International Research Journal of Pharmacy and Medical Sciences
ISSN (Online): 2581-3277

A Case Report of Insulin Edema Syndrome

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Abstract*—Insulin therapy rarely causes edema when rapidly correcting glucose. It is often unrecognised. Although uncommon, this occurrence usually occurs in individuals who are started on insulin treatment and have increased glucose levels. This causes a rapid correction and tight control of glucose levels. After alternative causes of acute edoema have been ruled out, insulin-induced edoema is diagnosed. Additionally, we will talk about the hypothesised mechanisms behind insulin's ability to cause edoema in this case report.*

***Keywords*—** *Insulin, pedal edema, ketoacidosis*.

I. INTRODUCTION

A very uncommon side effect of insulin therapy is insulin edoema syndrome. A less-known consequence is edoema brought on by starting insulin therapy or tightening glycemic control. When starting insulin therapy, with rapid correction and strict glucose control, this behaviour has often been described in patients with poorly controlled diabetes mellitus. However, it is difficult to determine the prevalence, provide an accurate diagnosis, and develop treatment recommendations because this syndrome is uncommon and little known. In order to emphasise this complication, investigate the aetiology and pathophysiology of insulin-related edoema, and describe a case of insulin edoema that occurred during rapid correction and strict glucose control in a diabetic patient with poorly managed glucose levels and raised haemoglobin A1c level, we present this case.

II. CASE PRESENTATION

A 52 year old male presented with complaints of severe abdominal pain and vomiting since 2 weeks, aggravated since morning. Gradual in onset. He is a known case of diabetes Mellitus and is on insulin for the past 3 years. He has not taken insulin for the past 2 weeks. He is a known case of hypertension for the past 10 years. He is also a known case of pancreatitis and has taken treatment six months back. He was admitted in an outside hospital 1 week back before these symptoms started for diabetic ketoacidosis. He was treated with insulin infusion followed by subcutaneous insulin. He was advised to continue insulin mixtard, but he stopped taking insulin after 2 days of his discharge because of his bilateral pitting pedal edema up to the knee. Edema resolved after stoppage of insulin.

He is a smoker, smokes 10 cigarettes per day for the past 20 years and alcoholic for the past 25 years. He also has diabetic neuropathy for the past 1 year. His blood pressure was 148/90 mmHg, pulse rate- 98 beats per minute, respiratory rate of 19, temperature of 98.5 degree Fahrenheit. Cardiovascular system was normal, respiratory system was normal. Bilateral pitting pedal edema present 3+ up to knees. Investigations showed diabetic ketoacidosis with anion gap of 24. Sodium is 132mmol/L, potassium is 3.5mmol/L, chloride is 94 mol/L, urea 40mg/dl, creatinine is 0.7 mg/dl, glucose was 380 mg/dl and urine ketones was 3+. CT abdomen showed features suggestive of acute pancreatitis. ECG showed normal sinus rhythm and transthoracic echo was normal.

III. DISCUSSION

A side effect of insulin therapy that results from the strict management of hyperglycemia in patients with poorly managed diabetes mellitus is known as insulin edoema syndrome. A low body mass index increases the chance of developing insulin oedema in addition to poor glycemic control. Younger patients with newly discovered type 1 diabetes mellitus also exhibit it. The lower extremities are typically affected by the mild edoema. There have been cases of severe presentations that result in ascites, pleural effusion, and anasarca.

It was thought that our patient's history of recurrent pancreatitis, also known as pancreatogenic diabetes or Type 3c, in conjunction with alcohol usage was what caused his insulin-dependent diabetes.

The development of edoema has been attributed to a number of causes, including an increase in capillary permeability and salt retention in the kidneys [6]. Insulin is known to stimulate the Na+/K+-ATPase and the expression of Na+/H+ exchanger 3 in the proximal tubule, which leads the kidneys to reabsorb salt. It also produces vasodilation, which aids in fluid retention. In addition to raising vascular permeability, hyperglycemia independently contributes to the development of edoema in patients with chronically high glucose levels Patients with diabetes who have chronically high blood sugar levels have less-than-optimal vascular membrane integrity, which raises their chance of developing edoema from the rapid variations in serum osmolality.

The "refeeding edoema" occurrence lends support to the postulated mechanism. After a protracted period of fasting, refeeding edoema develops in response to an increase in endogenous plasma insulin.

Treatment options for insulin edoema range from conservative care, which limits salt and fluid consumption, to pharmacotherapy with diuretics, epinephrine, or ephedrine in cases where other methods have failed. It has also been demonstrated that changing the insulin dose can lessen the edoema. As was mentioned above, the patient in this case saw

Preethi CD, N.Anuradha, Viknesh Prabhu, Konda Sanjith, and Nethaji Sree Vaishnavi, "A Case Report of Insulin Edema Syndrome," *International Research Journal of Pharmacy and Medical Sciences (IRJPMS)*, Volume 6, Issue 4, pp. 3-4, 2023.

a decrease in leg swelling after stopping his insulin use, which regrettably led to his non-compliance and other complications that required hospitalisation due to DKA. He took 20 mg of furosemide daily during his hospitalisation, and his condition quickly improved.

IV. CONCLUSION

A very uncommon side effect of insulin therapy is insulin edoema syndrome. The differential diagnosis of suitable patients who appear with edoema should include this clinical condition. The cardiovascular and respiratory systems may experience difficulties; they should be taken into account. It is plausible to assign the aetiology to this underappreciated phenomena when alternative cardiac, hepatic, vascular, and nephrotic causes of abrupt onset edoema have been ruled out. Patients may feel more at ease and confident about their insulin regimen if they are aware of this illness.

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Preethi CD, N.Anuradha, Viknesh Prabhu, Konda Sanjith, and Nethaji Sree Vaishnavi, “A Case Report of Insulin Edema Syndrome,” *International Research Journal of Pharmacy and Medical Sciences (IRJPMS)*, Volume 6, Issue 4, pp. 3-4, 2023.