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Case Study

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HYPER-OSMOLAR HYPER GLYCEMIC STATE: A CASE REPORT

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ABSTRACT

Hyperosmolar Hyperglycemic State (HSS) is an urgent medical condition associated as a complication of Type 2 Diabetes. It usually occurs by certain triggering factors and needed sudden intensive care. This case study is mainly aimed to represent the actual scenario for treatment of HHS in a 50-year-old female patient who presented with severe breathlessness, cough and wound infection. From past 2 days she had intense thirst and on vital examination everything was found to be normal except respiratory rate which was slightly elevated. On the basis of laboratory examination, she was diagnosed with HHS and proper treatment was given with insulin, oral hypoglycemic agents, antibiotics, fluid correction, and electrolyte correction. She was discharged after complete resolution of symptoms and a review after 3 months, the HBA1C value showed a good decline from initial value. This case report emphasizes that early recognition and proper treatment of HHS improves clinical outcome and prevents serious complications.

KEYWORDS: Diabetes, breathlessness, hyperosmolar hyperglycemic state, HBA1C.

INTRODUCTION

Hyperosmolar hyperglycemic state is a severe complication of Type 2 Diabetes mellitus having a mortality rate up to 5% to 20%. It is a condition in which there is extreme hyperglycemia with elevated plasma osmolality and absence of ketogenic substances.[1] The common triggering factors of HHS include nonadherence to diabetes treatment, acute infections, carbohydrate rich beverages and medications like diuretics, beta blockers, glucocorticoids and atypical antipsychotic drugs.[2] HHS has similar pathophysiology when compared to diabetic ketoacidosis due to deficiency of hormone insulin which leads to decreased glucose utilization by the tissues causing hyperglycemia. This causes the release of counter regulatory hormones glucagon, catecholamine and cortisol which stimulates gluconeogenesis and glucogenolysis which in turn cause increases the level of glucose in serum resulting in hyperosmolar state.[3] The current diagnostic criteria for HHS is glucose level above 600 mg/dl and elevated serum osmolality of >295mmol/kg. The serum osmolality is determined by the formula 2Na+Glucose/18+BUN/2.8. When compared to DKA the occurrence of ketone body is absent in HSS patients but there is an increase in serum urea which reflects dehydration level. Here, we report a rare case of HSS as a complication of Type 2 Diabetes mellitus in a 50-year-old female patient and to discuss the importance of early management of hyperglycemic crisis in dehydration.[4]

CASE REPORT

A 50 year old female patient was admitted to the emergency department with severe breathlessness, cough, facial puffiness and wound infection on her right leg. She had Type 2 Diabetes mellitus for past 8 years and was on Metformin 500mg twice daily. For last 1 month she was nonadherent to her medications. She had intense thirst and lethargy for past 2 days. There is no family history of endocrine disease including diabetes mellitus, thyroid diseases. The patient was conscious and disoriented at the time of admission. All the vitals were checked and found to be normal except respiratory rate which was 26/min. She was 160cm tall and weighed 50kg and observed a body mass index of 19.5kg/m^2.

INVESTIGATIONS

A complete hemotogram was performed which revealed an elevation in WBC count 12000cells/cmm, polymorphs 85%, ESR 50mm/hr, blood urea 70mg%, Random blood glucose level 658 mg%, HBA1c level 11% and serum osmolality was 328.735 mmol/kg. Liver function test and creatinine(0.7mg/dl) was found to be normal. GRBS was found to be 422mg/dl and 525 mg/dl during evening and night .on the next day, FBS was found to be 146mg/dl and GRBS was 375mg/dl and 391mg/dl during evening

and night. Urea level was found to be normal (35mg%) as the treatment was progressed. On the basis of her laboratory values elevated glucose levels, serum osmolality and urea she was diagnosed with Hyperosmolar hyperglycemic state.

TREATMENT

She was prescribed with, intravenous venous human Actrapid 6U at noon and 4U at night and subcutaneous insulin Human actrapid 8U-8U-10U, 0.9% sodium chloride infusion on the first day of admission. On the second day the dose of insulin was changed to 10U-16U-20U -10U. She was also administered with 500 ml 0.9% normal saline along with 20 meq Potassium Chloride over 5hrs on the next day since the patient showed hypokalemia (3.1 mmol/l). On day 3, 500 ml 0.9% normal saline along with 20meq potassium chloride and 50% $MgSO_4$ infused over 5 hour. Tablet Glimepiride 2mg BD, Tablet voglibose 0.3mg TID, Tablet azithromycin 500mg OD was given throughout the hospital stay. Tablet Ebastine 10mg OD, injection ceftriaxone and tazobactum 1.125mg BD, INJ Pantoprazole 40mg OD were given during on all 4 days of hospital stay.

OUTCOME AND FOLLOW UP

After 4 days of hospital stay patient was discharged with Tab. cefpodoxime (200 mg) and clavulanic acid (125 mg) twice daily, Tab.azithromycin 500 mg OD and Cap rabeprazole 20 mg BD for 3 days followed by Cap. Acebrophylline 100 mg BD, Tab glimipride 2 mg BD, Tab. Voglibose 0.3 mg TID, Tab Teneligliptin 20 mg BD for 10 days. A review after 3 months of discharge her HBA1c level was found to be 8.7%.

DISCUSSION

Hyperosmolar hyperglycemic state and diabetic ketoacidosis represent two major complications of type 2 diabetes mellitus manifested by insulin deficiency and severe hyperglycemia with an estimated mortality rate of 2.5 to 9%.[5] In India the large epidemiological study revealed that annual incidence of HSS is 4.6 per 100000 of diabetic population and 0.14 cases per 100000 general population. It has been estimated that out of all primary diabetic hospital admissions, less than 1% are for HHS.[2] As the prevalence of type 2 DM increases, the occurrence of HHS will likely increase as well.

Higher elevation in serum glucose level and hyper osmolality without ketosis are the major diagnostic parameter for HHS. The interruption in metabolism causes increase in the amount of counter regulatory hormones like cortisol, glucagon, catecholamine and growth hormones. Increased gluconeogenesis and conversion of glycogen to glucose resulting in inadequate utilization of glucose by peripheral tissues thus lead to hyperglycemic state. Worsening of hyperglycemia produce glycosuria which further lead to loss of water and electrolytes resulting in dehydration which precipitate hyper osmolar hyperglycemic state.[3]

A more than few differences exist in the treatment of DKA and HSS. However intensive fluid hydration is the mainstay of initial management in both the conditions. The recommended hydration by isotonic solution is 10-20 Ml/kg/hr during the first hour. During this phase serum sodium level should be monitored since rapid correction may lead to cerebral edema and central pontine myelinosis.[1] After the primary fluid resuscitation, the corrected sodium level should be calculated using the formula

Corrected sodium = measured sodium + [(glucose – 5)/3.5]

If the corrected sodium is below 135 meq/l the isotonic saline should be continued at a rate of 250-500ml/hr. if the corrected sodium is normal or elevated then 0.45% saline should be used at a rate of 10ml/kg/hr for 18-24hrs. When the glucose level reaches 250-300 mg/dl infusion should be changed to 5% dextrose in 0.45% normal saline.[2]

After first litre of saline has been used insulin treatment should be initiated at 0.1U/kg IV bolus followed by an infusion of 0.1U/Kg/hr. When plasma glucose level reaches 300mg/dl insulin infusion should be decreased to 1 -2 U/hr.[2,6] In our case report the patient was managed with subcutaneous and intravenous insulin therapy instead of insulin infusion and the patient responded well to the given therapy.

Rapid correction of hyperglycemia with insulin may lead to hypokalemia. So serum potassium should be monitored continiously throughout the infusion. The correction of serum potassium is similar to that of DKA that is if the potassium level is less than 3.3 mEq/L administer potassium at 40 mEq/hr. If serum potassium is between 3.3 -4.9 then 20 mEq/L should be given. In this case report the initial serum potassium was 3.1 mEq/L but the patient was given with 20 mEq/L of potassium.

The limitation of our report is that proper estimation of GRBS at an interval of 2 hr is not performed so the effectiveness of the insulin cannot be properly understood. And as the patient refused to take insulin after the discharge, the 3 month HBAIc cannot maintained on its normal range.

REFERENCES

1. Young min Cho MD, Byung Sung Park MD, Min Jae Kang MD: A case report of hyperosmolar hyperglycemic state in a 7-year-old child an anusual presentation of first apperence of type 1diabetes mellitus; Medicine, 2017 Jun; 96(25): e7369.
2. www.medscape.com- Hyperosmolar hyperglycemic state
3. Francisco J. Pasquel and Guillermo E. Umpierrez: Hyperosmolar Hyperglycemic State: A Historic Review of the Clinical Presentation, Diagnosis, and

Treatment-American diabetic association Diabetes Care, 2014 Nov; 37(11): 3124-3131.
4. Annabel L. Bradford, a,d Courtney Champagne Crider, b Xizheng Xu, c and Syed Hasan Naqvic: Predictors of Recurrent Hospital Admission for Patients Presenting With Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State; journal of clinical medicine research, 2017 Jan; 9(1): 35–39.
5. Mohd Ashraf Ganie, Satish Koul, Hilal A Razvi, Bashir Ahmed Laway, Abdul Hamid Zargar: Hyperglycemic emergencies in Indian patients with diabetes mellitus on pilgrimage to Amarnathji yatra; Indian journal of endocrinology and metabolism, 2012; 16(7): 87-90.
6. Abbas E. Kitabchi, PHD, MD, Guillermo E. Umpierrez, MD, John M. Miles, MD and Joseph N. Fisher, MD: Hyperglycemic Crises in Adult Patients with Diabetes; American diabetic association Diabetes Care, 2009 Jul; 32(7): 1335-1343.