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NUTRITIONAL INTERFERENCE IN THE TREATMENT OF PATIENTS WITH *DIABETES MELLITUS* TYPE 1

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ABSTRACT

The treatment of *Diabetes Mellitus* Type 1 (DM1) involves adherence to insulin treatment, diet and physical activity, aiming at glycemic control. The objective of this study was to observe the effects of nutritional interference in the treatment of patients with DM1. It is a quantitative, prospective and longitudinal study developed at the UFTM Diabetes Outpatient Clinic. Data collection was performed between March 2013 and September 2014. Thirty-one children and adolescents between 6 and 17 years old were evaluated for anthropometry, glycemic and lipid control in four stages: M1 at the beginning of follow-up; M2 after conventional nutritional counseling; M3 after learning the carbohydrate count (CCHO) and M4 in full count. Statistical analysis was descriptive and inferential. The anthropometry showed that CCHO did not result in weight gain and was effective in males, demonstrated by the reduction in the concentrations of fructosamine (p=0.050) and HbA1C (p=0.041) in M4 compared to M1. Considering the fructosamine, the CCHO group differed from the non-CCHO M4 group (p=0.035). CCHO-associated insulin therapy has been shown to be an important resource to be integrated into the treatment of DM1 to achieve effective targets in reducing complications.

Keywords: Child. Adolescent. *Diabetes Mellitus*, type 1. Dietary carbohydrates. Nutrition assessment.

INTRODUCTION

According to the International Diabetes Federation (IDF), 425 million people (20-79 years) currently live with *Diabetes Mellitus* (DM) being 90% of type 2 (DM2). The projections for 2045 (630 million) indicate the magnitude of the increasing impact of the disease on health systems in most countries, and especially in Brazil, which has 12.5 million diabetics and ranks 4th place worldwide[1,2].

The number of children and adolescents with DM also evolves at an increasing rate of 3% per year, and the IDF-2017 estimates the presence of 1,106,000 cases of DM type 1 (DM1) in people under 20 years of age in the world. Brazil occupies the third place in cases of DM1 (88,300 in people under 20 years) and in prevalence (9600 new cases/year). These data indicate the need for treatment policies and strategies that reverse such an unfavorable situation[1,2].

DM1 is characterized by insulin deficiency as a result of the immuno-mediated destruction of pancreatic β-cells, with mandatory insulin administration as a treatment associated with a food plan and exercise[3]. The study Diabetes Control and Complications Trial (DCCT) has demonstrated that adequate glycemic control in patients with DM1 delays the onset and progression of chronic complications of diabetes[4].

The targets and therapeutic results indicated by longitudinal studies such as DCCT, which used intensive insulin pump treatment, multiple doses and carbohydrate counts are difficult to replicate in clinical practice. In Brazil, according to a study by Mendes et al.[5], only 15% of type 1 diabetics had glycated hemoglobin (HbA1C) lower than 7% recommended by guidelines (American Diabetes Association - ADA and Brazilian Society of Diabetes - SBD)[3,6], indicating barriers to be overcome as reported by García-Pérez et al[7].

One of the available and proven strategies for achieving glycemic control and therapeutic targets is the use of carbohydrate counts (CCHO)[4,8], It is a method that rationalizes insulin administration, promoting the adjustments of fast-acting insulin doses before meals, based on the amount of CHO consumed, preventing large postprandial oscillations

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and reducing glycemic variability, resulting in improved control and reduction of chronic complications[8,9].

Although several authors have demonstrated that CCHO, as a nutritional approach, provides positive changes in glycemic control and in the lifestyle of children and adolescents with DM1[8,10,11], in practice, most receive conventional nutritional guidance, that is, quantitatively dissociated from the insulin doses to be given at each meal. In addition, the practice of CCHO requires that the patient, or those in charge, have a level of education appropriate to the assimilation of the guidelines and that the team that assists the patient is integrated and motivated[12,13].

In Brazil, few authors reported the effect of CCHO in the treatment of patients with DM1[9,14], especially in children and adolescents[14]. To remedy this gap, the objective of this study was to observe the effects of nutritional intervention using CCHO comparing with conventional intervention in children and adolescents with DM1 attended at the Federal University of the Triângulo Mineiro (UFTM).

METHODS

This is a prospective, longitudinal quantitative study developed at the UFTM Diabetes Outpatient Clinic and approved by the Research Ethics Committee under protocol no. 1597/2010. In the files of the outpatient clinic, 73 medical records of patients with DM1 were found in regular follow-up. After identification, during the period of waiting room where they waited for the consultation, the purpose of the study was explained, and the patients were invited to participate. Parents and/or guardians signed a Free and Informed Consent Form and their identities were protected by the substitution of names by numbers. Data collection was performed between March 2013 and September 2014, in an office available at the outpatient clinic, on the same day of the consultations, lasting one hour.

Thirty-one patients with a previous diagnosis of DM1, aged between 6 and 17 years, participated in the study, and whose family participation met the conditions required to understand treatment guidelines. Patients with irregular outpatient follow-up, pregnant adolescents or with advanced chronic complications such as nephropathy, neuropathy and diabetic retinopathy were excluded.

For the development of the study were used: semi-structured questionnaire with demographic data (age, sex, ethnicity, schooling); eating habits (household food register); lifestyle (sedentary and or practitioner of PA); and socioeconomic level, according to the Brazilian Association of Research Companies, whose defined market division is of economic classes, besides estimating purchasing power[16].

Patients were evaluated for clinical and anthropometric parameters and for glycemic and lipid control at the beginning of the study and at the end of three follow-up periods of three months each, and the study design is shown in Figure 1.

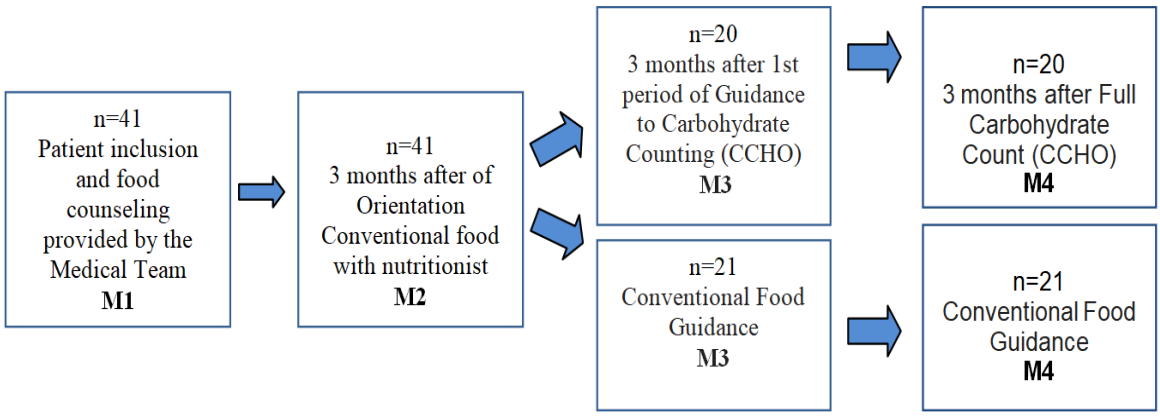

Figure 1. Division of the individuals studied in two groups, with and without carbohydrate counts.

In 1st stage (M1), 41 individuals were included in a regular follow-up who received guidelines from the medical team according to the guidelines of the SBD[6] regarding insulin doses, physical activity and

diet without proportionality to the dose of insulin given according to the meals. In a 2nd stage (M2), the selected ones had already been advised by a nutritionist regarding a food plan. In a 3rd stage (M3), participants were allocated into two groups: a) 20 patients who agreed to make CCHO with insulin administration according to CHO of each meal; b) 21 individuals who remained with the 2nd intervention approach, being considered as a control group for the 1st group in the follow-up period that included the CCHO. It lasted 3 months and was considered a time of learning and adaptation to the CCHO, optimization of insulin dose/gram of CHO that ranged from 1 unit/8 grams to 1 unit/30 grams. The sensitivity factor for correction of pre-meal glycemia ranged from 1:20 mg/dL to 1:50 mg/dL. At the 4th stage (M4), the analysis occurred after the period in which the patients already knew the CCHO and the form of insulin application and the data were obtained after 3-4 months of full count.

The anthropometric evaluation included the weight, obtained in digital scale Soehnle® brand; stature in the Alturaexata® stadiometer[17]; the skin folds (bicipital, tricipital, subscapular and supra iliac) obtained with Cescorf® scientific adipometer[18]; abdominal circumference (AC) measured around the maximum anterior bulge of the abdomen at the umbilical scar level with Cescorf® inextensible tape[17]. Being considered: undernourished, participants in the BMI percentile (BMIP) <P3; eutrophic (≥P3<P85); overweight (≥P85<P97) and obese (≥P97)[17]. The percentage of body fat (%BF) was calculated using tricipital (TSF) and subscapular (SESF) skinfolds, according to the SLAUGHTER equations[18].

Biochemical measurements employed the Roche-Hitachi Cobbas c501 automation system: fasting glycemia (Reference Value=60-99 mg/dL), postprandial (RV=<140 mg/dL) and fructosamine (RV=205-285 μmol/L) were determined by the enzymatic method with hexokinase;[19] glycated hemoglobin (RV=4.8-5.9%) determined by turbidimetric inhibition[19] total cholesterol (RV=<150); LDL-C (RV=<100); HDL-C (RV=45) and triglycerides (RV=<100) by the enzymatic colorimetric method[19]. The results were analyzed according to the glycemic and lipid control goals recommended by the ADA and V Brazilian Guideline for Dyslipidemias and Prevention of Atherosclerosis, respectively[3,20].

For food anamnesis, it was advised to fill out a household register and a frequency of consumption questionnaire. The conventional orientation was performed by calculating the energy needs according to age groups[3,6]. Every 3 months, the result of the orientation received, referring to that period, was evaluated by the exams cited.

The data obtained were analyzed for normality by the Shapiro Wilk test followed by the Levene test. Data of interest, anthropometric variables, glycemic control and lipid concentrations were analyzed, compared and described in the four stages. Variables with non-parametric behavior were analyzed by the test of variance proposed by Friedman and/or by ANOVA for repeated variables, followed by the multiple comparison test. Comparisons between two independent groups employed the Mann Whitney Test. And, in comparing variables that expressed control goals, Cochran's Q-Test was used for several related samples. A value of $p < 0.05$ was considered significant. In the analysis was used the program "Statiscal Package for Social Sciences" (SPSS) version 20.0 (SPSS Inc., Chicago, United States).

RESULTS

According to the ethnic group, the patients were distributed in whites (56.1%), browns (24.4%) and blacks (19.5%). In relation to schooling, 82.92% of the patients attended elementary school and 17.1% of the high school. According to the economic class, 43.9% of the participants were in classes C1 and C2 (income of $ 516.7 - 790.3/ American dolars) and 56.1% in classes D and E (income of 212.4 – 365.0), according to the minimum salary in 2014 ($ 304.0). Patients were on baseline-bolus regimen, with NPH insulin (73.2%) or glargine (26.8%); and three or more regular insulin boluses (56.1%) or lispro (34.2%), asparte (7.3%) and glulisine (2.4%) according to pre-meal capillary glucose.

The anthropometric data obtained in the four stages in both sexes were represented in table 1. In the female, significant differences were observed in relation to age; BMI (p=0.034); AC (p=0.005) and ΣSF (p<0.0001). In males, there was a significant difference in relation to age (p<0.0001); AC (p<0.0001); ΣSF (p<0.0001) and% BF (p<0.0001). Comparing the female to the male group, at the beginning of the evaluation, it was observed that ΣSF and %BF were significantly higher in females (Table 1).

Table 1. Anthropometric data of children and adolescents with type 1 *Diabetes Mellitus* (DM1) according to sex, analyzed at the initial time of intervention (M1), after conventional intervention period (M2), and intervention with carbohydrate counting (M3 and M4) - Uberaba, 2018.

	FEMALE					F x M START	MALE					F x M START
	M1 $\bar{x} \pm$ DP	M2 $\bar{x} \pm$ DP	M3 $\bar{x} \pm$ DP	M4 $\bar{x} \pm$ DP	#p	'p	M1 $\bar{x} \pm$ DP	M2 $\bar{x} \pm$ DP	M3 $\bar{x} \pm$ DP	M4 $\bar{x} \pm$ DP	#p	'p
Age&	12.8 (6.3- 17.6)	13.1 (6.6– 17.9)	13.5 (6.9 - 18.2)	13.7 (7.2-18.5)	< 0.0001 M4>M1, M2, M3 M3 > M1	0.153	10.8 (6.8–17.9)	11.0 (7.1- 18.2)	11.36 (7.4 – 18.5)	11.7 (7.7-18.8)	< 0.0001 M4 > M3, M2, M1 M3 > M2, M1 M2 > M1	0.153
BMI&	19.1 ± 3.327 (15.9 – 27.5)	19.8 ± 2.8 (16.5 – 27.0)	20.6 ± 2.9 (17.0 – 27.0)	20.5 ± 3.08 (16.7- 27.9)	0.034 M4 > M1 M3 > M1	0.105	18.3 ± 3.1 (14.4– 26.1)	18.5 ± 4.0 (8.0 – 27.0)	18.7 ± 3.3 (15.0 – 26.0)	18.9 ± 3.5 (14.5- 25.6)	0.093	0.105
BMIP&	61.2 ± 30.3 (14.0 – 98.0)	62.7 ± 25.9 (21.0 – 99.5)	66.0 ± 25.5 (15.0 – 99.7)	66.2 ± 25.1 (14.0- 98.1)	0.077	0.669	55.2 ± 26.7 (11.0– 99.0)	56.7 ± 29.9 (9.0 – 99.7)	54.7 ± 28.2 (9.0 – 99.0)	58.4 ± 27.8 (13.9- 99.5)	0.182	0.669
BMISD&	0.7 ± 1.9 (-1.1 – 7.8)	0.8 ± 1.8 (-0.8 -7.7)	0.64 ± 1.0 (-1.0 – 3.0)	0.6 ± 0.9 (-1.1-2.0)	0.619	0.624	0.3 ± 1.0 (-1.2-2.7)	0.3 ± 1.1 (-1.13– 2.70)	0.3 ± 1.2 (-1.47 – 3.2)	0.5 ± 1.3 (-1.1-3.9)	0.410	0.624
AC&	71.7 ± 10.6 (56.0 – 95.0)	72.1 ± 9.3 (57.0 – 91.0)	73.3 ± 9.2 (58.0 – 92.0)	72.7 ± 9.2 (57.5- 91.5)	0.005 M3 > M4	0.105	66.5 ± 9.5 (53.0– 89.0)	68.3 ± 9.6 (55.0 – 88.0)	69.5 ± 10.3 (55.0 – 91.0)	68.9 ± 9.9 (55.0- 89.5)	< 0.0001 M4, M3, M2 >M1	0.105
ΣSF&	55.3 ± 29.1 (23.0– 123.0)	56.0 ± 24.7 (27.0– 114.0)	60.6 ± 26.2 (23.0– 114.0)	58.3 ± 25.2 (25.0- 114.0)	< 0.0001 M3 > M1, M2 and M4	0.004 F > M	34.2±19.7 (15.0- 106.0)	39.2 ± 18.6 (16.0– 102.0)	44.1 ± 25.0 (19.0– 117.0)	41.6 ± 21.4 (17.5- 109.5)	< 0.0001 M4 > M2, M1 M3 > M4, M2, M1 M2 > M1	0.004 F > M
%BF&	25.4 ± 8.8 (15.5– 44.0)	26.0 ± 8.2 (13.6– 43.0)	27.0 ± 8.6 (11.6– 42.5)	26.6 ± 8.3 (12.6- 42.7)	0.054	0.0001 F > M	16.0 ± 9.3 (4.7– 49.4)	18.7 ± 10.3 (5.8 – 57.3)	20.2 ± 10.2 (9.4 – 52.5)	19.5 ± 10.0 (7.5-54.9)	< 0.0001 M4 > M2, M1 M3 > M4, M2, M1	0.0001 F > M

Source: Authors
Analysis of variance by Friedman.
Analysis of variance for repeated variables, followed by multiple comparison test.
&Mann-Whitney test for comparisons between 2 independent groups.
&Body Mass Index.
&Body Mass Index Percentile.
&Body Mass Index Standard Deviation.
&Abdominal Circumference.
&Sum of Skinfolds.
&Body Fat Percentile.

In the analysis of glycemic control, the patients were subdivided according to gender and CCHO (Table 2). In females, intragroup analysis did not show a significant difference at the time of the intervention; but in the intergroup analysis (CC versus without carbohydrate counting -WOC), FG concentrations were lower in M3 in the CC subgroup (222.6±86.2 versus 126.6±62.2 mg/dL, p=0.023). In the male group, a significant difference was observed in the intragroup analysis: in the subgroup CC, the concentrations of HbA1C and fructosamine, at the M4 stage were lower than in M1. The intergroup analysis showed that individuals who did CCHO (CC) started from lower AMG concentrations (M1: p=0.015) in relation to WOC, but this difference was not confirmed during the follow-up, and in M4, individuals who did CCHO (CC) presented lower values of fructosamine (425.8 ±81.4 versus 345.8±52.0, p=0.032). The comparison between the glycemic control parameters between the sexes showed only difference in M4 in relation to the fruit that was lower in males in the CC subgroup (p=0.049).

Table 2. Biochemical data related to the glycemic control of children and adolescents with DM1 according to gender and analyzed at the initial time of intervention (M1), after a period of conventional intervention (M2) and intervention with carbohydrate counting (M3 and M4), but with individuals subdivided between those who did not (WOC) and those who did (CC) the carbohydrate count - Uberaba, 2018.

		FEMALE				MALE			
		M1 $\bar{x} \pm$DP	M2 $\bar{x} \pm$DP	M3 $\bar{x} \pm$DP	M4 $\bar{x} \pm$DP	M1 $\bar{x} \pm$DP	M2 $\bar{x} \pm$DP	M3 $\bar{x} \pm$DP	M4 $\bar{x} \pm$DP
FG (mg/dL)	**WOC**	198.1 ± 72.5 (85.0-310.0)	210.8 ± 96.0 (76.0-364.0)	222.6 ± 86.2[a] (98.4-355.4)	198.6 ± 102.1 (108.0-453.9)	215.2 ± 108.3 (71.0 – 390.0)	161.5 ± 92.7 (33.0-336.0)	167.0 ± 89.0 (54.0-394.0)	154.5 ± 88.9 (68.9-390.3)
	CC	146.7 ± 84.4 (75.0-343.0)	132.8 ± 41.6 (78.0-204.0)	126.6 ± 62.6[a] (54.5-230.0)	166.2 ± 65.7 (75.6-297.7)	201.7 ± 103.8 (54.0-394.0)	153.6 ± 72.7 (60.0-275.0)	161.9 ± 94.8 (54.0-2950)	146.9 ± 57.0 (63.2-260.0)
AMG (mg/dL)	**WOC**	317.5 ± 202.7 (56.0-653.0)	278.4 ± 162.0 (102.0-589.0)	287.4 ± 147.4 (104.0-559.0)	243.5 ± 172.8 (50.9-604.2)	246.7 ± 94.8[d] (107.0-453.0)	174.0 ± 115.7 (58.0-424.0)	208.3 ± 108.5 (43.0-427.0)	161.7 ± 74.0 (60.8-343.0)
	CC	227.3 ± 172.0 (44.0-657.0)	183.5 ± 66.3 (102.0-292.0)	188.6 ± 107.8 (50.0-377.0)	141.8 ± 60.5 (64.0-228.4)	144.0 ± 80.2[d] (59.1-319.0)	183.7 ± 114.7 (57.0-442.0)	196.6 ± 106.5 (47.0-359.0)	162.2 ± 66.0 (63.6-240.9)
HbA_{1c} %	**WOC**	10.3 ± 3.7 (7.0-20.0)	9.6 ± 1.8 (7.0-13.0)	9.7 ± 1.8 (7.0-13.0)	9.4 ± 2.4 (6.2-13.6)	8.5 ± 2.09 (4.0-12.0)	8.0 ± 2.2 (4.0-12.0)	84 ± 1.7 (6.0-11.0)	8.6 ± 1.4 (6.1-10.4)
	CC	8.3 ± 1.6 (6.0-11.0)	9.1 ± 1.8 (6.6-12.0)	9.2 ± 2.3 (6.6-15.0)	9.0 ± 1.5 (6.9-11.2)	9.5 ± 2.0[b] (7.0-14.0)	8.9 ± 1.0[b] (8.0-11.0)	9.1 ± 1.5[b] (7.0-12.0)	8.2 ± 1.4[b] (6.8-11.5)
FRUCTOSAMINE (umol/L)	**WOC**	425.6 ± 118.8 (282.0-630.0)	441.5 ± 97.1 (310.0-631.0)	465.4 ± 102.3 (320.0-617.0)	451.9 ± 135.9 (294.0-638.0)	439.1 ± 64.6 (365.0-605.0)	365.8 ± 76.2 (246.0-485.0)	392.0 ± 74.1 (275.0-486.0)	425.8 ± 81.4[e] (305.0-574.0)
	CC	397.7 ± 117.3 (260.0-617.0)	419.2 ± 136.9 (230.0-609.0)	413.1 ± 152.4 (200.0-651.0)	432.5 ± 103.3[f] (240.0-581.0)	433.4 ±110.7[c] (240.0-579.0)	378.2 ± 85.6[c] (227.0-513.0)	404.8 ± 77.6[c] (311.0-513.0)	345.8 ± 52.0[c; e; f] (278.0-414.0)

Source: Authors

º Mann-Whitney test for comparisons between 2 independent groups.

a : p= 0.023.

b: p= 0.041 (CC; M4 < M1)

c: p= 0.05 (CC; M4 < M1)

d: p= 0.015 (CC < WOC)

e: p= 0.032 (CC < WOC)

f: p= 0.049 (M < F)

Concentrations of HbA1C and fructosamine presented great individual variability; but there were more individuals off goal in those who did not do CCHO. Considering, specifically, HbA1C, 26 individuals (63.4%) were outside the strict control target in M1; and in M4, 23 (56.1%) remained out. Reductions in their concentrations were calculated individually at each follow-up time and data were plotted in Table 3.

The combined analysis, without gender separation or CHO count, did not show any difference between the reductions obtained between the various stages. When categorizing the sample for CCHO, the reductions of HbA1C were significant (p=0.003) in those that did CCHO, and in the M4-M2 comparisons with M3-M1 (Table 3).

Table 3. Difference between values of glycated hemoglobin (HbA1C) in patients with DM1 at the stages analyzed, but with the same subgroups without counting (WOC) and counting (CC) - Uberaba, 2018.

Difference between HbA_{1C} concentrations at 4 stages	WOC	CC
	$\bar{x} \pm$ DP (minimum and maximum)	$\bar{x} \pm$ DP (minimum and maximum)
M2-M1	- 0.175 ± 1.65 (- 2.4 → 4.0)	0.01 ± 1.5 (- 3.2 → 2.9)
M3-M1	0.315 ± 1.7 (- 2.7 → 4.4)	0.14 ± 1.5 (- 3.0 → 3.4)
M4-M1	- 0.04 ± 1.89 (- 3.4 → 3.4)	- 0.34 ± 1.47 (- 4.1 → 3.1)
M3-M2	0.46 ± 1.78 (- 1.9 → 4.4)	0.13 ± 1.58 (- 2.3 → 3.7)
M4-M2	0.135 ± 1.86 (- 4.0 → 3.4)	- 0.35 ± 1.42 (- 2.3 → 4.1)
	# p= 0.069	# p= 0.033 M4-M2 > M3-M1

Source: The author
Analysis of Variance by Friedman's Posts.
Analysis of variance for repeated variables, followed by multiple comparison test.

Table 4. Biochemical data related to the lipidic control of children and adolescents with DM1 according to gender and analyzed at the initial time of intervention (M1), after a period of conventional intervention (M2) and intervention with carbohydrate counting (M3 and M4), but with individuals subdivided between those who did not (WOC) and those who did (CC) the carbohydrate count - Uberaba, 2018.

		FEMALE				MALE			
		M1 $\bar{x} \pm$ DP	M2 $\bar{x} \pm$ DP	M3 $\bar{x} \pm$ DP	M4 $\bar{x} \pm$ DP	M1 $\bar{x} \pm$ DP	M2 $\bar{x} \pm$ DP	M3 $\bar{x} \pm$ DP	M4 $\bar{x} \pm$ DP
COL (mg/dL)	**WOC**	182.3±65.4 (128.4-356.4)	172.8±33.2 (120.0-231.0)	174.6±28.6 (138.0-220.0)	173.7±267 (129.0-211.7)	147.4±23.8 (108.8-184.0)	150.5±28.6 (100.1-198.0)	145.9±26.0 (92.0-191.0)	148.2±25.7 (104.0-194.0)
	CC	164.5±34.2 (126.0-249.8)	157.6±44.7 (91.8-254.0)	160.5±35.0 (120.7-221.3)	159.0±34.5 (116.0-237.6)	156.2±19.0 (115.0-177.0)	158.7±26.2 (122.3-203.9)	157.0±20.5 (108.6-178.0)	157.9±21.2 (115.4-187.4)
LDL-C (mg/dL)	**WOC**	109.9±50.9 (60.2-240.0)	98.4±32.0 (54.0-150.0)	98.6±35.6 (67.0-155.0)	98.5±29.4 (60.5-146.0)	77.5±20.8 (49.5-116.0)	85.2±21.9 (42.0-121.7)	73.9±24.8 (31.0-123.7)	79.54±22.1 (41.5-122.7)
	CC	95.5±32.8 (54.0-173.0)	89.0±39.9 (28.0-176.0)	90.0±24.7 (59.9-133.2)	89.5±29.2 (51.7-150.5)	85.2±14.9 (66.8-115.0)	88.0±19.8 (53.3-119.0)	86.6±12.3 (63.2-100.4)	87.28±14.0 (66.15-108.0)
HDL-C (mg/dL)	**WOC**	53.6±11.6 [b] (31.0-67.0)	56.0±7.9[b] (40.0-63.0)	60.2±10.7[b] (42.0-70.0)	58.1±8.1[b] (44.5-66.0)	57.9±13.3 (35.9-87.0)	59.5±15.0 (34.0-90.0)	61.0±13.9 (40.0-86.0)	60.58±14.0 (37.0-87.0)
	CC	54.7±11.9 (35.0-73.0)	55.7±12.6 (38.0-73.0)	51.4±13.8 (31.0-79.0)	53.6±10.6 (38.5-76.0)	58.7±16.7 (35.0-82.0)	58.3±19.2 (28.0-91.0)	59.7±17.1 (34.0-85.0)	59.0±17.9 (32.5-88.0)
VLDL-C (mg/dL)	**WOC**	22.4±19.0 (7.0-64.8)	18.6±5.2 (13.4-26.0)	15.9±5.6 (9.0-27.6)	17.3±3.8 (11.2-24.0)	11.8±3.5 (7.4-19.0)	11.5±4.4 (0.8-15.3)	10.5±2.4 (7.4-14.0)	11.0±3.0 (4.2-14.2)
	CC	14.3±4.75 (8.0-21.8)	12.8±3.9 (6.41-18.2)	19.5±17.2 (7.4-65.0)	16.2±9.7 (6.8-40.0)	12.3±2.6 (6.8-15.2)	12.5±3.7 (7.6-20.8)	10.7±4.0 (6.0-18.4)	11.6±2.7 (7.3-14.6)
TG (mg/dL)	**WOC**	98.7±80.6[a] (53.0-324.0)	93.2±25.0[a] (67.0-131.0)	78.5±27.5[a] (46.0-138.0)	85.8±19.0[a] (57.0-119.5)	60.0±17.3 (37.0-95.0)	66.5±13.5 (47.0-80.0)	56.8±19.0 (37.0-104.0)	61.7±12.7 (45.5-92.0)
	CC	74.3±20.6 (42.0-107.0)	68.5±25.2 (30.6-113.0)	77.1±35.4 (36.9-155.0)	72.8±24.9 (33.75-115.5)	63.1±12.7 (34.0-73.7)	66.5±20.0 (38.0-104.0)	56.6±20.9 (28.0-92.0)	61.5±18.8 (36.5-90.5)

Source: The author
Analysis of variance by Friedman.
Analysis of variance for repeated variables, followed by multiple comparison test.
º Mann-Whitney test for comparisons between 2 independent groups.
a: p = 0.045 (M3 <M4, M2 and M1, M4 <M2)
b: p = 0.016 (M3> M4, M2e M1, M4> M2)

DISCUSSION

The present study demonstrates the difficulty of reaching therapeutic targets recommended as ideal, in order to protect children and adolescents from chronic complications, independently of the nutritional approach used, with a discrete advantage for interference that used CCHO.

The mean values of glycemic control parameters of short (fasting and postprandial glycemia), medium (fructosamine) and longer term (HbA1C) remained inadequate at the different stages of the evaluation. The reductions in HbA1C concentrations over time were discrete expressing significant differences between M4 and M2 (Table 3). These data agree with some authors who suggest that the improvement in the control parameters in the period of CCHO is cumulative and with positive results after six months[8.10].

In the male patients, reductions in HbA1C were lower at the end of the evaluation (M4<M1 p=0.041) indicating that, although the mean values were not less than 7.5% recommended (ADA), 8.2±1, 4% is better than 9.5 ± 2.0 (Table 2) because for each reduction of 1% HbA1C there is a significant reduction of the microvascular disease of type 1 diabetes[4]. These results were repeated with the fructosamine, which was also lower in males than females in M4 in those with CCHO (p=0.049 - Table 2). It is questionable why the male group presented better results than the female group. Would they be more motivated for better adherence to CCHO or would it be a sum of factors such as greater physical activity in this age group?

These findings regarding fructosamine as a glycemic control marker during CCHO did not find similar data in the literature, since this is an original study. It is a marker that reflects the most current glycemic averages (~30 days) and is an interesting finding for short-term studies, or even in clinical practice.

It is questioned why the CCHO-associated insulin therapy does not result in more significant reductions in glycemic control markers. Many authors describe the need for a pre-training period for CCHO to be effective[10,11]. In the present study, we applied this recommendation within 3 months of individualized learning (M3). However, it is possible that not all patients, and their parents, have assimilated the instructions equally, or that they have allowed greater intake of foods rich in simple sugars in this period.

Another possibility is the inadequate use of insulin for several reasons such as: insecurity in interfering with the doses before the consultation; fear of hypoglycaemic events; lack of inputs; food transgressions; difficulty in performing self-monitoring by adolescents in the absence of parents during the work day or in the school period; and glycemic variability, common among patients with DM1[13].

It should be noted that, from the beginning and throughout the intervention, most of the individuals were within the recommended lipid control goals. And those who were out of target had borderline concentrations of CT, LDL-C and TG. Throughout the intervention, this profile was kept, showing the non-interference of CCHO as demonstrated by other authors[10].

Analyzing the differences as to the ages, since the research was developed in 18 months, that is the reason why the ages between the analyzed stages were significantly different. The patients were in the growth phase explaining the differences in the anthropometric parameters.

The comparison between the sexes showed a higher % BF and ΣSF in females from the initial stage. For the purposes of analysis, the patients were also subdivided according to the intervention (CC and WOC), showing that differences in females occurred only in the CC group, adding to the other factors previously listed. In the male group, the anthropometric alterations occurred independently of the CCHO.

An important question is whether CCHO results in weight gain. There was no difference between M4/M3 and M1 in BMI, BMI% and BMISD in the CC group. And such data did not differ from the WOC group indicating that there was only redistribution of body fat. Therefore, these data and those in the literature(8) indicate that CCHO does not lead to weight gain although there may be changes in body composition in patients in growth phase.

CONCLUSION

Although glycemic control parameters did not reach the therapeutic targets, at the end of the periods of the proposed interventions, the individual analysis shows that more patients reached or were closer to the appropriate therapeutic indexes in M4

than in M1, with a slight advantage for the subgroup CC, justifying the effort of the patients, the family and the team that assists patients with DM1.

STUDY LIMITATIONS

Although the patients were assisted in an outpatient clinic specialized in DM1 for children and adolescents, the available supplies were provided by SUS. Thus, the insulin preparations, the number of reagent strips for home self-monitoring varied and even faced periods of scarcity, giving heterogeneity to the groups. In addition, the study involved a broad age group with specific challenges. The level of education, availability and motivation of patients and their families interfered in the resistance, adhesion and effectiveness of the proposed.

We emphasize that one of the research's strengths is that it is a real-life study and the team seeks to use the resources inherent to each patient.

INTERFERÊNCIA NUTRICIONAL NO TRATAMENTO DE PACIENTES COM *DIABETES MELLITUS* TIPO 1

RESUMO

O tratamento do *Diabetes Mellitus* tipo 1 (DM1) constitui-se na adesão ao tratamento insulínico, na alimentação e na atividade física, visando ao controle glicêmico. O objetivo deste estudo foi observar os efeitos da interferência nutricional no tratamento de pacientes com DM1. Trata-se de estudo quantitativo, prospectivo e longitudinal desenvolvido no Ambulatório de Diabetes da UFTM. A coleta de dados foi realizada entre março de 2013 e setembro de 2014. Foram avaliados 41 crianças e adolescentes entre 6 e 17 anos, quanto à antropometria, controle glicêmico e lipídico em 4 momentos: M1 no início do seguimento; M2 após orientação nutricional convencional; M3 após aprendizagem da contagem de carboidratos (CCHO) e M4 em contagem plena. A análise estatística foi descritiva e inferencial. A antropometria comprovou que a CCHO não resultou em ganho de peso e foi efetiva no sexo masculino, demonstrada pela redução nas concentrações de frutosamina (p=0,050) e HbA1C (p=0,041) no M4 comparado ao M1. Considerando a frutosamina, o grupo com CCHO se diferenciou do grupo sem CCHO M4 (p=0,035). A terapêutica insulínica associada à CCHO demonstrou ser um recurso importante a ser integrado no tratamento do DM1, visando atingir alvos efetivos na redução das complicações.

Palavras-chave: Criança. Adolescente. *Diabetes Mellitus* tipo 1. Carboidratos da dieta. Avaliação nutricional.

INTERFERENCIA NUTRICIONAL EN EL TRATAMIENTO DE PACIENTES CON *DIABETES MELLITUS* TIPO 1

RESUMEN

El tratamiento de la *Diabetes Mellitus* tipo 1 (DM1) se constituye en la adhesión al tratamiento medicamentoso, en la alimentación y en la actividad física, centrando en el control glucémico. El objetivo de este estudio fue observar los efectos de la interferencia nutricional en el tratamiento de pacientes con DM1. Se trata de estudio cuantitativo, prospectivo y longitudinal desarrollado en el Ambulatorio de Diabetes de la UFTM (Universidade Federal do Triângulo Mineiro). La recolección de datos fue realizada entre marzo de 2013 y septiembre de 2014. Fueron evaluados 41 niños y adolescentes entre 6 y 17 años, en cuanto a la antropometría, control glucémico y lipídico en 4 momentos: M1 en el inicio del seguimiento; M2 tras orientación nutricional convencional; M3 tras aprendizaje del conteo de carbohidratos (CCHO) y M4 en conteo pleno. El análisis estadístico fue descriptivo e inferencial. La antropometría comprobó que el CCHO no resultó en ganancia de peso y fue efectivo en el sexo masculino, demostrado por la reducción en las concentraciones de fructosamina (p=0,050) y HbA1C (p=0,041) en el M4 comparado al M1. Considerando la fructosamina, el grupo con CCHO se diferenció del grupo sin CCHO M4 (p=0,035). La terapéutica insulínica asociada al CCHO demostró ser un recurso importante a ser integrado en el tratamiento del DM1, a fin de alcanzar blancos efectivos en la reducción de las complicaciones.

Palabras clave: Niño. Adolescente. *Diabetes Mellitus* tipo 1. Carbohidratos de la dieta. Evaluación nutricional.

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