Survey on Acute and Chronic Complications in Children and Adolescents with Type 1 Diabetes at Muhimbili National Hospital in Dar es Salaam, Tanzania

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Running Title: Complications of Type 1 Diabetes in Children and Adolescents

Abbreviations: DKA, diabetic ketoacidosis
DCCT, Diabetes Control and Complications Trial

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ABSTRACT
OBJECTIVE-To assess glycaemic control and complications of type 1 diabetes in children and adolescents in Tanzania.
RESEARCH DESIGN AND METHODS-This demographic and clinical survey, included 99 children attending Muhimbili National Hospital Clinic for Diabetes, aged between 5 and 18 years. A structured questionnaire was used for evaluating socio-economic data and for estimation of the prevalence of acute complications occurring over the last 6 months. Prevalence of retinopathy was determined by fundus ophthalmoscopy and diabetic nephropathy by microalbuminuria.
RESULTS-All these children were treated with conventional insulin regimen mean duration of diabetes was 4.76±3.58 years. Only one child (1%) had good glycaemic control (HbA1c <7.5%), 60 children (60.6%) had moderate glycaemic control (HbA1c 7.5-10%), 14 (14.1%) had poor glycaemic control (HbA1c >10-12.5%) and 24 (24.2%) had very poor glycaemic control (HbA1c >12.5%). At onset of diabetes 75% of children presented with diabetic ketoacidosis (DKA). Eighty-nine children (89.80%) had at least one episode of DKA and 55 children (55.67%) presented symptomatic hypoglycemic episodes. Microalbuminuria was present in 29 (29.3%) and retinopathy in 22 (22.68%) patients.
CONCLUSIONS-Although there are some methodological limitations, this survey highlights the difficulties of getting good metabolic control and the high prevalence of acute and chronic complications in Tanzanian children with type 1 diabetes. These results clearly show that major efforts are needed to improve quality of care in children with type 1 diabetes in Tanzania.
INTRODUCTION
Type 1 diabetes is one of the most frequent chronic disease in children and represents a public health challenge globally. Its burden is huge in developing countries due to lack of basic means for reaching a reasonable glycaemic control. Due to unavailability of reliable epidemiological data, the natural history of type 1 diabetes, including its complications is largely unknown (1). The few data available on Sub-Saharan African children estimated an incidence of 1.5/100,000 in Tanzania (2), and an increase in incidence from 9.5/100,000 in 1991 to 10.3/100,000 in 1995 in Sudan, has been reported (3). The prevalence is higher in western countries (4,5), suggesting the possibility of missed diagnosis in Sub-Saharan Africa. In fact the problem of missed diagnosis of childhood diabetes, though not unique to developing countries (6), is certainly much more common (7). In a Sudanese study it was reported that 10% of children were not admitted at the time of diagnosis, being admitted only after they develop diabetic ketoacidosis (DKA) or hypoglycemia (3). This contributes to omission of patients in the registry as well as the possibilities of death before diagnosis especially for those below the age of five years. In Sub-Saharan Africa, most children present with DKA at the time of diagnosis (8,9), which could easily be overlooked as cerebral malaria or meningitis in a busy emergency reception of most hospitals in Africa (7). Poor facilities in most of these countries may as well contribute to death before diagnosis. The precipitating factors for DKA in Sub-Saharan Africa are mainly newly diagnosed diabetes, missed insulin and infections (10). In the developed world enormous efforts are made to reduce the chronic complications of diabetes, yet in developing world the incidence of these complications in children is not known, making their management more difficult.

Information on chronic complications in Sub-Saharan Africa is scarce; however, the incidence has gone hand in hand with the growing disease prevalence, demonstrating the importance of assessing complications. The few studies on chronic complications of diabetes in Sub-Saharan Africa included type 1 and 2 adult diabetic patients (11,12). The only study evaluating both adult and children reported a prevalence of retinopathy to be 14% and nephropathy to be 7.5% (13). Since there have been no data to date on the complications of type 1 diabetes in children in Tanzania, we performed a hospital based survey to evaluate glycaemic control and complications of type 1 diabetes in children and adolescents.

RESEARCH DESIGN AND METHODS
This cross-sectional survey was performed at Muhimbili National Hospital clinic for diabetes, between June 2005 and February 2006, by including children aged between 5 and 18 years, who had the diagnosis of type 1 diabetes for at least six months. A total of 99 (57 females and 42 males) out of 104 children were studied.

Ethical approval was obtained from the Research and Publicat ion Committee of Muhimbili University College of Health Sciences. Parents/guardians and children above 12 years gave their informed consent.

A structured questionnaire was used to collect demographic data from patients, parents/guardians. The variables included were age, sex, education, occupation, frequency of complications, insulin availability, accessibility, dosage and cost, clinic visits as well as the possibility of blood glucose monitoring.

Each participant was interviewed on the education of both child and parents/guardians, children reported if they were currently going to school, if not why especially if it was because of type 1 diabetes. Occupation of parents was asked as well as the sick leaves they had taken because of their child’s sickness. The age at diagnosis of diabetes and the presence of DKA at the time of diagnosis were recorded.
The frequency of DKA and hypoglycemia over the last six months were recorded. In particular, DKA was defined as the presence of ketones in the urine, a history of altered level of consciousness of the patient or coma at the time of assessment or on admission to hospital over the last six months.

Hypoglycemia was defined as the presence of three or more episodes of the following symptoms: sweating, hunger, tingling around the mouth, tremor, anxiety, weakness, headache, visual disturbances, slurred speech, vertigo and dizziness, difficulty in thinking, tiredness, drowsiness, change in affect (e.g. depressed, angry, argumentative), mental confusion, coma, and convulsion, these symptoms being relieved by giving the child sugar added foods.

Insulin availability and accessibility was assessed. Furthermore, the dosage of insulin and cost of insulin per vial was recorded. The possibility of getting insulin at a reduced cost or free was enquired as well as any assistance from relatives, employer, diabetes clinic or Non Governmental Organization. We grouped children who missed insulin over the last six months into: those who never missed insulin, missed 5 times in a month, missed 3 days in a week, missed 3 or more consecutive days. In addition, we recorded the reasons for missing insulin. Finally we enquired on the possibility of measuring blood/urine glucose at home.

There were no verifications with medical records, since most of the patients did not have past medical records. Physical measurements were made by a specially trained nurse.

**Anthropometric measurements**
The anthropometric measurements included body weight (rounded to the nearest 100 grams) and height (to the nearest 0.1 cm), which were taken on subjects light clothes without shoes. Body Mass Index was calculated according to the Quetelet equation (w/h²).

**Laboratory procedure**
Two milliliters of venous blood were taken from the anterior cubital fossa of each child, using a sterile disposable syringe and needle after a thorough cleaning of the venipuncture site with a swab soaked in 70% alcohol. One drop of blood was immediately put on the glucometer (Lifescan, Milpitas, CA, USA). The remaining blood was put in an EDTA bottle for HbA1c. The HbA1c was determined using DCA2000 Hemoglobin A1c reagent kit (Bayer diagnostic, Mulgrave, Australia).

**Urine examination for microalbuminuria**
Two milliliters of spot fresh urine were collected in an empty clean bottle. The urine was then examined by multisticks (Medi Test Combi 10 SGL) to determine the presence of proteins, ketones and sugar.

The strip was dipped into the bottle of fresh urine for 30-60 seconds; the test strip was then compared to the color scale on the bottle. The minimum sensitivity of the test strip was 10 mg/dl of protein in urine. Any urine samples that were negative for proteinuria using the sticks were re-tested by microtex for microalbuminuria (indirect latex slide test for detection of microalbuminuria). One drop of clear urine was put on the glass slide using a disposable pipette, and then one drop of anti-human albumin was added on the drop of urine on the slide. The antihuman albumin reagent and urine were mixed for 30 seconds. One drop of well mixed albumin latex reagent was added to the mixture. It was mixed uniformly over a circle. Agglutination was then observed for 3 minutes while rocking the slide back and forth. Agglutination meant a negative result, no agglutination meant positive results indicating the presence of albumin in urine (concentration above 2.5 mg/dl).
Retinal examination
The fundus ophthalmoscopy was performed by the same ophthalmologist dedicated to patients with diabetes. One drop of atropine was put in each eye and left for 10-30 minutes for the pupil to dilate. Fundoscopy was then performed to examine the optic disc, the macula and retinal vessels. Retinopathy was considered when there were diabetic retinal changes.

Statistical analysis
All values were expressed as mean ± SD. Overall prevalence of common complications was calculated using frequency distributions. Differences in sex variables and dichotomy variables were analyzed by Fisher’s exact test. The participants were divided into three age groups (i.e. 5-11.5, 11.5-15.5 and 15.5-18 years). Given the non normal distribution of the variables, differences between the three groups were tested by Kruskal-Wallis testing. Statistical significance level was \( P < 0.05 \). Differences between groups of interests were tested by Mann-Whitney testing, adjusting the level of significance by \( P < 0.0167 \). All calculations were made with the computer programme SPSS (Statistical Package for the Social Science) version 10.

RESULTS
Baseline clinical characteristics, anthropometric measurements and HbA1c levels are reported in Table 1. The female to male ratio was 1.3:1, mean age was 12.6 ± 3.5 years, mean duration of diabetes was 4.76 ± 3.58 years and mean HbA1c was 10.65 ± 2.09%. Seventy five percent of children presented with DKA at the time of diagnosis.

Children were grouped into prepubertal (5-11.5 years), pubertal (11.5-15.5 years) and post pubertal (15.5-18 years) groups. We chose these groups because children in Tanzania start puberty at the age of 11.5 years, and they are in puberty up to 15.5 years.

All children had poor glycaemic control, except one who had good control (HbA1c <7.5%). A high percentage of children (24.2%) had very poor glycaemic control (HbA1c >12.5%). However, there was no statistical difference between the HbA1c in all the three groups (\( P = 0.815 \) for moderate HbA1c, \( P = 0.141 \) for poor HbA1c and \( P = 0.394 \) for very poor HbA1c). The mean duration of diabetes was similar in all the three groups.

The height Standard Deviation Score (SDS) was low in all the age groups -2.52 ± 2.69, with even lower rates in the pubertal group -3.4 ± 2.44 (\( P = 0.026 \)).

The overall prevalence of both acute and chronic complications was high: DKA (89.9%), hypoglycemia (55.6%), retinopathy (22.2%) and microalbuminuria (29.3%). As expected no differences were found in acute complications (DKA and hypoglycemia) in all the age groups.

Surprisingly, there was already a high frequency of retinopathy in the prepubertal group (25.8%). The pubertal group had lower occurrence of retinopathy (8.6%), compared to other groups (\( P = 0.043 \)).

No significant differences were found between the three groups regarding microalbuminuria. All children were on conventional insulin regimen (0.5-2 U/Kg/day). None of the children had glucose monitoring at home. Table 2 shows the frequency of children’s missing insulin; there was no difference between the age groups (\( P = 0.172, P = 0.410 \) and \( P = 0.837 \), respectively), however there was a significant difference between those who missed insulin and chronic complications (retinopathy and microalbuminuria) (\( P = 0.004 \) and \( P = 0.040 \), respectively). No association was found between the number of insulin missed and the occurrence of acute complications (DKA and hypoglycemia) (\( P = 0.335 \) and \( P = 0.834 \), respectively).

The clinic visits were similar in all the groups, with 35.4% of the children visiting the clinic 4-6
times in six months. No association was found between the clinic visits and complications (data not shown).

Parents’ education as well as parents’ occupation was not associated with complications and glycaemic control. There was no difference in child’s education level, parents’ education and occupation, marital status, parents’ sick leaves and insulin availability as well as accessibility (data not shown). There was no relationship between prevalence of complications and duration of diabetes.

**DISCUSSION**

This is one of the few studies on complications of type 1 diabetes in children and adolescents from Sub-Saharan Africa. In this survey we documented a high prevalence of complications and poor glycaemic control reflecting a complex unfavorable social and economical environment, in which children with type 1 diabetes in Africa are living.

On the ground of DCCT’s recommendations, a high proportion of children and adolescents with type 1 diabetes receive intensive treatment in the developed countries (14). In contrast children in Tanzania received only irregular conventional insulin regimen. This determines a higher mean HbA1c (i.e.12.6 ± 3.5%) than those reported in studies performed in the developed world (14,15). However, our findings were similar to a study done in Sudan, where Elamin et al (16) found a high incidence of poor glycaemic control estimated by HbA1c. Most likely the underlying cause is the association between limited insulin supply and lack of self monitoring of blood glucose. Furthermore, in addition to limited insulin supply, patients reduce the insulin dose to ensure longer periods of insulin treatment. Nevertheless insulin storage might influence the effect of insulin itself as many families store the insulin in a pot with cold water, exposing insulin to high temperatures. Correlation reported in other studies between HbA1c and age (14,17), number of visits (18,19), and duration of diabetes (14), were not found in this study, probably due to small sample size and overall very poor glycaemic control.

It was not possible to assess the correlation between HbA1c and frequency of self-monitoring of blood glucose (18,20-23) since none of the children had glucometers or glucosticks to perform daily blood glucose measurements. More importantly, even in hospitals blood glucose measurements cannot be routinely carried out due to lack of facilities. In agreement with different studies (22) and different cultural backgrounds, pubertal girls presented higher mean HbA1c than boys. It is has been shown that linear growth might also be impaired in children even when reasonably glycaemic control had been achieved (23,24), and this growth pattern is likely to be more pronounced in a setting where metabolic control is very poor. In fact, most of our children had short stature which was more pronounced in puberty, in accordance with other studies (25,26). At variance (27,28), there was no correlation between growth and duration of diabetes or insulin requirement (18, 21) however, we cannot rule out the contribution of other well known factors like malnutrition and chronic infections.

The high prevalence of DKA observed in this survey (89.9%), although comparable to the prevalence found in Congo Brazzaville (10), might have been overestimated due to the arbitrary definition of DKA leading to a higher prevalence than that reported in the international literature (25-30%) (28). Our data reflect the estimated prevalence of DKA at diagnosis (29), but we observed much higher occurrence of DKA in children already receiving insulin supplementation. This high frequency of DKA during therapy is not likely to be affected by the arbitrary definition but reflects more likely limited insulin supply and lack of self monitoring. In fact lack of insulin and recurrent infections are the main reported precipitating factors for DKA in Sub-Saharan Africa (30). The high percentage of symptomatic hypoglycemic
episodes found in our study reflects again the poor level of glycaemic control and the lack of self monitoring. However, the true prevalence of hypoglycemia remains unknown in our study population and the percentage might be again exaggerated as our study depended solely on self/parental reporting of these episodes in absence of self monitoring of blood glucose. Some studies reported that patients with type 1 diabetes who are in chronically poor blood glucose control perceive hypoglycemia at higher levels than those in acceptable blood glucose control (31), suggesting the possibility that children in our survey might have had normal blood glucose levels when having symptoms of hypoglycemia.

Along with the high prevalence of acute complications we documented a high prevalence of background retinopathy. Our data are similarly to those of a study done in Kilimanjaro, Tanzania, by Neuhann et al (13) who found a prevalence of retinopathy of 14% in the whole type 1 diabetic population. Surprisingly, in our study we detected a higher prevalence of retinopathy in prepubertal children compared to the pubertal group. This age distribution of retinopathy is alarming as it gives the possibility that some adolescents might have died because of diabetes complications. Although, no studies are available on mortality in children and adolescent with diabetes in Africa this is likely. However, it also underlines the importance of metabolic control before puberty as young children are able to be affected by retinopathy. This is in contrast with some longitudinal studies where the duration of prepubertal diabetes seemed to have limited effect on long-term complications (32).

This study also demonstrated a high prevalence of microalbuminuria which was higher in the older children, in accordance with other studies (33). Nevertheless, we were able to detect a significant prevalence of microalbuminuria in younger children demonstrating again that poor metabolic control is able to cause long-term complications independent of the duration of diabetes (34). This is also reflected by the absence of any correlation between duration of diabetes and microalbuminuria. It is worthwhile to acknowledge that, the prevalence of microalbuminuria might have been overestimated because of the methodological limitations.

In many Sub-Saharan African countries insulin supply is erratic and the monthly cost of insulin for an average treated patient equals the 25% of the minimum wage (13). In our survey 63.3% of children missed insulin at least once over the preceding six months mainly due to the lack of funds and insulin availability. These difficulties are exacerbated by intermittent availability of supplies such as syringes, urine and blood strips and perhaps most crucially, by a small experience in the management of diabetes by most health care workers (35).

In conclusion these data clearly demonstrate that insulin scarcity, poverty and lack of adequate health care increases the incidence of acute and chronic diabetic complications in children and adolescents with type 1 diabetes in Tanzania, a problem which previously has not been fully recognized. This survey further highlights the difficulties of getting good metabolic control, implying that major efforts are needed to improve quality of care in children with type 1 diabetes in Sub-Saharan African countries. The first step is obviously to make insulin available for all children all the time.

Acknowledgments
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References


Ahmed ML, Connors MH, Drayer NM, Jones JS and Dunger DB. Pubertal growth in IDDM is determined by HbA1c levels, sex and bone age. *Diabetes Care* 21:831-835, 1998


Table 1 – Subjects characteristics

<table>
<thead>
<tr>
<th></th>
<th>Groups</th>
<th>Significant differences (P&lt;0.05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>Prepubertal</td>
</tr>
<tr>
<td>Age (years)</td>
<td>12.6±3.5</td>
<td>8.18±1.85</td>
</tr>
<tr>
<td>Sex</td>
<td>42M/57F</td>
<td>10M/21F</td>
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<tr>
<td>Height</td>
<td>136.15±21.38</td>
<td>123.94±23.68</td>
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<tr>
<td>Ht-SDS</td>
<td>-2.52±2.69</td>
<td>-2.25±3.23</td>
</tr>
<tr>
<td>Weight</td>
<td>38.19±11.32</td>
<td>32.10±12.95</td>
</tr>
<tr>
<td>BMI</td>
<td>20.04±2.15</td>
<td>19.55±2.0</td>
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<tr>
<td>BMI-SDS</td>
<td>0.82±1.38</td>
<td>2.11±1.51</td>
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<td>HbA1c &lt; 7.5%</td>
<td>1 (1%)</td>
<td>0 (0%)</td>
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<td>HbA1c 7.5-10%</td>
<td>60 (60%)</td>
<td>22 (68.8%)</td>
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<tr>
<td>HbA1c 10-12.5%</td>
<td>14 (14.1%)</td>
<td>5 (15.6%)</td>
</tr>
<tr>
<td>HbA1c &gt; 12%</td>
<td>24 (24.2%)</td>
<td>5 (15.6%)</td>
</tr>
<tr>
<td>DOD-&lt;1 year</td>
<td>10 (10.1%)</td>
<td>4 (12.2%)</td>
</tr>
<tr>
<td>DOD-1-5 years</td>
<td>51 (51.1%)</td>
<td>20 (62.5%)</td>
</tr>
<tr>
<td>DOD-&gt;5 years</td>
<td>38 (38.4%)</td>
<td>8 (25.0%)</td>
</tr>
<tr>
<td>DKA</td>
<td>89 (89.9%)</td>
<td>28 (90.3%)</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>55 (55.6%)</td>
<td>21 (9.7%)</td>
</tr>
<tr>
<td>Retinopathy</td>
<td>22 (22.2%)</td>
<td>8 (25.8%)</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>29 (29.3%)</td>
<td>9 (29.9%)</td>
</tr>
<tr>
<td>ID-0.5-0.8 U/kg</td>
<td>39 (39.4%)</td>
<td>16 (50.0%)</td>
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<tr>
<td>ID-0.8-1.2 U/kg</td>
<td>29 (29.3%)</td>
<td>9 (28.1%)</td>
</tr>
<tr>
<td>ID-&gt;1.2 U/kg</td>
<td>31 (31.3%)</td>
<td>7 (21.9%)</td>
</tr>
<tr>
<td>IA-Yes</td>
<td>57 (57.5%)</td>
<td>18 (56.3%)</td>
</tr>
<tr>
<td>IA-No</td>
<td>42 (42.4%)</td>
<td>14 (43.8%)</td>
</tr>
<tr>
<td>CV/6 Months-1-3</td>
<td>33 (33.3%)</td>
<td>11 (33.3%)</td>
</tr>
<tr>
<td>CV/6 Months-4-6</td>
<td>37 (35.4%)</td>
<td>10 (27.0%)</td>
</tr>
<tr>
<td>CV/6 Months-&gt;6</td>
<td>29 (29.0%)</td>
<td>11 (37.9%)</td>
</tr>
</tbody>
</table>

DOD-Duration of diabetes, IS-Insulin dosage, IA-Insulin available, CV/6Months-Clinic Visits done in the last 6 months, NA-Not Applicable, Ht-Height, SDS-Standard deviation scores.
Table 2 – Missed insulin by age, sex, glycaemic control and complications

<table>
<thead>
<tr>
<th>Groups</th>
<th>Never missed insulin N (%)</th>
<th>Missed insulin 5times/month N (%)</th>
<th>Missed insulin 3times/Week N (%)</th>
<th>Missed insulin ≥3 consecutive days N (%)</th>
<th>Significant differences (P≤0.05)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5-11.5 years</td>
<td>13 (35.1)</td>
<td>6 (42.9)</td>
<td>4 (21.1)</td>
<td>8 (27.6)</td>
<td>0.172</td>
</tr>
<tr>
<td>11.5-15.5 years</td>
<td>14 (37.8)</td>
<td>5 (35.7)</td>
<td>6 (34.5)</td>
<td>10 (35.4)</td>
<td>0.410</td>
</tr>
<tr>
<td>15.5-18 years</td>
<td>10 (27.0)</td>
<td>3 (21.4)</td>
<td>9 (47.4)</td>
<td>11 (37.9)</td>
<td>0.837</td>
</tr>
<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>Male/Female</td>
<td>16(43.2)/21(56.8)</td>
<td>6(42.9)/8(57.1)</td>
<td>7(36.8)/12(63.2)</td>
<td>13(44.8)/16(55.2)</td>
<td>0.956</td>
</tr>
<tr>
<td>HbA1c &lt; 7.5%</td>
<td>1 (2.8)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
<tr>
<td>HbA1c 7.5-10%</td>
<td>25 (69.4)</td>
<td>10 (71.4)</td>
<td>9 (47.4)</td>
<td>16 (53.3)</td>
<td>0.181</td>
</tr>
<tr>
<td>HbA1c 10-12.5%</td>
<td>5 (13.9)</td>
<td>2 (14.3)</td>
<td>1 (5.2)</td>
<td>6 (20.0)</td>
<td>0.804</td>
</tr>
<tr>
<td>HbA1c &gt; 12%</td>
<td>5 (13.9)</td>
<td>2 (14.3)</td>
<td>9 (47.4)</td>
<td>8 (26.7)</td>
<td>0.271</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>DKA</td>
<td>31 (83.3)</td>
<td>14 (100)</td>
<td>17 (89.5)</td>
<td>27 (93.1)</td>
<td>0.335</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>19 (51.4)</td>
<td>9 (64.3)</td>
<td>10 (52.6)</td>
<td>2 (6.9)</td>
<td>0.834</td>
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<td>Retinopathy</td>
<td>4 (10.8)</td>
<td>0 (0)</td>
<td>8 (42.1)</td>
<td>10 (34.5)</td>
<td>0.004</td>
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<td>Microalbuminuria</td>
<td>8 (21.6)</td>
<td>1 (7.1)</td>
<td>9 (47.4)</td>
<td>11 (29.3)</td>
<td>0.045</td>
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</tbody>
</table>

N-Number of patients, NA-Not applicable