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# DIABETES MELLITUS IN AN INFANT: AN UNUSUAL PRESENTATION

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## INTRODUCTION

Diabetes mellitus (DM) predominantly Type 1 is the most common endocrine disorder seen in children[1,2]. However, it is an uncommon problem in infancy. It occurs in all age groups, with peak ages of presentation at 5-7 and 12-15 years.[1,3,4]. The age of onset has been noted to be later in black Africans than Caucasians. In Africa, the true prevalence is controversial, although, studies have shown increasing incidence[2,3]. A hospital prevalence of 1.2/1,000 patients was recorded in Nigeria.[4] DM is extremely rare in infancy.[1,2] We present a case of type 1 DM with an unusual presentation.

## CASH REPORT

A 10 month old, male infant presented with a two-week history of intermittent fever, cough and convulsion. One episode of convulsion had occurred in the present illness. This was followed by inability to move both upper limbs and to sit nor stand. He had a previous history of seizures at 7 months of age, and was admitted in a private hospital for one week. There was no family history of febrile seizures. He was delivered by emergency caesarean section for severe pre ecclampsia at term, with a birth weight of 4.5kg. The mother was not a known diabetic. He cried immediately after delivery. He had septicaemia and possible disseminated intravascular coagulopathy (DIC) on the second day of life, and was treated in the special care baby unit (SCBU) with ceftriaxone (50mg/kg/day) and gentamycin (5mg/kg/day) for 2 weeks and an exchange blood transfusion (EBT). He was not breastfed exclusively; had artificial milk and cereal started at 1 and 6 months of life respectively. He had a normal neurological development prior to the present illness. He's the first child of a married couple from a low social class. They had no family history of DM.

On examination he was well nourished (weight 9.3kg), febrile, mildly pale, dyspnoeic, conscious but irritable. He had left hemiparesis. His blood pressure was normal (90mmHg systolic). There was mild tachypnoea (48breaths/min), but normal chest auscultation. Possible meningitis was suspected as well as malaria. Full blood count was normal but the blood film showed the presence of plasmodium falciparum. Initial blood glucose (BG) estimation done during CSF collection was 28mmol/L. He was admitted and treatment was started with intravenous Ceftriaxone (100mg/kg) and Gentamycin (5mg/kg). When the cerebrospinal fluid analysis result obtained 2days later did not confirm meningitis, the antibiotics were discontinued.

He received intramuscular Artesunate 3.2mg/kg on day 1, then 1.6mg/kg twice daily for 2 more days. On the third day of admission he became increasingly dyspnoeic, tachypnoeic and dehydrated with acidotic breathing. Further history revealed that he had been passing large volumes of urine as the diapers were changed more frequently and he was drinking a lot of water in the preceding few days. Urinalysis showed 4+glucose, 4+ketones, 1+nitrite, and no blood. Urgent BG done was 30mmol/l. He was very acidotic (bicarbonate 8mmol/L), with normal potassium, urea and creatinine. Emergency management of diabetic ketoacidosis (DKA) was commenced, using intravenous normal saline (20mls/kg) and soluble insulin 0.1 unit/kg stat (Humulin) and hourly for six hours. Dehydration was corrected over 36hours using 1.8L of intravenous fluid, initially normal saline until BG fell lower than 16.7mmol/L, then 4.3%Dextrose/1/5 saline was replaced.

Subcutaneous insulin was then commenced at a dose of 0.2units/kg 6hourly. His blood glucose was estimated hourly initially, and later 4hourly when he was out of DKA. The level fluctuated between 20.6 and 10.6mmol/L for the next 5 days, and the insulin dose was increased to 2units/8hourly in order to achieve a blood glucose level of 6.3mmol/L. By the 7$^{th}$ day, intermediate acting insulin was introduced. He was receiving 2/3 of total insulin requirements as intermediate acting, and 1/3 as short acting, given twice daily. The parents were taught how to inject the insulin subcutaneously, do urine testing and monitor BG using a glucometer. They were also counselled on hypoglycaemic symptoms. Other details of management included regular physiotherapy and nutritional advice by the dietician. The patient remained normoglycaemic for one week and was discharged home after 3 weeks of hospitalization.

He presented to the clinic a week later without complaints except for fluctuating levels of BG, between 3.8-13.3mmol/L. His glycosylated haemoglobin was 6 percent (normal). By the next weekly visit, he had fever and vomiting for 2 days and was observed to be very weak and hypoglycaemic

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(blood glucose < 1 mmol/L). He was readmitted for glycaemic control and was treated for malaria with quinine. His BG improved with I.V infusion of glucose/saline. He was discharged after one week of admission, to be followed in the out patient clinic at weekly interval.

## DISCUSSION

To our knowledge, this is the first time that such a case is being reported in our country. The rarity of a literature search on DM in infants may be due to high mortality in undiagnosed cases.[2] Type 1 DM is characterized by autoimmune destruction of pancreatic islet B cells.[1] Although a non-immune mediated destruction of islet B cells may occur in some children of African or Asian origin, both genetic susceptibility and environmental factors contribute to the pathogenesis of type IDM. The effects of stress, infection, and glucose infusion in the pathogenesis of hyperglycaemia have been documented, but they are usually transient.[1,2] Also, early introduction of cow milk and cereals has been noted to trigger an autoimmune process leading to the destruction of the islet cells[1,5,6]. Our patient had stressful conditions in-utero due to maternal pre eclampsia, EBT and infection in the neonatal period. He was also introduced to artificial feeds early in life. However, the role of infant feeding in the pathogenesis of DM is still controversial.[7]

The diagnosis of DM is usually straightforward, although in young children the symptoms may be non-specific as in this case. The frequent seizures experienced by our patient led to the suspicion of meningitis and cerebral malaria. DM is rare in infants and is usually not a differential diagnosis of seizures in children.

Clinical features of DM develop when approximately 80% of insulin secretory reserve has been destroyed, and the speed varies among individuals.[1] Higher titers of spontaneous auto insulin antibodies and islet cell antibodies are characteristic of the more active islet cell destruction seen in the younger patient and may prove useful in predicting evolving diabetes. Unfortunately, facilities for these investigations are not available in developing countries. Achieving adequate metabolic control with stable glycaemia in preschool children with DM is difficult[8] In addition; hypoglycaemic episodes tend to be frequent and severe in this age group. Specific glucose targets are provided for this age group: premeal levels of 6-12 mmol/L (110-220 mg/ dl) with bedtime levels above 8mmol/L (140 mg/ dl).[8] The large variations in the glucose response to small changes in insulin doses and high insulin sensitivity common in this age group account for the hypoglycaemia seen.

Children in malaria endemic areas may have additional risk of hypoglycaemia from vomiting and diarrhoea due to malaria infections as seen in the present case.[9] Efforts should be made to avoid frequent hypoglycaemic episodes which can cause subtle cognitive deficits during adolescence.[1,9]

The mortality rate of type 1 DM is very high[2-4]. In developing countries, factors such as ignorance, failure of diagnosis, poverty, belief in traditional medicine and poor health facilities are responsible for early demise of these patients[2-4,10]. We are worried about the future of this infant, considering his low socio-economic background. This case confirms that type IDM can occur in infants in our environment.

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