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CASE REPORT

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The obstacles of diagnosing and achieving glycaemic control in breastfeeding infant with type 1 diabetes mellitus with diabetic ketoacidosis- a case report

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Abstract: Diabetes mellitus (DM) with diabetic ketoacidosis (DKA) is uncommon in infancy and its presentation can mimic some common childhood illnesses. The spontaneity of the young infant, inherent communication difficulties, insulin treatment and eating pattern poses major technical, medical and psychological challenges. Training the family to care for the diabetic infant is also challenging for all. We present a case of a 10-month old diabetic infant with poor glycaemic control and DKA. The justification for this is because DM with DKA is uncommon in infancy and its presentation may be mistaken for other illnesses such as meningitis, encephalitis and cerebral malaria among others, so it needs a high index of suspicion in other to avoid morbidity and mortality. The objective of reporting this case is to create awareness that although DM is uncommon in infancy, but can still occur. It should therefore be looked for.

Key Words: Infant, Diabetic mellitus, Diabetic ketoacidosis

Introduction

Diabetic mellitus (DM) is a common health problem world-wide, presently becoming an epidemic.[1,2,3] It is the most common endocrine disease seen in Nigeria.[1]It is classified as type 1 (T1DM) or Insulin dependent and Type 2 (T2DM) or Non-insulin dependent diabetes mellitus.[1,2]Diabetes in children and adolescence is almost invariably type1DM.[1]Diabetes mellitus is a disorder of inappropriate hyperglycaemia with disturbance of carbohydrate, protein and fat metabolism resulting from absent or diminished insulin secretion or resistance at cellular level.[1,2] The incidence and prevalence rates of T1DM vary between countries, region and race. The incidence is rapidly increasing and shows a rapid trend towards onset at an earlier age. The prevalence is highest in Finland, 40/100,000/year and lowest in Karachi, 0.7/100,000/year. [1,2]In Africa, the prevalence is controversial. In Port Harcourt, southern Nigeria, a hospital prevalence of 1.2/1000 was reported in children aged <16 years, there is often a positive family history as well as consanguinity.[2,3] The precise aetiology is unknown but it is reported as a multifactorial with genetic, autoimmune and environmental components. Clinical manifestations of DM occur after loss of 90% of beta cell function.[1] The onset occurs predominantly in childhood, with a median age of 7 to 15 years but it may present at any age.[2,3] The peak ages of presentation are 5- 7 years and at time of puberty. The classical presentations are polyuria, nocturia or enuresis, polydipsia, polyphagia and weight loss. Lethargy which may progress to DKA, with kausmal respiration, acetone breath, diminished neurocognitive function and possible coma may be another way of presentation.[3,4]Diabetic ketoacidosis may be the first presentation of diabetes ever in some children.[1,2]The justification for this is because DM is uncommon in infancy and its presentation may be mistaken for other illness such as meningitis, encephalitis and cerebral malaria among others, so it needs a high index of suspicion in other to avoid morbidity and mortality. The objective of reporting this case is to create awareness that although DM is uncommon in infancy, but can still occur. It should therefore be looked for.

Case report

A 10-month-old girl presented to emergency paediatric unit ABUTH Zaria 2 years ago with body weakness and loss of achieved developmental milestones (sitting and crawling) for 14 days, recurrent convulsions and loss of consciousness for 10 days and inability to see for 8 days. There was no history of fever, vomiting, diarrhea or abdominal distension. No cough or swelling of the anterior fontanelle. Blood glucose level was noticed by the mother to be high (between 20 to 30mmol/l and sometimes unrecordably high) despite insulin administration for a week prior to the onset of illness. At the onset of illness, mother presented to EPU where she was admitted.

She was diagnosed with T1DM at the age of 8-months with polyuria evidenced by increased use of pampas and

polydipsia. Her pregnancy, labour and delivery were uneventful and weighed 2.5 kilogram at birth. She was on breastmilk and complementary feeds(mainly pap made from wheat and soya bean) and golden morn. Mother has reduced child s feeds from 50 to 100mls 6 to 8 times in a day to 3times in a day due to fear of hyperglycaemia. The childs Maternal grandmother and paternal grandfather have TIDM. She was admitted in several hospitals and had 4 different insulin formulations with no control of diabetes.

On examination, she was unconscious, afebrile, anicteric, not pale, not dehydrated and no pedal oedema.

Weight 9kg(94% of expected), Length 70cm (95% of expected)

Occipito-frontal circumference 43cm, Chest circumference 40cm, OFC:CC >1

Central nervous system: Unconscious, in decerebrate posture, anterior fontanelle measure 2x2 cm and normotensive, pupils are equal bilaterally and slowgishly reacting to light. She was globally hypertonic and hyperreflexic with normal fundoscopic findings

Chest: Had deep and rapid breathing with respiratory rate of 50cycles /min. Lung fields were clinically clear

Cardiovascular system: Pulse rate 108/min, moderate volume, blood pressure 70/50mmHg, apex beat not displaced, heart sounds I and II only, no murmur

Other systemic examinations were normal

Random blood glucose done was un-recordable (high)

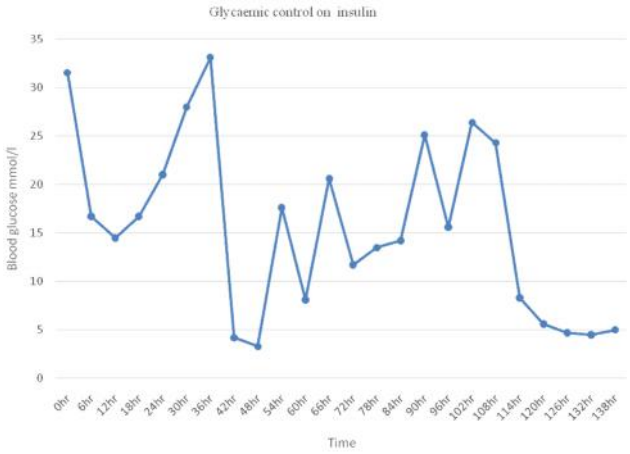
Diagnosis: Poorly controlled Diabetes Mellitus with Diabetic ketoacidosis with differentials: !) Acute Bacterial Meningitis, 2) Viral meningo-encephalitis

Investigation results available: as show in the table 1 below.

The child was commenced on multiple insulin injection regimens (soluble, neutral protamine hagedorn (NPH)), soluble insulin was initially commenced at 0.1iu/kg/hr until when the blood glucose was <250mg/dl when it was reduced to 0.05iu/kg/hr and at the end of 36hrs, it was changed to subcutaneous insulin 6hrly. Had normal saline at 20mls/kg over 1hr and the remaining deficit and maintenance was given over 36hrs using 0.18% saline in 4.3% glucose. Intravenous ceftriaxone at 100mg/kg and artesunate were also administered. Nasogastric tube feeding with unsweetened pap was also given. Mother was taught how to check for blood glucose and administer insulin injections to the child and was continued on sodium valproate. Random blood sugar was maintained between 4.7mmol/l and 11.7mmol/l. Mother had diabetic education. She was discharged and is on follow up in the endocrinology clinic.

Consent from the parents to use the childs data in this report was duly obtained.

Investigation	Result
U/E/C, done 3 times	At presentation Urea-6mmol/l, Sodium-138mmol/l, Potassium-4mmol/l, Chloride-96mmol/l, Bicarbonate-15mmol/l, Creatinine-46mmol/l 3rd day,Urea-6mmol/l,Sodium-133mmol/l,Potassium5.3mmol/l,Chloride-98mmol/l, Bicarbonate-23mmol/l, Creatinine 42mmol/l 5th day Urea-4mmol/l, Sodium-135mmol/l, Potassium-5.5mmol/l, Chloride-98mmol/l, Bicarbonate-25mmol/l. Creatinine 44mmol/l
Urinalysis	Glucose-+++, Ketones-++, Protein-trace
FBC and Differentials	WBC-5.5x10^9/l, Lymphocytes-40%, Granulocytes-56%, PCV-45%
CSF analysis	Not under excessive pressure, crystal clear, protein-30mg/dl, Glucose- 120mg/dl, Culture- no growth.
Blood culture	No growth
Brain –CT	Features of generalized cerebral atrophy



Discussion

Insulin (soluble and NPH) is being administered 6hrly to our patient. Small doses of intermediate (usually NPH) and fast acting insulin (humalog) are given twice in infants.[5,6]The major challenge is setting up a treatment regimen that is both reasonable and realistic in infants with Diabetic mellitus with ketoacidosis.[6,7] The goal of very tight metabolic control may result in episodes of severe hypoglycaemia. The therapeutic regimen must balance the naturally erratic and exercise patterns.[7] Setting a blood glucose target range of 6 to 12mmol/l usually allows this to be accomplished.[7,8] Parents of diabetic infants have concern giving insulin before they know how much their child is going to eat.[9,10] This played out in this child that was not being fed by the mother for fear of hyperglycaemia. Blood glucose monitoring is a useful tool in the management of the youngest children with diabetes.[8] The role of experienced dietitian in the care of diabetic infant cannot be over emphasized.[9,10]

Current treatment of insulin-dependent diabetes mellitus is imperfect, constantly changing and very dependent on clinical bias rather than absolute standard. Details of management are likely to differ widely between centers providing care for children with IDDM.[3,4]

Our patient had Diabetes mellitus complicated by Diabetic ketoacidosis who was managed according to four major considerations in the management of diabetes in childhood. First is to resuscitate ketoacidosis safely and avoid cerebral oedema, which largely accounts for the two folds increase in mortality in childhood diabetes compared to the general population, 2,) to avoid severe hypoglycaemia, which may lead to impaired cognitive function in early childhood, 3) to minimize the risk of long-term complications. Prevention will remain unattainable until cure or treatment that gives perfect metabolic control without the daily cooperation of the patient becomes available and 4) To ensure that the child achieves normal growth, psychosocial development and a lifestyle comparable to his or her peers.[3,4,11]

The conventional insulin treatment which is the most suitable for children relies on once- or twice-daily injections of a mixture of modified or unmodified insulins given before breakfast and before the evening meal. The neutral protamine hagedorn (NPH) insulins, in which the duration of action is prolonged by the addition of protein to the insulin molecule, are those most commonly used, and probably preferable.[4,5]

For most children who are fully insulin-dependent, a daily insulin dose of 1 unit/kg body weight is sufficient. When given twice daily the total dose is normally given in the proportion of two-thirds before breakfast and one-third before the evening meal, reflecting the meal intake during the day and night.[3,4]

Insulin requirements commonly fall after initial stabilization, and unless the administered dose is promptly and progressively reduced, hypoglycaemia will occur.[4,7,8] This phase in the natural history of diabetes can result from partial recovery of insulin secretion or improved tissue sensitivity to insulin.[4] Factors associated with this remission include age of onset (the younger the patient at diagnosis the less likely there will be remission), male sex and the presence of islet cell antibodies at diagnosis.[8] A number of efforts have been made to induce or prolong this remission phase, including the administration of steroids, azathioprine, cyclosporine, nicotinamide, combinations of these and irradiation of the pancreas.[7,11]

Conclusion

Diabetes mellitus with Diabetic ketoacidosis is uncommon in infancy and its presentation may be mistaken for other illness such as meningitis, encephalitis and cerebral malaria among others, so it needs a high index of suspicion in other to avoid morbidity and mortality. The care of a diabetic child requires expertise and proper diabetic education for the caregiver and patients. Such patients should be referred early to prevent complications.

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