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Southeast Asian Journal of Health Professional

Journal homepage: https://www.sajhp.com/

Case Report

A case report on diabetic ketoacidosis in 5 years old female child

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ARTICLE INFO

Article history:
Received 13-12-2024
Accepted 17-01-2024
Available online 09-03-2024

Keywords:
Diabetic ketoacidosis
Gestational diabetes
RBS
GRBS
Insulin therapy

ABSTRACT

Diabetic ketoacidosis is characterised by an insulin deficiency, metabolic acidosis and increased ketone concentration in the body which can be normalised by insulin replacement and electrolyte replacement therapy. One in three children suffers with type 1 diabetes (T1D), across the US and globally, the incidence of the disease is rising among the paediatrics by over 3% annually. The diagnosis of diabetic ketoacidosis (DKA) was based on the presence of hyperglycaemia (blood glucose > 11 mmol/L), acidosis (serum bicarbonate < 15 mmol/L) and ketonuria (urine ketone ≥1+). Gestational diabetes is a common abnormality of glucose metabolism during pregnancy which affects foetal development and an alteration in the balance of glucagon and insulin is affected due to an overproduction of glucose and ketones in the liver, with free release of fatty acids from adipose tissue. A case of 5 years old female child presented with increased urination, increased appetite and sudden weight loss since 1 month and her RBS range was found to be 426mg/dl. Child underwent laboratory investigations, GRBS monitoring every $3^{rd hourly}$ and assessed as Diabetic ketoacidosis with uncontrolled sugars. The child was treated with insulin supplementation for 3 days, recovered and child was hemodynamically stable and got discharged.

1. Case Presentation

History of Present Illness – A five years old female child consulted a paediatrician with chief complaints of increased urination at night time, increased appetite, and sudden weight loss since 1 month. H/O patient was asymptomatic since one month back and suddenly developed these symptoms. Doctor suspected it as Type-1 diabetes mellitus, he did RBS at his clinic and her RBS was found to be 426mg/dl and he sent for urine ketone bodies for analysis – report: urine ketone positive, urine sugar positive. HbA1C – 12. he diagnosed as case with type1 Diabetes with ketoacidosis with uncontrolled sugars and he referred to higher centre.

Baby admitted into the emergency department in hospital and Human actrapid insulin was started 2^{nd} hourly GRBS monitored

Table 1: Day 1

Hours	GRBS
1^{st} hour	426mg/dl
2^{nd} hour	225mg/dl
3^{rd} hour	210mg/dl
4^{th} hour	180mg/dl

IVF-NS @ 40ml/hr slow infusion and 20U HA insulin/SC were given

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1.1. Day 3

1. Patient GRBS controlled
2. Ketone bodies were negative
3. Urine sugar decreased

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https://doi.org/10.18231/j.sajhp.2024.005

Table 2: Day 2

Time	GRBS	insulin
8:00 AM	180mg/dl	6U HA
2:00 PM	226mg/dl	10U HA
8:00 PM	156mg/dl	NIL

4. Patient discharged with HA supplementation 6U(FN)-4U(AN)-6U(HS).
5. Review after 4days should maintain diabetic diet.
6. After 4 days the patient came for follow up and child was stable with controlled GRBS.

1.2. Past history

No significant past history.

1.3. Social history

No significant social history

1.4. Family history

No significant family history

1.5. Obstetric history

1. IVF conception
2. LSCS
3. Precious pregnancy at the age of 39 years
4. Mother was diagnosed as gestational diabetes during her pregnancy.

1.6. Birth history

1. Baby birth weight – 3.5kgs
2. Baby cried after delivery
3. No NICU admission.

1.7. Immunisation history

Immunization done as per schedule.

2. Discussion

Diabetic ketoacidosis is characterised by a lack of insulin, which allows the fat cell to release large amounts of fatty acids, and an excess of glucagon, which causes the liver to convert fat acids into ketoacids. Increased hepatic glucose synthesis is also a result of low insulin and excessive glucagon.[1]

2.1. Epidemiology

One in three children suffers with type 1 diabetes (T1D), across the US and globally, the incidence of the disease is rising among the paediatrics by over 3% annually. The prevalence of diabetic ketoacidosis (DKA) in children with T1D remains as high as 30%, despite increasing statistics and awareness.[2]

2.2. Clinical characteristics

The diagnosis of diabetic ketoacidosis (DKA) was based on the presence of hyperglycaemia (blood glucose >11 mmol/L), acidosis (serum bicarbonate <15 mmol/L) and ketonuria (urine ketone $\geq 1+$).[3]

2.3. Pathophysiology

Diabetic ketoacidosis is brought about by an overproduction of glucose and ketones in the liver, with free release of fatty acids from adipose tissue owing primarily to an alteration in the balance of glucagon and insulin.[4]

Gestational diabetes is a common abnormality of glucose metabolism during pregnancy which affects foetal development and leads to peripartum complications such as diabetic ketoacidosis , hemorrhage ,postpartum depression.[5]

Some of the studies states that Gestational diabetes mellitus is associated with incident diabetes in offspring during childhood and adolescence. Parents and even children and youth to consider the possibility of diabetes if offspring of a mother with gestational diabetes mellitus develop signs and symptoms such as polyuria, polydipsia, weight loss or fatigue. Future studies are needed to examine longer-term outcomes in patients with pediatric diabetes with a history of maternal gestational diabetes mellitus.[6]

2.4. Complications

Cerebral edema, aspiration of stomach contents, hypoglycemia, excessive fluid accumulation with congestive heart failure, and aspiration are among the complications associated with DKA and its management.[7]

A rare but serious adverse effect of DKA is cerebral edema. In 0.7 to 1.0 percent of children with DKA, it exists; while it can also affect adults and is more common in younger patients. Headache, drowsiness, and fatigue are some of the initial symptoms of cerebral edema. Further possible side effects include diabetes insipidus, hypertension, hyperpyrexia, and papilledema.[8]

3. Treatment

3.1. Insuin therapy[9]

Following the first fluid expansion, insulin should be administered. It provides an initial glucose level which is more accurate.

A continuous infusion of 0.1 U/kg/hour is administered via a pump. To achieve 1 unit/mL, 50 units of regular insulin needs to be diluted in 50 millilitres of normal saline.

(or)

Low doses of insulin given as continuous intravenous infusions (4 to 10 units/hr) or as hourly intramuscular injections (20 units initially, then 5 units/ hr) are as effective as large doses in treating severe ketoacidosis. [10]

3.2. Fluid therapy [9]

To restore peripheral perfusion, 10–20 mL/kg 0.9% sodium chloride (NaCl) should be given over the first 1-2 hours.

The recommended dose for maintenance is 1000 mL for the first 10 kg of body weight, 500 mL for the next 10 kg, and 20 mL/kg over 20 kg, or 1500 mL/m2 of body surface area.

K (40 mEq/L or up to 80 mEq/L as needed). The concentration of serum K rises by approximately 0.6 mEq/L for every 0.1 pH drop.

4. Conclusion

Diabetic ketoacidosis in children was very rare, in my case report the cause for juvenile diabetes was gestational diabetes. Baby was treated with insulin supplementation and recovered within one week and discharged.

5. Source of Funding

None.

6. Conflict of Interest

None.

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Cite this article: Tejaswini G, Kavya G, Bindhu MH, Chillara T. A case report on diabetic ketoacidosis in 5 years old female child. *Southeast Asian J Health Prof* 2024;7(1):22-24.