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Subarachnoid Hemorrhage as a Complication of Diabetic Ketoacidosis in Early Onset Type 1 Diabetes Melitus in Limited Resource Settings

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ABSTRACT

Background: Diabetic ketoacidosis (DKA) is one of the most frequent causes of morbidity and mortality in type 1 diabetes mellitus (T1DM). Neurological complications that can occur in DKA patients are cerebral edema followed by ischemic and hemorrhagic strokes. Hemorrhagic strokes have higher mortality than ischemic strokes in T1DM patients. ***Case:*** An 11-year-old patient came to the emergency room with complaints of decreased consciousness since morning. During the previous 2 days the patient had nausea and vomiting. There was no history of trauma or fever. About 2 days before admission patient showed decrease of consciousness. On physical examination, he was found to be in a coma, kussmaul breathing, mydriatic pupil, and with kussmaul breathing. Laboratory examination showed hyperglycemia, leukocytosis, hyponatremia, ketonuria, and C-peptide of 0,560 ng/ml. Head CT-Scan showed subarachnoid hemorrhage and cerebral edema. The patient showed a positive response after rehydration, administration of mannitol, and blood sugar regulation with an insulin pump. ***Discussion:*** DKA is one of the manifestations of T1DM. DKA is confirmed by hyperglycemia, blood pH below 7.3 manifesting with kussmaul breathing and the presence of ketones in blood and urine tests. DKA is treated with rehydration and insulin administration. SAH is a rare manifestation of DKA. The diagnosis of SAH can be confirmed by neuroimaging examination. Management of SAH could be done through close monitoring of blood pressure, prevention of increased intracranial pressure, and surgical approach. ***Conclusion:*** Subarachnoid hemorrhage is a neurological complication that rarely happens in DKA and could be caused by several factors such as hypotensive episode, hyperketonemic state, arteriovenosus malformation rupture, and endothelial perturbation. Neuroimaging could detect early neurological complication in DKA.

Keywords: Subarachnoid hemorrhage; diabetic ketoacidosis; kussmaul breathing; neuroimaging

INTRODUCTION

Diabetic Ketoacidosis (DKA) is serious complication of Type 1 Diabetes Melitus (T1DM) [1]. DKA theoretically can also present in Type 2 Diabetes Melitus, but in T1DM, DKA is one of the most common complications. When insulin is very low, the body falls into a ketoacidosis state. DKA diagnosis could be established with hyperglycemia, metabolic acidosis, and ketosis [2].

DKA is one of the most common presentations of T1DM with almost one third of patient with newly diagnosed T1DM present with DKA. Data from United States Suggest that 3% of children with T1DM present with DKA and it is the most common cause of hospitalization, morbidity, and mortality [2].

Hemorrhagic stroke is a rare complication of DKA. Stroke itself is pretty uncommon in DKA, and could be further classified as hemorrhagic and ischemic stroke. Hemorrhagic stroke in DKA could manifest as subarachnoid or intraparenchymal hemorrhage. When this complication present, it could worsen the outcome of pediatric DKA [3,4].

We presented a 11 years old male with DKA as manifestation of T1DM complicated by the presence of cerebral edema and SAH.

CASE

A 11 years old male was referred by a local clinic because of continuous vomiting, nausea, and headache 3 days before admission. On admission day patient was referred by a local clinic with decrease of consciousness and Kussmaul breathing. On admission we found no history of trauma or previous medical history. On admission patient was comatose, dyspsnea, had midiatric pupil, with kussmaul breathing. From laboratory assessment we found hiperglycemia, leucocytosis, hyponatremia, ketonuria, and low C-peptide, and A1c level of > 14.5. Head CT-Scan showed subarachnoid hemorrhage, and cerebral edema.

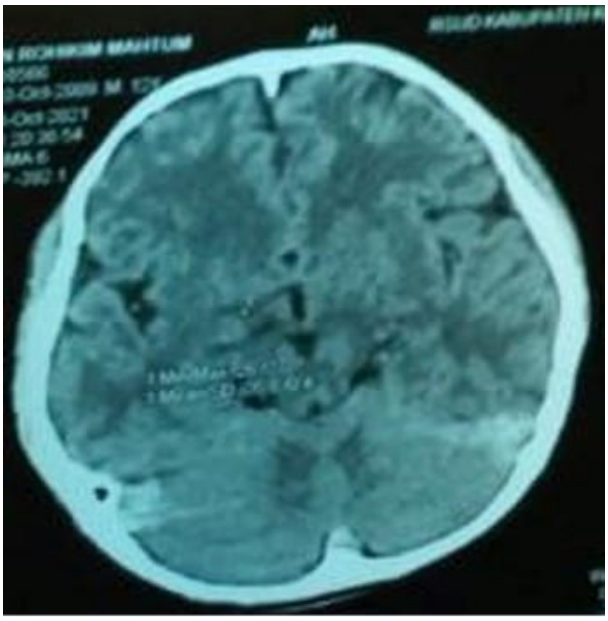

FIGURE 1: Head CT-Scan showed cerebral edema and subarachnoid hemorrhage.

FOLLOW UP

After rehydration, insulin treatment, electrolyte correction, and mannitol treatment, patient showed improvement. Then he was discharged after ten days of treatment.

DISCUSSION

An 11-year-old patient was consulted due to decreased of consciousness 14 hours before with kussmaul breathing, hyperglikemia and ketonuria. Metabolic acidosis in our case was not accompanied by blood gas examination data due to limited facilities, however the presence kussmaul breathing could be evidence of it.

Guidelines from International Society for Pediatric and Adolescent Diabetes, DKA could be defined by the presence of all the following variables: (1) Hyperglycemia (blood glucose > 200 mg/dl (2) Metabolic Acidosis (venous pH < 7,3 or serum bicarbonate < 15 mEq/L) manifesting kussmaul breathing, and (3) Ketosis (presence of ketones in blood or urine)[8].

Severity of DKA could also be classified further as mild, moderate, and severe based on pH and HCO_3 level [11].

TABLE 1: DKA classification. [11]

DKA classification	pH	HCO_3
Mild	< 7.3	< 15 mmol/L
Moderate	< 7,2	< 10 mmol/L
Severe	< 7,1	< 5 mmol/L

The etiology of DKA in children could be caused by several factors. Usually when the insulin falls below certain levels, glucose is no longer used as energy. In the absence of insulin, the body could not control the level on glucose in the body, and rises to dangerous level. There are few processes that occur in children with DKA, such as hypergylcemia, glucosuria, presence of ketones, potassium deficits, elevated kidney function, and phosphate deficit [2].

DKA is present in newly diagnosed children with T1DM. DKA is the most common cause of hospitalization, mortality, and morbidity in T1DM children. The fatality rate of DKA could reach 0.15-0.31% of cases.

There are several factors that could increase the risk of DKA in T1DM such as children from ethnic minority, children under 5 years, children from low socioeconomic family, and delayed diagnosis of diabetes [2].

In children with established T1DM diagnosis, there are several factors that could contribute to DKA, which happens at annual rate of 6-8%. These factors including: peripubertal and adolescent girls, failure of glycemic control, dehydration and gastroenteritis, psychiatric disorders, limited access to medical care, failure of insulin therapy [2].

There are several clinical signs that could rise a suspicion into DKA. The usual clinical manifestations are dehydration, tachycardia, tachypnea, Kussmaul breathing (deep sighing with ketones smell), nausea, vomiting, abdominal pain, confusion, decrease and or loss of consciousness [11].

There are several important laboratories works that should be done in DKA. Hyperglycemia usually occurs, but usually are mild (> 200 mg/dL). Some children could have severe hyperglycemia (>1000 mg/dL). Venous blood gas test also should be done and usually show a pH below 7.3, pH below 7.2 indicate worse prognosis and demand intensive care unit admission. Urinalysis is also important test and usually show the present of ketone in the urine. A1C test is also helpful to establish the diagnosis of T1DM. C-peptide level is a marker of beta cell function, C-peptide levels below 0.2 nmol/L indicates a diagnosis of T1DM [2].

There are several treatments for DKA. First one is replacement of dehydration. Dehydration should be assumed of 5-10%. In the first 1-2 hours, 10-20 ml/kg of 0,9% NaCl should be given to restore circulation. Maintenance fluid therapy should be calculated with 1000 mL for the first 10 kg body weight + 500 mL for the next 10 kg + 20 mL/kg over 20 kg or 1500 mL/m^2 body surface area. Insulin should be started after fluid therapy. The starting dose is 0,1 U/kg/hour with continuous pump. A bolus dose of insulin should be avoided because it could increase the risk of cerebral edema [13].

Cerebral edema is also a rare manifestation of DKA. But when it presents it could be a major risk factor for death in DKA. Risk factors for cerebral edema is pH below 7.1, decrease of consciousness, newly diagnosed T1DM, and young patients (< 5 years old). There are several diagnostic criteria for cerebral edema such as abnormal motor or verbal response to patient, posture showing decortication or decerebration, abnormal respiratory pattern. Treatment of cerebral edema could be done with IV mannitol with 0.5-1 g/kg [13].

Neurological complications of DKA occurs at the rate of 0,3-1%. Both ischemic and hemorrhagic stroke could be a rare manifestation of DKA with hemorrhagic stroke carries worse prognosis and higher morbidity. Hemorrhagic strokes could present as both parenchymal hemorrhage and SAH [3,4].

Pediatric strokes have morbidity of around 66% and mortality rate of 7-28%. There are lack of studies examining relationship between DKA and SAH. But there are several possible etiologies for this case such as vascular injury, hyperketonemic state, decreased level of glutathione, and endothelial perturbation. Vascular injury could be caused by a hypotensive episode and softening of vascular tissue. Hyperketonemic state and decreased of glutathione level caused oxidative injury.

Endothelial perturbation could be caused by increased level of proinflammatory markers. Rupture of arteriovenous malformation is also possible cause of hemorrhage [6,12]

Decrease of consciousness and other neurological deficits warrant further neuroimaging in the background of DKA. Non-contrast brain CT is a good first option. If a non-contrast brain CT show high degree of suspicion of SAH, lumbar puncture should be done [10].

Treatment of SAH could be done with close monitoring of systolic blood pressure (SBP). SBP should be maintained between 160-130 using calcium channel blockers and beta blockers. Neurosurgical consult must be done for surgical options of SAH. For children with increased intracranial pressure, patient's head must be elevated at 30 degree but no more than 40 degree. Intravenous mannitol is also a possible treatment option for elevated intracranial pressure [1,9].

CONCLUSION

Subarachnoid hemorrhage is a neurological complication that rarely happens in DKA and could be caused by several factors such as hypotensive episode, hyperketonemic state, arteriovenosus malformation rupture, and endothelial perturbation. Neuroimaging could detect early neurological complication in DKA.

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