

Complications and Glycaemic Control of Type 1 Diabetes Mellitus amongst Children Aged 5 to 19 Years Attending Diabetic Clinic at Kamuzu Central Hospital In Malawi - Amos Msekandiana - ¹Paediatrician and Pediatric Endocrinologist, Kamuzu Central Hospital, Malawi

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Abstract

Background

Diabetes mellitus type 1 (T1DM) is a disease of public health importance in Africa affecting mostly children and adolescents. Currently, there is insufficiency of impeccable epidemiological data from sub-Saharan Africa on T1DM in children and adolescents. According to WHO report published on their website on 1st June 2018, non-communicable diseases have become an issue of global concern with about 41 million deaths per year and 1.6 million deaths occurring as a results of Diabetes alone. In addition, over 85% of the deaths occur in the low and middle income countries. Thus the aim is to reduce premature deaths from NCDs globally by one third by 2030 [1]. As a matter of fact, Diabetes type 1 (T1DM) is becoming a disease of public health importance in Africa affecting mostly children and teenagers. Currently there is insufficiency of impeccable epidemiological data from sub-Saharan Africa on T1DM in children and adolescents. However, incidence in Tanzania was estimated to be 1.5/100,000, and an increase in incidence in Sudan from 9.5/100,000 in 1991 to 10.3/100,000 in 1995 has been reported [2]. Furthermore, there is no published data seen from Malawi to reflect the burden of T1DM in children and adolescents.

Diabetes type 1 (T1DM) is an autoimmune disease which results from cellular-mediated destruction of the beta cells of the pancreas characterized by deficient insulin production and requires daily administration of insulin [3]. Children with T1DM usually may classically present with symptoms such as polyuria, polydipsia, polyphagia and weight loss. However, in about 19% of younger children below 5 years parents or guardians may report non-classical symptoms such as

nocturnal enuresis, 10.5% of the under-five may also present with constipation and 25% of the children inclusively, may present with diabetic ketoacidosis [4]. The latter is a common observation in our clinical practice in Malawi due to delay in diagnosis, misdiagnosis due to lack of knowledge in the subject or even loss of opportunity to diagnose T1DM due to poor health seeking behaviour.

The pathogenesis of T1DM is quite complex with an interplay of genetic, epigenetic, immunologic and environmental factors. As a matter of fact, Type 1 diabetes is mostly present in individuals with no family history. For example, only 10-15% of the patients have a first- or second-degree relative with the disease. However, the lifetime risk for developing T1DM is significantly increased in relatives of patients, as about 6% of children, 5% of siblings and 50% of monozygotic twins present with the disease compared to 0.4% prevalence of the general population. Additionally, there are more than 50 T1DM loci which have been identified by genome-wide association studies and meta-analyses [5]. The human leukocyte antigen (HLA) gene located on the major histocompatibility complex (MHC), on chromosome 6 is the main gene which predisposes to T1DM. Furthermore, many other different genetic loci have been found to contribute in a lesser degree to the genetic susceptibility for T1DM alone or in combination with other autoimmune diseases

Methods

This was a hospital based cross-sectional study. A total of 41 children and adolescents aged 5 to 19 years with ≥ 6 months duration of disease and on treatment were enrolled. Standardised questionnaire, medical files and health passport books were used to collect social demographic and clinical information on episodes of severe hypoglycaemia and DKA. Albumin to creatinine ratio was done to screen for

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microalbuminuria and HBA1C was done to determine glycaemic control. Slit lamp ophthalmoscopy was done to screen for diabetic retinopathy. Chi square test was used to test for associations and $p \leq 0.05$ was considered statistically significant. Spearman's correlation coefficient was used to measure the correlation between complications of T1DM and HBA1c.

Results

In our study the overall proportion of DKA and severe hypoglycaemia was 63.4% and 56% respectively. Only 4.88% had microalbuminuria, none had diabetic retinopathy and 97.56% had poor glycaemic control. DKA had a statistical significant relationship with mode of insulin storage ($\chi^2 = 6.477$, $p\text{-value} = 0.0039$) and there was a positive correlation between DKA and HBA1c. The mean duration of diagnosis of T1DM was 3.2 years (SD = 2.6). The study participants had been diagnosed with T1DM between the periods of 6 months and 10 years prior to the study with a majority belonging to the period between 1.1 years to 3 years (36.59%, $n = 15$). Additionally, 19.51% ($n = 8$) had duration of disease of 5 years (Table 1). Family history of DM was elicited in 24.39% ($n = 10$) of the cases. The commonest means of storage of insulin was traditional methods (storing insulin in a clay pot with sand and water) in 75.61% ($n = 31$) of the cases (Table 1). Good compliance to insulin administration as defined by having a participant not missing a dose in the past three months before the interview was reported in 80.49% ($n = 33$) of the cases. Inspection of the injection sites showed that 82.93% ($n = 34$) had good injection sites. Additionally, the mean body mass index of the participants was 17.35 kilogram per meter squared (STD = 2.56). Tanners staging for pubertal assessment was done and majority of the patients were Tanners stage 1 (53.66%, $n = 22$).

The symptoms that led to the diagnosis of T1DM included: Recurrent infections in 46.34% ($n = 19$), weight loss in 85.37% ($n = 35$), coma in 36.59% ($n = 15$), convulsions in 21.95% ($n = 9$), polyuria in 95.12% ($n = 39$), polydipsia in 95.12% ($n = 39$), enuresis in 46.34% ($n = 19$) and abdominal pain in 29.27% ($n = 12$).

Conclusion

The proportion of DKA and severe hypoglycemia was high. Additionally, there was a very high proportion of patients with poor glycemic control.

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