGTT
At 4 hours he was noticed to have symptoms of perspiration, palpitation and feeling of light headedness – same symptoms that had made him seek medical attention. The blood glucose done at the time, showed a value of 48mg/dl. We were able to reproduce the symptoms that the patient had and corroborate it with low blood glucose levels. His symptoms resolved at 5 hours spontaneously without any

| Haemoglobin | 18.5 g/dl | ESR/CRP | 2 / <3.4mg/dl |
| Total WBC count | 7500 cells/cu.mm | Total protein/albumin | 7.1 / 4.5 g% |

| Platelet count | 2.34 lac cells/cu.mm | SGOT / SGPT / ALP | 16 / 13 / 80 U/L |
| Sodium/Potassium | 136/4.4 mg/dl | AC / PC | 120 / 155 mg% |
| Calcium / Phosphorus | 9.4/3.7 mg/dl | Lipid profile (total cholesterol/triglyceride/HDL / LDL) | 125/ 171/ 36/ 70 mg% |
| TSH | 1.62 μU/l (0.3 - 4.5) | Fasting C peptide | 4.2 ng/ml (1.1 -5.0) |
| Creatinine | 1.2 mg/dl | Fasting Insulin assay | 21.0μU/ml (up to 30) |
| Total to Direct Bilirubin | 0.4 / 0.1mg % | 24 hour urine metanephrines | 150/594 μg/24 hrs |

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<th>The results of the OGTT were as follows. Blood glucose values given are laboratory values.</th>
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<td><strong>Fasting</strong></td>
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<td>Blood glucose (mg %)</td>
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<td>Insulin assay (μU/L)</td>
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medical intervention. Thus we were able to demonstrate the Whipple’s triad.(1) – 1. Symptoms consistent with hypoglycaemia 2. A low plasma glucose concentration measured with a precise method (not a glucose monitor) 3. Relief of those symptoms after the plasma glucose level is raised.

DISCUSSION

Reactive or post prandial hypoglycaemia is a term used to describe the timing of hypoglycaemia that occurs within 4 to 5 hours after a meal. It is not a diagnosis per se. The presence of reactive hypoglycaemia requires an evaluation to rule out the underlying cause of the same. The causes of reactive hypoglycaemia are 1. Alimentary hypoglycaemia – e.g. occurring after gastrectomy, vagotomy and pyloroplasty. (2, 3) 2. Factitious hypoglycaemia due to self administration of insulin or oral hypoglycaemic agents. 3. Insulin autoimmune hypoglycaemia – due to the spontaneous generation of insulin antibodies that bind to the insulin released after a meal which then dissociates in an unregulated fashion causing late postprandial hyperinsulinemia and hypoglycaemia. 4. Non insulinoma pancreatogenous hypoglycaemia syndrome (NIPHS) - characterized by endogenous hyperinsulinemic hypoglycaemia that is not caused by an insulinoma rather by nesidioblastosis with islet cell hypertrophy. (4) Rarely reactive hypoglycaemia has been reported in patients with insulinoma, (5) children with hereditary fructose intolerance, (6) occasionally in Type 1 diabetic patients who have undergone whole organ pancreatic transplantation, (7) or by the ingestion of large amounts (three drinks or more) of ethanol and simple carbohydrate (gin and tonic). (8) Interestingly reactive hypoglycaemia has also been reported previously from patients with insulin resistance or very mild Type 2 Diabetes Mellitus. (9) The proposed mechanism is a supranormal insulin response to a supranormal rise in plasma glucose after glucose ingestion, followed by an excessively rapid response to the insulin. In the patient that we described, we were able to rule out insulinoma with fair certainty. He had not had any prior surgery there was no evidence of psychoneuroses to suggest a factitious cause for hypoglycaemia. Insulin autoimmune and NIPHS were considered unlikely as the patient was found to have glucose intolerance, which would have been unlikely in a patient with the above diagnosis. This patient also had early mild diabetes as described by Faludi et al (9) in that he was diagnosed to be diabetic only by an OGTT and not by fasting and post prandial blood glucose measurements. Dietary modification has been advised for patients with reactive hypoglycaemia - consuming frequent small meals or snacks, consuming foods high in fibre, avoiding foods high in sugar although the evidence for the same is doubtful. (10) Although evidence is limited, some advocate a trial of alpha-glucosidase inhibitors to delay carbohydrate absorption and thereby reduce the insulin response to a meal. (11) Our patient was advised small frequent meals (every 2-3 hours), with food with a low glycaemic index. He was also started on Voglibose (an alpha glucosidase inhibitor) which would help control his blood glucose and relieve the symptoms of post prandial hypoglycaemia.

CONCLUSION:

Reactive hypoglycaemia is an often overlooked disorder, with symptoms being branded as functional. However the diagnosis of reactive hypoglycaemia should prompt a search for the underlying cause. Rarely reactive (post prandial) hypoglycaemia
may be the presenting symptom of a patient with early latent diabetes.

References


