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Diabetic Ketoacidosis in type 1 Diabetes Mellitus

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ABSTRACT

Diabetic ketoacidosis (DKA) is a state of metabolic decompensation/disorder characterized by the triad of hyperglycemia, acidosis and ketosis, caused by absolute or relative insulin deficiency and increased counter-regulatory hormones. Immediate therapy in DKA patients determines the patient's prognosis. A 19-year-old male treated in the internal medicine ward of Dr. M djamil Padang with diabetic ketoacidosis, type 1 DM, and Abscess capitis. The patient was treated with rehydration and intravenous insulin infusion can show clinicall improvement. The diagnosis in patients is based on anamnesis, physical examination and supporting examinations. Immediate therapy is needed in the management of DKA in patients. After DKA is resolved, look for the causes of the risk of DKA. On the third day of treatment the patient's condition improved clinically and the laboratory.

Keyword: Diabetic Ketoacidosis, Diabetes Mellitus type 1

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1. Introduction

Diabetic ketoacidosis (DKA) is a state of metabolic decompensation/disorder characterized by the triad of hyperglycemia, acidosis and ketosis, caused by absolute or relative insulin deficiency and increased counter-regulatory hormones. Diabetic ketoacidosis is one of the acute complications of diabetes that is closely related to the quality of education in people with type 2 diabetes mellitus (DM) and is often the starting point for the diagnosis of type 1 DM.[1,2]

The most common precipitating factor that plays a role in the occurrence of DKA is infection. Other triggers include stopping or reducing insulin doses, myocardial infarction, acute pancreatitis, use of drugs. Data from the RSCM showed that infection was 80% of the causal factors for DKA, and infections that were often found were urinary tract infections and pneumonia, while other studies reported that the causes of DKA that had been reported included inappropriate insulin doses (48.6%), gastroenteritis (14.1%), technical problems with the insulin pump (12.7%), infections (13.4%), social problems (1.4%) and idiopathic (5.6%).

2. Case Report

19-year-old male patient with loss of consciousness since 4 days ago. Often feel thirsty, hungry and urinate since 2 years ago. Shortness of breath since 5 days before admission to the hospital. Heartburn since 5 days

before admission to the hospital. Pain accompanied by nausea. There is vomiting, the frequency is 2 times filled with liquid and food. The patient also complained of a lump on his head and pus coming out since 1 month ago. The patient also complained of fever 1 week ago. On physical examination the patient looked seriously ill with sopor, BP 80/50 mmHg, pulse 110x/minute, respiratory rate 26x/minute, Kussmaul breathing pattern. There is a mass measuring 4x4 cm in the frontoparietal region, fluctuating Laboratory tests were carried out with leukocytes 18,470/mm3, GDS 496 mg/dL. Urinalysis showed glucosuria (+2) and proteinuria (+1), and urine ketones +2. Blood gas analysis obtained pH 7.00; HCO3- 5.3 mmol/L with the impression of metabolic acidosis and hypernatremia (Na 162 mmol/L). There was also an increase in D-dimer 3545 ng/mL, creatinine 2.1 mg/dL, HbA1c > 15. HOMA-IR and C-peptide examination with low C-peptide results and high HOMA-IR due to bias in the use of insulin, an impression of type 1 diabetes mellitus.

Based on the anamnesis, physical examination and support, the patient was diagnosed with decreased consciousness ec diabetic ketoacidosis, type 1 diabetes mellitus, sepsis ec Abeses capitis a/r frontotemporalis, acute on CKD, hypernatremia ec dehydration and high risk of VTE. Patients are treated according to the diabetic ketoacidosis protocol until the patient is conscious, GDS is controlled, urine ketones are negative, and insulin is then given.long actingAndshort actingand given antibiotics in the form of Metronidazole 3x500 mg IV, Cefepime 3x2 gram IV and Levofloxacin 1x750mg IV.

3. Discussion

Diabetic ketoacidosis (KAD) is an emergency in the field of internal medicine which is characterized by hyperglycemia, acidosis and ketosis. The diagnosis of DKA is established in patients according to the DKA triad. The patient had a blood sugar level of 497 mg/dL, acidosis characterized by shortness of breath with a Kussmaul breathing pattern which was confirmed by blood gas analysis results obtained blood pH 7.0, serum bicarbonate 5.3 mmol/L, high anion gap 55.3 mEq/L (normal value 8-12 mEq/L), as well as the presence of ketosis on a positive urine ketone examination 2. This is in accordance with the criteria of the American Diabetic Association, namely DKA is enforced if blood sugar levels are found > 250 mg/dl, pH acidosis is below 7.35, serum bicarbonate <18 mEq/dl and ketosis.

DKA is defined as a decompensated state of metabolic derangement characterized by a triad of hyperglycemia, acidosis and ketosis, primarily caused by absolute or relative insulin deficiency. This insulin resistance occurs simultaneously with an increase in counterregulatory hormones such as glucagon, catecholamines, cortisol and growth hormone. Both of these will result in changes in glucose production and glucose expenditure and increase lipolysis and production of ketone bodies. Hyperglycemia occurs due to increased hepatic and renal glucose production due to gluconeogenesis and glycogenolysis and decreased utilization of glucose in peripheral tissues.

Increased hepatic glucose production suggests the main pathogenesis responsible for hyperglycemia in DKA patients. Hyperglycemic state and high ketone levels cause osmotic diuresis which will result in hypovolemia and decreased glomerular filtration rate which will exacerbate the hyperglycemic state. Activation of hormone-sensitive lipase in fat tissue due to insulin deficiency and increased counterregulatory hormones, increases the activity of breaking down triglycerides into glycerol and free fatty acids. Glycerol is an important substrate for gluconeogenesis in the liver, while free fatty acids are oxidized to ketone bodies whose process is mainly stimulated by glucagon, resulting in increased ketogenesis.

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DKA can occur in patients with type 1 and 2 diabetes mellitus, but is estimated to be more than twice as common in patients with type 1 diabetes mellitus. In patients with type 2 diabetes mellitus, DKA usually occurs due to inappropriate treatment or complications from infection and dehydration. In patients with type 1 diabetes, DKA is often the first feature at diagnosis. The incidence of type 1 diabetes is increasing by 3% to 5% annually worldwide. According to research it is said that one third of children show DKA as the initial presentation of type 1 diabetes. These children and adolescents experience hyperglycemia, acidosis, and

ketosis. This is in accordance with the patient's condition when he was first diagnosed at the age of 19 years with clinical manifestations of DKA.[3]

The most common clinical manifestations are nausea and vomiting, followed by abdominal pain. According to Set et al. (2015) reported nausea, vomiting, and pain in the abdomen as the most common clinical presentations in patients with DKA. In the study by Shaltout et al (2016) severe vomiting and abdominal pain were reported as the most common symptoms. Ongoing catabolism and acidosis in DKA patients can lead to extreme vomiting. According to research dehydration occurs in 30% of patients with DKA. Osmotic diuresis due to glycosuria causes dehydration and electrolyte disturbances.[4,5]

In the diabetes mellitus population, catabolic stress due to acute illness or injury such as trauma and surgery, as well as infection can trigger DKA. Common precipitating factors for DKA are nonadherence to therapy, newly recognized diabetes, and other acute illnesses. In the patient, a source of abscess infection was found in the frontotemporal capitis region and drainage was carried out on the abscess and the patient was just diagnosed with DM type 1.[6]

In patients found a C-peptide value of 0.21 ng/dL with a low C-peptide effect. Measuring diabetes-specific autoantibodies in adults with early diagnosis of type 1 diabetes (serum C-peptide). It is advisable to confirm the diagnosis of type 1 DM by examining anti-islet cell antibody (ICA). Diabetic-related autoantibodies are glutamicacid decarboxylase 65 autoantibodies (GAD); tyrosine phosphatase like insulinoma antigen 2 (IA2); insulin autoantibodies (IAA); and β-cell-specific zinc transporter 8 autoantibodies (ZnT8). The presence of one or more of these autoantibodies helps confirm the diagnosis of type-1 DM. Diagnosis and management Children and adolescents with type 1 diabetes should be screened for celiac disease and thyroid disease at diagnosis (and annually thereafter). retinopathy, Microalbuminuria and blood pressure were evaluated annually from the age of 12 years.[7]

The management of DKA in these patients is carried out by replacing lost body fluids and salts, suppressing lipolysis of fat cells and suppressing liver cell gluconeogenesis by administering insulin, overcoming metabolic stress as a trigger for diabetic ketoacidosis, and restoring normal physiological conditions and close monitoring and adjustment of treatment. According to Kitachi et al. criteria for improvement in DKA were glucose levels <200 mg/dl, serum bicarbonate ≥18 mEq/l, venous pH >7.3 and ketosis resolved. Fluid therapy to improve organ perfusion is the first priority. The liquid of choice is 0.9% NaCl. Treatment of the patient was found to be sodium 162 with the choice of 0.45% rehydration fluid followed by continuous intravenous drip insulin. On the 2nd day of treatment the patient has experienced clinical and laboratory improvements, where blood sugar is obtained when it is below 250 mg/dl, pH is 7.409 and urine ketones are negative.[8]

4. Conclusion

In type 1 diabetes melitus, DKA is often the initial presentation when diagnosed. According to the criteria of the American Diabetic Association. DKA is enforced if blood sugar levels > 250 mg/dl are found, pH acidosis is below 7.35, serum bicarbonate < 18 mEq/dl and ketosis. The trigger for the patient's DKA was infection Sepsis ec abscess capitis et frontoparietal region. Proper treatment of abscesses and DKA conditions was saved life of the patient.[5-6]

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