Acta Biomed 2014; Vol. 85, N. 3: 285-288

Case report

Diabetic ketoacidosis in a 9 month old child and the journey to 4 years

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Summary. We report a case of diabetes mellitus in a 9-month-old male, a product of a non-consanguinous marriage with no family history of diabetes mellitus. He presented initially with ketoacidosis in our emergency room and recovered from coma after 48 hours but was discharged after 14 days. He is now 4 years old and doing well and caregivers are coping even with the challenges of caring for a very young diabetic child. This article will help to build up the data bank for diabetes in infancy and childhood in our environment as well as highlight the challenges faced by both caregivers and physicians in the management of very young diabetics irrespective of socioeconomic status.

Key words: Insulin dependent, infant, diabetes, ketoacidosis, hypoglycemia

Literature review

Diabetes mellitus (DM) is a chronic metabolic disorder caused by an absolute or relative deficiency of insulin, an anabolic hormone, produced by the beta cells of the islets of langerhans located in the pancreas. The absence, destruction, or other loss of these cells results in Diabetes mellitus type 1 (also known as type 1 diabetes, or T1DM; formerly insulin dependent diabetes or juvenile diabetes). Autoimmune destruction of insulin-producing beta cells of the pancreas and subsequent lack of insulin leads to increased blood and urine glucose. Incidence varies from 8 to 17 per 100,000 in Northern Europe and the U.S. with a high of about 35 per 100,000 in Scandinavia to a low of 1 per 100,000 in Japan and China (1).

The classical symptoms are polyuria, polydipsia, polyphagia and weight loss (2).

- The same symptoms that occur in older children and adults signaling the possibility of type 1 diabetes apply to infants and toddlers; the difference is that since their verbal communication skills are limited mostly to crying, detecting the signs and symptoms of diabetes in infants may prove difficult or not recognized quickly. However, there are some noticeable symptoms that appear frequently in, and are common to, diabetic infants: Excessive wet diapers. Diaper rash that doesn't resolve quickly or keeps recurring. Constant hunger and/or thirst, Irritability or fussiness that don't seem related to colic, Sleeping more than usual. Symptoms like fatigue are hard to discern in a baby because, they sleep a good deal of the day (3).

Two common forms of type 1 diabetes are the transient and the permanent forms. Transient DM of the newborn(TDNB) is defined as hyperglycemia occurring within the first month of life lasting at least 2 weeks and requiring insulin therapy (4). Most of these cases resolve spontaneously by 4 months. It has a reported incidence of 1 in 45,000 to 1 in 60,000 live births. Most likely etiology is a maturational delay of cAMP mediated insulin release. 30% of these children are likely to develop permanent diabetes.

Permanent DM is usually due to an interaction between the environment and genetic susceptibility. This interaction leads to the development of autoim-

mune disease directed at the insulin- producing cells of the pancreatic islet of langerhans (5,6).These cells are progressively destroyed, with insulin deficiency usually developing after the destruction of 90% of islet cells.

The role of genetics is supported by studies which show that monozygotic twins have a 60% lifetime concordance for developing T1DM while dizygotic twins have only 8% risk of concordance, which is similar to the risk among other siblings. The frequency of diabetes development in children with a mother who has diabetes is 2-3% and 5-6% for children with a father who has T1DM. The risk rises to almost 30% if both parents are diabetic. Human leukocyte antigen (HLA) class 2 molecules DR3 and DR4 carries the greatest risk of T1DM. Neonatal diabetes, including diagnosis in infants is most likely due to an inherited defect of the ikir6.2 subunit potassium channel of the islet beta cells, and genetic screening is indicated.

Environmental factors

Infections and diet are considered the two most likely environmental candidates

Viral infections- congenital rubella, enteroviral infection during pregnancy carries an increased risk of type 1 DM in the offspring.

Dietary factors are also relevant. Breast fed infants have a lower risk for T1DM (7). Nitrosamines, chemicals found in smoked foods and some water supplies, are known to cause T1DM in animal models. Other causes are streptozotocin and RH-787, a rat poison, these, selectively damage islet cells and can cause T1DM. Additional factors include congenital absence of the pancreas or islet cells, pancreatectomy, pancreatic damage, wolfram syndrome (Diabetes insipidus, dibetes mellitus, optic atrophy, deafness (DIDMOAD)), chromosomal disorders such as down syndrome, turner syndrome, klinefelter syndrome, or prader-willi syndrome.

Treatment

The five major variables in treatment are insulin dosage, diet, exercise, stress management and blood glucose and urine ketone monitoring. All must be taken into account to obtain safe and effective metabolic control. These patients do better when supportive parents continue to be involved in management of the disease.

Case report

K.C, a 9month old male presented to the children's emergency room of the Abia state university teaching hospital with a 3 day history of fever and a one day history of frequent stooling and vomitting. He was said to have been in apparent good health until onset of fever which was high grade and continous and for which he was placed on tablet artesunate, septrin, injection ceftriaxone and genticin at a private hospital. Fever was said to have subsided. However, in the early hours of the day of presentation he developed frequent watery stooling. Stool was of moderate quantity with no blood or mucus.

Patient was said to have had one episode of vomiting since onset of illness which was non projectile and non bilious consisting of recently ingested feeds. There was no associated abdominal distension. Further questioniong yielded additional history of ingestion of native herbal concoction within 24 hours preceeding admission. Volume was about 220mls given using a feeding bottle. Child was said to have been very thirsty and drank a lot of water most of the night. Further probing revealed that the average diaper change per day had been up to, at least 8 usually being heavily soaked. Also the child's food intake had been on demand which were almost on a 2 hourly basis. This consisted mostly of packaged cereals and milk but also a healthy appetite for whatever else that the family was eating.

Child is a product of non-consanguineous, monogamous marriage of a 32 year old mother and 40 year old father. Parents are both university graduates and in self employed businesses. Child is the 2nd of two children the first, a female and well. There is no family history of diabetes.

Examination findings revealed an acutely ill, severely dehydrated child with cold and clammy extremities, semi conscious with a GCS of 9/15 but lapsed into deeper coma 6/15 while being evaluated. Head circumference was 45cm and the anterior fontanelle,

which admitted only the tip of a finger, was depressed. His weight was 9 kg. Digestive system examination showed a full abdomen, liver was 3 cm enlarged and soft, spleen was not palpable. In the genitourinary system, the kidneys were not ballotable. The external genitalia showed both testes in the scrotal sac but with a penile shaft of 2.5cm (full stretched length)

Respiratory system- RR-51cpm, with good air entry. Cadiovascular system- HR-160b/m and heart sounds 1 and 11 only.

A diagnosis of enteritis with severe dehydration in shock was made. We also had a differential diagnosis of septicemia on treatment since the child had been placed on daily cephtriaxone by the previous hospital. Child had a random blood sugar test done using a glucometer and the reading was 23.8 mmol/l, bedside urinalysis using combur5 showed brick red glycosuria and 2+ of ketones.

Samples were picked up for laboratory evaluations including SEUC which showed decreased bicarbonate.

The infant had a diagnosis of diabetic ketoacidotic coma and was commenced on soluble insulin with 0.5iu/kg(1/2IV,1/2SQ), 2hrly until RBS was 14mmol/l then subcutaneous insulin was changed to 6hourly and Intravenous normal saline which was used for rapid hydration was changed to 4.3 dextrose in 0.18 normal saline. Replacement of potassium deficit was also commenced with Intravenous full strength darrows solution. Intravenous antibiotics were continued and a strict fluid chart kept.

After about 48 hrs on admission patient regained consciousness but blood sugar kept fluctuating and adjustment of feeds based on glucose levels became our major challenge. Four days following admission, with relative control of blood sugar, the insulin regimen was changed to mixtard. Blood sugar continued fluctuating, but with almost total exclusion of the packaged cereals and introduction of mashed family diet, child›s blood sugar was relatively controlled. Child was discharged after two weeks of admission, and has been followed up.

It was difficult getting his mother to accept the diagnosis but after series of counselling she tuned her mind to it and became a major resource in the management of the child. This has included blood sugar check, insulin administration, dietary management and psychological management especially when, as a toddler he throws tantrums while insisting on certain foods.

Investigations about 3 months after initial diagnosis showed low levels of C peptides with abnormal levels of glycated haemoglobin.

The child is 4 years old now and the major challenges have been with multiple dosing (3 times per day sometimes) which the mother has to go and administer in school. He is well controlled on mixtard 70/30 morning and evening and rapid acting (humulin) in the afternoon if the need arises. Recognition of hypoglycemia is also a challenge and this is a problem because the class teachers and even the school nurse find it difficult to detect this and child is not yet able to articulate this. Mother also has to cope with dietary indiscretions during children's parties but adjustment has been excellent and the child is growing well and doing well in school. He weighs 20.5 kg and standing height is 1.37meters. He has only been hospitalized once since diagnosis for confirmed severe malaria.

Discussion

This case of permanent diabetes first diagnosed with ketoacidosis at 9 months of age is one of the uncommon cases based on age but the late presentation is in keeping with previous studies (8,9) where caregivers were unable to interprete signs of the disease. The age of the patient was a major factor in the mother not detecting the possibility of a major illness. This child who was not exclusively breastfed stood a chance of succumbing to environmental insults that can trigger autoimmune responses but since further genetic testing for culprit genes have not been done, it merely stands here as speculations. The history of ingestion of oral liquid concoctions the active ingredients of which are not ascertained also leaves the possibility of poisoning by agents that selectively damage the islet cells as has been documented. The C peptide levels found to be present though low, a few months after initial diagnosis is in keeping with newly diagnosed T1DM because of the honeymoon phenomenon. This has widely been reported as part of the reasons for late presentation of young diabetics. The psychologic burden of the illness

which currently is mostly on the mother because the child is yet too young to appreciate, is in keeping with previous studies[3].The role of the physicians in terms of providing expertise and psychologic support for the parents so far, brings to the fore, the cooperation that must exist among those involved in the care of children with chronic illness

Conclusion

Since type1 diabetes mellitus in infancy is a real entity even in our environment, a call is made for care givers and physicians to be on the look out for the symptoms specific for very young children as well as checking blood glucose levels in ill children. Physicians are also called upon to play active roles to support families of children with chronic illnesses to lighten their journey on the long walk to adulthood.

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Received: 7 April 2014
Accepted: 18 July 2014
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