Pitfalls in the Management of Diabetic Ketoacidosis in a Hospital in Ghana

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Abstract

Diabetic Ketoacidosis (DKA) is a common acute complication among children and adolescents with type 1 diabetes in Africa. This is due to the fact that awareness about diabetes among children and adolescents is very poor in low income countries and in many cases there is little or no support for these patients. Ghana has no clinical guidelines for management of DKA and so mismanagement is likely very common. Misdiagnosis and mismanagement of DKA are worrying causes of morbidity and mortality among children with diabetes in Ghana and other resource constrained countries.

Keywords: Diabetic Ketoacidosis; High Mortality; Hypokalaemia; Type 1 Diabetes.

Introduction

Diabetic Ketoacidosis (DKA) is characterized biochemically by a triad of hyperglycaemia, ketonaemia and/or ketonuria and acidemia and clinically by polyuria, polydipsia, vomiting, abdominal pain, deep sighing respiration (Kussmaul breathing), lethargy, loss of consciousness and coma.[1,2]

The prevalence of DKA as a presenting diagnosis in children with type 1 diabetes is high in Africa. Reddy et al [3] in South Africa found out that 69.8% of patients with type 1 diabetes reported with DKA at first diagnosis. In Nigeria, Onyiriuka and Ifeibil reported in a survey that 77.1% of patients presented with DKA at diagnosis of new onset type 1 diabetes mellitus. In Tanzania, 75% of children with type 1 diabetes presented with DKA at initial diagnosis [4]. This may be reflective of poor awareness of diabetes among patients and hence late presentation. In Africa, mortality due to DKA is unacceptably high with some centers recording death rate of 26 to 29% [5-7]. This may be possibly due to the fact that many countries in Africa do not have national guidelines for management of DKA, thus leaving health practitioners with knowledge deficit in the management of diabetes in children. Management of DKA follows a specific methodical order and if they are well followed, mortality can be totally avoided or drastically reduced [8]. We report a 13 year old Ghanaian girl, known for type 1 diabetes mellitus who was referred from a private clinic with DKA to the emergency room and died during the acute phase of management due to lack of adherence to treatment protocol.

Case Report

A 13-year-old female was admitted to the emergency department with a referral diagnosis of DKA from a private clinic. She had been diagnosed as having diabetes mellitus about a year prior to presentation and was being managed at the private facility. She reported with one week history of polyuria, polydipsia, vomiting and severe abdominal pain. She was given 2 litres of normal saline and 12 units of insulin and then referred.

On examination, at the referred hospital, she weighed 35kg, conscious but tachypnoeic with a respiratory rate of 36 breaths/minute but clear chest on auscultation. Pulse rate was 120 beats/min and a blood pressure of 110/60 mmHg. Temperature was 36.2°C. The abdomen was diffusely tender and bowel sounds were present. She was fully conscious.

a) Laboratory Investigations Revealed: Blood glucose -16.7 mmol/l

b) Rapid Diagnostic Test for Malaria: (RDT) - negative

c) Urine Dipstick Test: Protein +, Glucose ++++, Ketones (Large) ++++, Leukocyte esterase -negative

d) She was Diagnosed as: DKA in a known Type 1 DM patient.
Management of the Patient and Monitoring

Intravenous cannula was inserted and blood samples were taken for blood culture, blood smear for malaria parasites, urea and creatinine, and urine for culture and sensitivity.

She was given 1 liter of normal saline within the first hour of management and reviewed about one and half hours after starting iv fluids. Laboratory results then were: Random blood sugar = 13.9 mmol/L, Urine ketones= ++++,

Electrolytes

Potassium = 4.9 mmol/L, Sodium= 137 mmol/L, Chloride= 110 mmol/L Urea = 2.54mmol/l, Creatinine = 42µmol/l, BUN/Cr = 28.3 (normal) Blood smear for malaria was negative Urine and blood cultures revealed no bacterial growth after three and seven days of incubation respectively.

The intravenous fluid was changed from normal saline to 5% dextrose saline and regular insulin was set up in a perfuser at a rate of 3.5units/hour. In about 30 minutes she was given 500mls of dextrose saline.

Subsequent Review

Her conscious level had decreased to Glasgow coma score of 9/15, temperature 37.1oC, pulse 69/min regular. No alarm was raised and IV dextrose and regular insulin were continued. Table 1 Electrolytes were not checked again and potassium was not given throughout the treatment.

| Time     | Fluid    | RBS (mmol/L) | Urine out put (mls) |
|----------|----------|--------------|---------------------|
| 640 am   | 500ml DS |              |                     |
| 805 am   | 500 ml NS|              |                     |
| 8.20 am  | 500ml D/S| 15.2         |                     |
| 10.20 am |          | 14.8         |                     |
| 11.20 am |          | 13.9         |                     |
| 11.40 am | 500 ml D/S|            |                     |
| 1.20 pm  |          | 17.9         | 1230 mls            |
| 1.35 pm  |          |              |                     |
| 2.20 pm  |          | 15.9         |                     |
| 4.23 pm  |          | 15.7         |                     |
| 6.00 pm  |          | 14.9         |                     |
| 8.10 pm  |          | 14.1         |                     |

Table 1: Reviews and Monitoring with Interventions.
Patient died 14 hours after admission whilst still on insulin and N/S.

Discussion

There is no organized data on DKA among children in Ghana. However, Onyiriuka et al [1] in Nigeria found out that 77.1% of children with diabetes presented with DKA at diagnosis while Reddy et al [3] found 69.8% of DKA at diagnosis in South Africa. Also in Tanzania, 75% of children presented with DKA at initial diagnosis [4]. This is due to lack of awareness of diabetes among patients and hence presenting late to hospital in DKA. Access to supplies such as insulin, meters, strips etc needed for effective management of diabetes are not directly provided by the Ghana National Health Insurance and so the cost of care is burdensome to the families of children and adolescents with diabetes [9]. This trend cuts across many African countries. Lack of pediatric endocrinologists in Africa leaves gap in the management of diabetes among children. Ghana has only two pediatric endocrinologists catering for about fifteen million children and adolescents in the country.

The principles of management of a child or an adolescent with diabetes in DKA include:[8]

1. Correction of shock, if present
2. Correction of dehydration
3. Correction of hyperglycemia
4. Correction of deficits in electrolytes
5. Correction of acidosis
6. Treatment of infection, if present
7. Treatment of complications

Management of DKA include fluid resuscitation, insulin administration and correction of electrolytes imbalance, more importantly potassium as well as looking for the precipitating factors and any complication and managing them [8,10]. The order of management is important. It is always fluid resuscitation before insulin management. Adequate fluid resuscitation, given cautiously, restores hemostasis and improves central and peripheral perfusion [2,10,11]. Therefore, a history of pre-referral fluid management was important in the management of this patient irrespective of her hydration status and conscious level [11]. This patient had been seen at a different hospital and given 2 liters of normal saline before referral. This should have been taken into consideration in the fluid management of the patient. However, within two hours of admission the patient had been given 1.5 liters of fluid. This was far in excesses of her fluid requirement. On review, she was found to have decreased conscious level but no notice was made by the attending doctors. At this point the patient may have developed cerebral edema yet it was not realized and diagnosed and so was not managed as such. Besides, throughout the management the patient was not given potassium. Insulin administration causes intracellular shift of potassium causing hypokalaemia [12]. After initial electrolyte investigation that was done before the insulin administration, blood electrolytes were not done again throughout the management. It is, therefore, likely that the patient developed hypokalaemia which compounded the possible cerebral edema. Heart and skeletal muscles are vulnerable to hypokalaemia. Hypokalaemia can cause cardiac arrhythmias including ventricular fibrillation, as well as muscle weakness, cramps and finally pa-
ralysis and death [12,13]. Any patient receiving continuous intravenous soluble insulin risks hypoglycaemia and hypokalaemia. In this patient attention was paid only to hypoglycaemia, which was prevented with 5% dextrose saline. However, prevention of hypokalaemia did not receive the requisite attention. Potassium chloride was readily available in the hospital and was even covered by the National Health Insurance Scheme. We think that the probable cause of death was cerebral oedema and hypokalaemia, which could have been prevented if due diligence was paid to treatment guidelines and emerging danger signs.

Conclusion

Adherent to protocol for DKA management is important.

Recommendation

Continuous professional development in diabetes and DKA for all health workers is important, as well as the development of national protocols for the management of DKA in children and adolescents.

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References

1. Onyiriuka AN, Ifeji E (2013) Ketoadicosis at diagnosis of type 1 diabetes in children and adolescents: frequency and clinical characteristics. J Diabetes Metab Disord 12:47-51.
2. Rosenbloom AL (2010) The management of diabetic ketoacidosis in children. Diabetes Ther1:103-120.
3. Reddy Y, Ganie Y, Pillay K (2013) Characteristics of children presenting with newly diagnosed type 1 diabetes. S Afr J CH 7:46-48.
4. Majaliwa ES, Munubhi E, Ramaitya K, et al. (2007) Survey on acute and chronic complications in children and adolescents with type 1 diabetes at Muhimbili National Hospital in Dar es Salaam, Tanzania. Diabetes Care 10:2187-2192.
5. Azevedo M, Alla S (2008) Diabetes in Sub-Saharan Africa: Kenya, Mali, Mozambique, Nigeria, South Africa and Zambia. Int J Diabetes Dev Ctries28:101-108.
6. Murunga AN, Owira PMO (2013) Diabetic ketoacidosis: an overlooked child killer in sub-Saharan Africa? Trop Med Int Health. 18:1357-1364.
7. Rwiza HT, Swai ABM, McLarty DG (1986) Failure to diagnose diabetic ketoacidosis in Tanzania.Wiley Online 3:181-184.
8. Brink SJ, Lee WRW, Pillay K, Kleinebreil L (2011) Diabetes in Children and adolescents. Tunesia: Britt Friis Graphic Design 51-58.
9. Ogle GD, Kim H, Middlehurst AC, Sillink M, Jenkins AJ (2016) Financial costs for families of children with Type 1 diabetes in lower-income countries. Diabet Med33.
10. Abbas E, Kitabchi D, Barry W (1999) Management of Diabetic Ketoacidosis. Am Fam Physician. 60:455-464.
11. Wolfsdorf J, Glaser N, Sperling MA (2006) Diabetic Ketoacidosis in Infants, Children, and Adolescents A consensus statement from the American Diabetes Association. Diabetes Care 29:1150-1159.
12. Kjeldsen K (2010) Hypokalemia and sudden cardiac death. ExpClinCardiol15:e96-e99.
13. Carlotti AP de CP, St George-Hyslop C, Bohn D, Halperin ML (2013) Hypokalemia during treatment of diabetic ketoacidosis: clinical evidence for an aldosterone-like action of insulin. J Pediatr163:207-212.