Case Report

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Diabetic ketoacidosis associated seizures: a case report

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Abstract

Diabetic ketoacidosis is the complication of Diabetes mellitus, which can result due to extreme hyperglycemia and ketonaemia. This condition is prevalent among patients with untreated diabetes or drug incompliance. Hyperglycemic hyperosmolar state can clinically be presented with episodes of seizures with high blood sugar levels and absence of ketones and has low incidence rate. A 55 year old male patient presented with episode of seizure, frothing, tongue bite, uprolling of eyes, shortness of breath, pedal edema and high blood sugar levels. Past medical history includes known hypertensive and diabetic since 10 years and is on inj regular insulin with non-adherence to medication. Lab investigations were performed and patient was diagnosed as hyperglycemic diabetic ketoacidosis secondary to drug incompliance. The patient was managed by insulin regimen- regular and basal insulin, antibiotics, antiepileptic drugs and by continuous monitoring of blood sugar levels every 3rd hourly. Diabetic ketoacidosis was recovered and later, there were no episodes of seizures. Creating awareness and proper patient education regarding the disease state, pharmacotherapy and drug compliance plays a vital role in managing the condition and as well as by preventing further complications that might be fatal in some cases.

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Introduction

Diabetic Ketoacidosis (DKA) is one of the acute complications of Diabetes mellitus, DKA is characterized by plasma glucose >250 mg/dl, arterial pH <7.30 or serum bicarbonate < 15 mEq/L and moderate ketonaemia with ketonuria. This is the most frequent endocrine emergency. It has a mortality of 6 to 10%. All the abnormalities can be traced to an absolute or relative insulin lack, which develops over a period of several hours or days. It is more common in type 1 diabetes. Patient present with polydipsia, polyuria and weakness, anorexia, vomiting and abdominal pain due to ketonaemia. Ileus and gastric dilatation may cause aspiration. A characteristic type of breathing called kussmaul's breathing (deep, sighing) occurs as a Respiratory compensation for metabolic acidosis especially when pH <7.2. Patients may have altered sensorium and 10% of them are comatose. Clinical examination shows fruity or musty odor in breath, loss of skin turgor, dry tongue and decreased intraocular pressure resulting in sunken eyes, hypothermia, Tachycardia, hyperpnoea or kussmaul's breathing depending on degree of acidosis, hyporeflexia (decreased potassium), signs simulating surgical abdomen, hypotonia, stupor or coma, evidence of precipitating illness. Hyperosmolar Coma: When insulin deficiency is partial as in Type 2 DM patients, the anti catabolic effect of insulin may be relatively well-preserved while its anabolic action is more seriously defective. In these circumstances lipolysis is not markedly accelerated and the concentrationof ketone bodies in the blood remains relatively normal despite severe hyperglycaemia. This state is called hyper osmolar coma. Most of the patients have mild type II diabetes or noprior history of diabetes. Lack of ketosis in this syndrome has been explained by insulin levels high enough to prevent lipolysis and ketogenesis but not high enough to prevent hyperglycaemia. Clinical Features: Polyuria, polydipsia, weight loss, weakness, altered sensorium, evidence of underlying conditions and seizures are common. Patient is severely dehydrated and may present with neurological deficits [1].

Diabetic emergencies: Hypoglycaemia and extreme hyperglycaemia, causing diabetic ketoacidosis or hyperosmolar hyperglycaemic state, constitute the three acute emergencies associated with diabetes. Treatment of diabetic ketoacidosis- Treatment comprises fluid volume expansion (initially with 0.9% sodium chloride), correction of hyperglycaemia and the presence of ketones (by infusion of insulin), prevention of hypokalaemia, and identification and treatment of any associated infection. Once the patient is better, they should be reviewed by the diabetes team in order to discuss how to avoid future episodes of diabetic ketoacidosis. Treatment of HHS- Treatment requires fluid replacement to stabilise blood pressure and improve circulation and urine output. Sodium chloride 0.9% or 0.45% (if serum sodium is greater than 150mmol/L) is given and monitoring of blood pressure and cardiovascular status undertaken. Potassium may be added if required. Insulin treatment is started via intravenous infusion

but is not aggressive, since fluid replacement also lowers serum glucose levels. Prophylaxis or treatment for thromboembolism may also be required [2].

The mainstay in the treatment of DKA involves the administration of regular insulin via continuous intravenous infusion or by frequent subcutaneous or intramuscular injections. Randomized controlled studies in patients with DKA have shown that insulin therapy is effective regardless of the route of administration. The administration of continuous intravenous infusion of regular insulin is the preferred route because of its short half-life and easy titration and the delayed onset of action and prolonged half-life of subcutaneous regular insulin [3].

Regular insulin is used to rapidly correct the metabolic abnormalities. A bolus dose of 0.1-0.2U/kg i.v is followed by 0.1 U/kg/hr infusion, the rate is doubled if no significant fall in blood glucose occurs in 2 hr. fall in blood glucose level by 10% can be considered adequate response. Usually, within 4-6 hours blood glucose reaches 300mg/dl. Then the rate of infusion is reduced to 2-3 U/hr. this is maintained till the patient becomes fully conscious and routine therapy with s.c insulin is instituted [4].

Case Study

A 55 year old male patient presented to causality with chief complaints of seizures one episode GTCS type, frothing, tongue bite, up rolling of eyes, complain of SOB grade-2, bilateral pedal edema, abdominal distention and facial puffiness since 3 days. History of present illness includes complain of bleeding per rectum and pain, bladder incontinence, decreased urine output, burning micturition, Urgency. Known complaint of hemorrhoids. Past medical history included hypertension since five years on unknown medication's and diabetes since 10 years on irregular intake of regular insulin 20 U and 25 U. He is a known smoker and chronic alcoholic. On examination patient was in an altered mental state. Vital signs: BP: 100/70, PR: 86bpm, CVS: S1S2, SPO2: 97%, RS: BAE+ no crepts, per abdomen: hypogastric tenderness, CNS: power- 5/5 in both UL and LL. Tone normal, plantar reflexes- decreased. On external Inspection and DRE: skin tag present at 7'o clock position, no external hemorrhoids/fistula, EAS tone normal.

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
GRBS(mg/dl)	300	532	710	520	615	504

Lab Investigations

Arterial blood gas values: pH- 7.307, pCO2- 20.8, pO2- 108, HCO3- 12.6 mEq/L,CBP: WBC- $5.77\times10^3/\mu L$, RBC- $1.68\times10^6/\mu L$, HGB- 4.1 g/dL, HCT- 14.6%, MCH- 24.4pg, MCHC- 28.1g/dL, PLT- $208\times10^3/\mu L$(Imp- Microcytic Hypochromic Anaemia), INR- 1.5sec,HbA1c- 9.0%, LFT values: ALT- 168 U/L, AST- 320 U/L, TP- 5.73, Crea- 1.31 mg/dL, Urea- 57.2 mg/dL, Na- 130mmol/L, K-7.6mmol/L, Cl- 104mmol/L, upper GI endoscopy: Hiatus hernia prolapsing gastropathy antral gastritis, Urine for ketone bodies: negative.

Final Diagnosis

New onset GTCS seizures with hyperglycemic DKA 2° to Drug Incompliance with Lower GI bleed 2° to fissure with severe anaemia.

Treatment

The patient was prescribed by the following initially for four days: Inj Regular Insulin 30U in 250ml NS @50 ml/hr (till GRBS <250mg/dL), Inj Levipil 500 mg IV BD (D1), Inj Pantop 40mg IV OD, Inj midazolam 2mg dil in 4ml IV slow (D1, D2), Inj Thiamine 100 mg IV OD, Inj ceftriaxone 1gm IV BD, Tab BC/MVTPO OD, Tab IFA 200 mg PO OD, Tab Vit C PO OD, Tab Eptoin 100 mg IV BD (D3, D4), planning for 2PRBC, Tab. Aten 25mg PO OD (D3, D4), plan for Sitz bath daily, Syr Lactulose 10ml PO BD, monitoring GRBS 3rd hourly and watch for seizures. Later the patient was maintained on Inj Regular Insulin 15U SC TID, Inj Basal Insulin 30U and 15U SC BD, Inj Pantop 40 mg IV OD, Tab Amlo 5mg PO OD, Syr Lactulose 15ml PO TID, Tab BC/MVT/Ca PO OD with monitoring GRBS 6th hourly. On Day 6th1PRBC transfusion was done. Treatment with above Insulin regimen, DKA was recovered and certain lab values such as CBP, ABG were repeated and there was moderate improvement in the counts and there were no episodes of seizures on follow up. Patient was counselled about importance and continuation of insulin therapy after the discharge.

Discussion

Many cases of DKA and HHS can be prevented by better access to medical care, proper patient education, and effective communication with a health care provider during an inter current illness. Paramount in this effort is improved education regarding sick day management, which includes the following: Early contact with the health care provider, Emphasizing the importance of insulin during an illness and the reasons never to discontinue without contacting the health care team, Review of blood glucose goals and the use of supplemental short- or rapid-acting insulin, Having medications available to suppress a fever and treat an infection, Initiation of an easily digestible liquid diet containing carbohydrates and salt when nauseated, Education of family members on sick day management and record keeping including assessing and documenting temperature, blood glucose, and urine/blood ketone testing; insulin administration; oral intake; and weight. Similarly, adequate supervision and staff education in long-term facilities may prevent many of the admissions for HHS due to dehydration among elderly individuals who are unable to recognize or treat this evolving condition. The use of home glucose-ketone meters may allow early recognition of impending ketoacidosis, which may help to guide insulin therapy at home and, possibly, may prevent hospitalization for DKA. In addition, home blood ketone monitoring, which measures β-hydroxybutyrate levels on a finger stick blood specimen, is now commercially available [3].

Seizures at initial presentation in the clinical setting of uncontrolled hyperglycemia, hyperosmolar state, and absence of blood and urinary ketones along with subcortical T2 hypo-intensity most likely represents hyperglycemia-induced seizures. Awareness of this entity helps avoid misdiagnosis,

inadvertent invasive workup and initiate appropriate management [5].

Conclusion

Diabetic ketoacidosis if left untreated can cause seizures and in some cases can lead to death. This case study have shown that involvement of healthcare professionals in effective patient education and counselling on diabetes and its obvious symptoms, related complications such as diabetic ketoacidosis, adherence to diabetic diet, best treatment outcomes, the need for monitoring blood glucose levels constantly and significance of insulin therapy as prescribed in drug compliance can have better health outcomes in improving quality of life.

Conflict of Interest

There is no conflict of interest behind this study.

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