DIABETIC KETOACIDOSIS DURING INFANCY: TWO CASE REPORTS

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INTRODUCTION

Diabetes mellitus is a syndrome of disturbed energy homeostasis caused by a deficiency of insulin or of its action and resulting in abnormal metabolism of carbohydrate, protein & fat. It is the most common endocrine metabolic disorder of childhood & adolescence with important consequences on physical & emotional development. Morbidity & mortality stem from metabolic derangements and from long term complications that affect small & large vessels.

With few exceptions, Diabetes in children is due to Type I Diabetes Mellitus (Insulin dependent DM) and it is characterized by severe insulinopenia and dependence on exogenous insulin to prevent ketosis and preserve life. Diabetic ketoacidosis (DKA) is responsible for the initial presentation of many (approximately 25%) diabetic children (1). The incidences of type 1 diabetes mellitus is rapidly increasing and show a trend toward earlier age onset. The incidence is highly variable among different ethnic groups. The overall age-adjusted incidence varies from 0.7/100,000 per year in Karachi to about 40/100,000 per year in Finland (2). Even though diabetes during infancy is not unusual the clinical presentation in these cases and the early age at onset necessitates a high index of suspicion to diagnose them and salvage their life.

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Case 1

This is an 11 and ½ months old male child from Addis Ababa who was brought by his parents with the major complaint of high grade fever and persistent vomiting of ingested material of 1 day duration. Otherwise she had no cough or grunting, no diarrhea, skin lesions, yellowish discoloration of the eye or any urine color change. She does not have any recent travel to a malarious area. No family history of diabetes mellitus.

On physical examination (P/E) the patient was sick looking with pulse rate (PR) of 136beats/min and respiratory rate (RR) of 48/min. Temperature was 38.7°C. No apparent sings of dehydration. The only impressive finding was on central nervous system examination which revealed irritability with constant cry. In this infant as there was no focus for the fever and as she was irritable with constant cry the first thing considered was meningitis. With this impression lumbar puncture (LP) was done and the diagnosis of DKA was suggested by an elevated cerebrospinal fluid (CSF) glucose level which was 369 mg/dl. Then the random blood sugar (RBS) was determined and it was > 450 mg/dl.

Case 2

This is an 11 month old female child from Addis Ababa who was brought to a Hospital by her father with a two days history of high grade intermittent fever and repeated episodes of vomiting of non bilious, ingested material. She also had runny nose. Otherwise she had no cough or grunting, no diarrhea, skin lesions, yellowish discoloration of the eye or any urine color change. She lives in non malarious area and she does not have any recent travel to a malarious area. For the above complaint she was taken to two other clinics where she was given Amoxicillin in the first clinic & ceftriaxone in the second one despite which her condition worsened and she became progressively weak and later non- communicating. She had no history of abnormal body movement, trauma to the head or prior history of toxin ingestion or drug intake apart from those mentioned above. No family history of Diabetes mellitus. She was fully immunized according to the EPI schedule.

On P/E she was acutely sick looking, with temperature of 36^0C PR= 136/min weak RR=54/min Weight = 7.8 kg

Sunken eyeballs and had whitish coat over the tongue. No bulging fontanel. Tympanic membrane and tonsils were normal. On chest examination she had intercostals retractions but clear to auscultation. She was tachycardic otherwise abdominal and genitourinary examinations were non- revealing except abdominal skin pinch which goes back slowly. On CNS examination she had spontaneous eye opening but not responding to painful stimuli. Pupils were bilaterally equal & reactive to light.

With the clinical impression of DKA to rule out meningitis the following investigations were done.

RBS was greater than 450 mg/dl.

Urinalysis revealed Glucose ++, Ketone ++, pus cells 2-3.

CSF analysis showed no cells, no gram stainable organism but the glucose was 388 mg/dl. Blood film for hem parasites was negative. The white blood cell count was 20,700/mm^3 with differential of lymphocyte 59% & neutrophils 41%.

Chest X-ray was normal.

Both cases were admitted to Tikur Anbessa hospital with the diagnosis of DKA and managed with fluids, electrolytes and insulin therapy and sent home after stabilization of their condition. Currently they are on Regular & Lente insulin on twice daily bases.

DISCUSSION

Diabetes Mellitus is reported to occur starting from the neonatal age group (2). It can occur as a transient or permanent phenomenon. But the onset of persistent Type I Diabetes Mellitus before the age of 6 months is most unusual. When Type I DM occurs during infancy there is an increased incidence of Diabetic ketoacidosis (DKA) at first presentation. DKA is a state of severe metabolic decompensation which is manifested by over production of ketone bodies & ketoacids resulting in metabolic acidosis, usually accompanied by hyperglycemia. In any child DKA should be considered if he/she presents with new onset of the following symptoms: abdominal pain, vomiting, dehydration, Kussmaul respiration or altered mental status.

The early manifestations may be relatively mild and consist of vomiting, polyuria & dehydration. In more prolonged and severe cases deep & fast breathing (Kussmaul respiration) are present. Abdominal pain or rigidity may be present and may mimic Appendicitis or pancreatitis. With disease progression cerebral obtundation and ultimately coma ensue. Laboratory findings include glucosuria, ketonuria, hyperglycemia, ketonemia & metabolic acidosis. Leukocytosis is common. Fever in a subject with DKA warrants evaluation and treatment for possible underlying infection. Blood, Urine & Throat cultures and Chest X- Ray films should be obtained. Lumbar puncture is performed only if meningitis is suspected and even then should be done with caution because intracranial pressure is often increased.

The management of Type I DM may be divided into three phases depending on the initial presentation: that of ketoacidosis; the post acidotic or transition period for establishment of metabolic control; and the continuing phase of guidance of the diabetic child and his or her family. In DKA the immediate aims of therapy are expansion of the intravascular volume, correction of deficits in fluids, electrolytes, and acid-base status, and initiation of insulin therapy to correct intermediary metabolism. The major life threatening complication in children treated for DKA is cerebral edema.

Cerebral edema remains a leading cause of death in diabetic children, accounting for ≈ 31% of deaths associated with DKA (3).

Prompt recognition of the condition as it evolves, and prompt therapy with mannitol and hyperventilation can be life saving.

In infancy during the transition period and subsequently management is complicated by the difficulty in administering small doses of insulin, monitoring blood glucose, complementing insulin administration with feedings, and hypoglycemia. The potential for brain damage with unrecognized episodes of hypoglycemia is always a concern in infants. The most important factors in the management of hypoglycemia are an understanding by the patient and family of the symptoms and sign of the reaction, especially of the patient's individual pattern, and avoidance of known precipitating factors. Patients with diabetes presenting at 6-24 months might be associated with a different clinical pattern and higher rate of celiac disease than diabetes presenting later in life (4). Therefore during follow-up the infant should be evaluated for such occurrence.

The physician should be aware of the psychosocial issues involving the family of an infant with diabetes. Optimism and ongoing support should be provided to the family, so that the infant can grow up healthy.

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