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Case Report

Factitious Hypoglycemia and Insulin Edema due to Surreptitious Insulin Use in a Patient with Type 1 Diabetes Mellitus

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Abstract

Background: Insulin edema is an uncommon but well-known complication of insulin therapy, mostly appreciated in patients soon after commencement or intensification of insulin therapy.

Objectives: To report a case insulin edema in Type 1 Diabetes due to surreptitious excess self-insulin use triggered by poverty driven psychosocial stressors.

Case Presentation: A 14-year girl with T1DM since age 8 years, with chronic poor glycemic control (Insulin 48 units/day basal-bolus; HbA1c 12.5%, despite 100% free medical care, medicines, diagnostics and counselling), presented with multiple episodes of unexplained hypoglycemia with documented low blood sugar readings (30-60 mg/dl) and "apparently" grossly decreased recent insulin requirements (4 units/day) This was associated with rapid weight gain, swelling of her face, limbs and abdomen as well as shortness of breath.

Work up for acute/sub-acute renal, hepatic, cardiac, pulmonary or infectious disorders was negative. Fluid and salt restriction, close clinical monitoring and compassionate counselling resulted in prompt amelioration of hypoglycemia, resolution of edema, weight gain and shortness of breath. In-depth clinical assessment suggested the diagnosis of surreptitious excess insulin use, triggered by psychosocial and economic stressors, leading to factitious hypoglycemia and consequent insulin edema causing fluid retention and dyspnea.

Discussion: To our knowledge, this is the first documented case of insulin edema in a patient previously diagnosed with T1DM due to the surreptitious use of insulin by the patient resulting from psychosocial stressors, similar to those in other published encounters.

Conclusion: Insulin edema resulted from transient surreptitious (non-therapeutic) excess self-insulin use, triggered by psychosocial stressors.

Keywords: Insulin edema; Hypoglycemia; Surreptitious insulin use; Psychosocial stressors

Introduction

Generalized insulin edema is an uncommon complication of insulin therapy [1]. It is mostly appreciated in patients with newly diagnosed or poorly controlled diabetes mellitus, shortly after starting intensive insulin therapy. In patients with type 1 Diabetes Mellitus, it is largely reported in the setting of hyperglycemic crises like diabetic ketoacidosis [2]. Rare instances of fluid overload leading to cardiac or renal failure have been reported [1].

We report, perhaps for the first time, a case of insulin edema due to surreptitious self-insulin administration by the patient.

Case Presentation

A 14-year-old adolescent girl, diagnosed with Type 1 Diabetes Mellitus (T1DM) at the age of 6 and primary hypothyroidism at the age of 9, is treated for her endocrine disorders at the free local clinic. Her physical examination prior to presentation was notable for the following parameters:

BMI- 29.5kg/m^2 (>3 SD above median for her age) and pubertal stage of Tanner stage 2.

Relevant baseline biochemical parameters were as follows:

Glycated hemoglobin (HbA1c) of 12.5mg/dl with prior values ranging between 9 and 13 mg/dl, Serum Creatinine of 0.59mg/dl with normal Albumin- Creatinine ratio and TSH of 0.02μIU/ml.

T1DM was managed using a self-administered basal bolus insulin regime comprising 12 units nightly of a long acting Insulin, 12 units of a rapid acting Insulin three times daily with meals and 250mg of Metformin twice daily. Primary hypothyroidism was treated with Levothyroxine 175microg daily, which was subsequently decreased to 150microg daily at her prior visit, based on her above-mentioned TSH level. The patient's self-measured blood glucose readings four times daily ranged between 150mg/dl and 350mg/dl. She had no history of

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diabetic ketoacidosis and no history of hypoglycemic episodes.

The patient presented with multiple episodes of unexplained recurrent hypoglycemia in the preceding week. Fingerstick glucose readings between 30mg/dl and 60mg/dl were recorded on her glucometer. Of note, glucose levels were highest in the fasting state immediately upon waking following which they decreased to below 70mg/dl. Hypoglycemic episodes occurred throughout the day, with no relation to meal timings and with no triggers identified. The patient had unchanged oral intake and physical activity levels. Based on the recorded low blood glucose values, the treating team recommended appropriate reduction of insulin dose from a consolidated 48 units daily to as little as 4 units daily. However, in the following days, the child's condition worsened. She experienced progressive rapid weight gain secondary to fluid retention leading to extremity edema and ascites and endorsed dyspnea on exertion.

Evaluation for possible etiologies through blood and urine investigations as well as imaging were unrevealing. With the resulting lack of a definitive diagnosis, the child was started on symptomatic management with fluid and salt restriction and placed under observation. Over the next two days, she experienced no further hypoglycemic episodes and displayed rapid clinical improvement with complete resolution of fluid retention and dyspnea. The dramatic disappearance of hypoglycemic episodes with increasing insulin requirements during supervision ruled out most organic diagnoses being considered. Acutely lowered Serum Fructosamine [Glycated Serum Protein] from 487mg/dl to 319mg/dl with unchanged high levels of HbA1c suggested a recent lowering of blood glucose levels. After thorough review of medical data and differential diagnoses, the possibility of surreptitious insulin use leading to factitious hypoglycemia and insulin edema leading to fluid retention were entertained. This would also explain the high glucose levels noted in the fasting state in the mornings due to lack of insulin use overnight. An extensive interview of the child and her parent was carried out by the child psychologist to evaluate her psychosocial history and thereby reveal precipitating triggers and underlying causes for surreptitious insulin use. Recent triggers included impending exams and change of school, which compounded long standing underlying sources of stress due to low socioeconomic status, lack of attention from a single working parent and stigma faced due to her illnesses. In addition, the child was aware that she would be given sweet treats when she experienced hypoglycemic episodes, motivating her to inject larger than prescribed doses of insulin.

The child and mother were provided intensive compassionate counselling, supervised medical care and 24-hour telemedicine support following the interview. The patient did not experience any further episodes of hypoglycemia. Her finger stick glucose readings returned to her prior baseline. The child and her mother were referred to a clinical psychologist for long term follow-up.

Discussion

Factitious disorders are characterized by falsification of physical or psychological signs or symptoms, or induction of injury or disease, associated with identified deception in the absence of obvious external rewards [3-5]. Individuals surreptitiously induce symptoms to portray physical and mental illness to assume the patient role [5]. It needs to be differentiated from malingering, involving the intentional falsification or induction of signs or symptoms to achieve personal gain, namely secondary gain like free food or shelter, absence from work or to avoid legal difficulties [6].

Factitious disorders in children and adolescents are estimated to have a prevalence rate of 0.7-1.8%, similar to adults but the prevalence of factitious hypoglycemia in this population is not known [7,8].

In many factitious disorders, symptoms are inconsistent and difficult to explain using organic disease processes [5]. However, hypoglycemia in patients with diabetes may be a part of the disease process itself or a side effect of the treatment. Thus, diagnosing this disorder is even more difficult. Another diagnostic challenge is the false notion that children will not engage in self-harm. However, it is known that suicide, the ultimate form of self-harm, occurs in children and adolescents [9]. A recent meta-analysis of anxiety and depressive symptoms in youth with type 1 diabetes revealed an alarmingly high prevalence of around 30% of depressive symptoms in youth with type 1 diabetes [10]. In addition, treating physicians are more likely to pursue intellectually challenging, organic diagnoses over the diagnosis of factitious hypoglycemia secondary to surreptitious insulin use [11].

Our patient exhibited several features that supported the diagnosis of factitious hypoglycemia: female gender, adolescent age, long-standing T1DM with poor blood glucose control, an atypical history of hypoglycemia, systemic symptoms that could not be explained by an organic disease process and acute on chronic psychosocial stressors.

Most cases upon review demonstrated similar reasons for inducing hypoglycemia [11]. In a review of 39 articles, the following reasons were discovered: psychiatric or psychological conditions (including personality disorders, depression, anxiety and psychosis) were among the most frequently cited. Trying family circumstances, school avoidance, frustration with disease and desire for more sweet foods were reported across a wide range of socioeconomic levels. In most cases, counseling or psychiatric treatment were cited as useful interventions after the diagnosis of factitious hypoglycemia had been made. In addition, earlier detection and treatment of the underlying psychosocial pathology could decrease the likelihood of development of factitious hypoglycemia and its complications [11].

The same review revealed solely overuse of insulin as the mode of hypoglycemia induction [11]. They did not identify cases which occurred due to ingestion of oral hypoglycemic agents. Unlike in our patient, the use of insulin pens or insulin pumps allows a record to be maintained of the time and dose of insulin injection, potentially permitting doctors and caregivers to cross check and confirm or rule out the surreptitious use of insulin. However, the parallel use of an undisclosed route of insulin administration, for example, a syringe in addition to a pump prevents the use of this recorded data as a means of monitoring insulin dosage.

Generalized edema is an extraordinary complication of insulin therapy. It has been reported in patients with newly diagnosed diabetes following the initiation of insulin therapy, during hyperglycemic crises and in underweight patients on large doses of insulin [1,12]. Reported cases of insulin-induced edema in childhood and adolescence are scarce [13-15]. However, we present the patient

who presented with the same after many years of treatment due to a sudden increase in the dose of insulin injected. The reason for fluid retention is postulated to be multifactorial and can be attributed to increased vascular permeability and altered renal sodium handling [12,13].

It is possible that insulin edema is more common than recognized in patients. Any edema and weight gain in patients being treated for hyperglycemia is largely attributed to re-gain of fluid depleted due to osmotic diuresis. The higher incidence of thiazolidinedione induced edema in insulin users compared to non-insulin users may reflect a mechanistic similarity and synergy with fluid retention due to insulin use [16].

Conclusion

This case of factitious hypoglycemia and insulin edema secondary to surreptitious insulin use in a patient with long standing type 1 diabetes mellitus due to acute on chronic psychosocial stressors highlights the importance of providing holistic healthcare with special focus on mental well being and social support especially in children and adolescents [17]. To the best of our knowledge, this is the first report where surreptitious self administered excess insulin has led to insulin edema and factitious hypoglycemia.

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