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Case Report: Herein, we present a case of a 19-year-old male with newly diagnosed T1DM. The patient was first put on an insulin regime. Twenty days later, he shifted towards the paleolithic ketogenic diet and was able to discontinue insulin. Strict adherence to the diet resulted in normal glucose levels and a more than three-fold elevation of C-peptide level indicating restored insulin production. Currently, the patient is on the paleolithic ketogenic diet for 6.5 months. He is free of complaints, and no side effects emerged.
Conclusion: We conclude that the paleolithic ketogenic diet was effective and safe in the management of this case of newly diagnosed T1DM. Marked increase in C peptide level within two months indicates that the paleolithic ketogenic diet may halt or reverse autoimmune processes destructing pancreatic beta cell function in T1DM.

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Keywords: Type 1 diabetes mellitus, Ketogenic diet, Paleolithic-ketogenic diet, C-peptide, Evolutionary medicine

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INTRODUCTION

Diabetic patients are, generally, recommended to follow a diet that is low in fat and high in carbohydrates [1]. Clinical studies, conversely, showed metabolic benefits conferred by carbohydrate-restricted diets including the ketogenic diet [2, 3] and the paleolithic diet [4, 5] in type 2 diabetes. Much less data on the use of low carbohydrate diets in type 1 diabetes (T1DM) are available. Two studies by Nielsen et al. showed that a low carbohydrate diet lowers the need for insulin as well as the number of hypoglycemic episodes in T1DM [6, 7]. It was also suggested that a low carbohydrate diet is sustainable on the long-term [6, 7]. Ketogenic diets have long been used in epilepsy [8]. There are three cases in literature, where concurrent epilepsy and T1DM were treated with the classical ketogenic diet and both conditions improved [9–11]. Recently, we published a case of childhood absence epilepsy where seizure freedom was achieved using a modified ketogenic diet we refer to as the paleolithic-ketogenic diet [12]. Herein, we present a case of T1DM, where the same diet resulted in remission of T1DM as assessed by normalization of blood glucose levels and elevation in C-peptide level allowing for discontinuation of external insulin replacement.

CASE REPORT

A 19-year-old male complained of increased thirst, polyuria, itchy skin, malaise, and weight loss. The symptoms were present for about two weeks prior to

diagnosis. On November 24, 2013 self monitoring of blood glucose showed 384 mg/dL. Previous medical history was unremarkable. Anamnestic data included consuming of muscle boosting protein through a month prior to symptom onset. Laboratory assessment on November 25, 2013 (Table 1) showed elevations in glucose (218 mg/dL) and HbA1c (9.2%). Testing for glutamic acid decarboxylase (GAD) antibodies showed positivity (52 U/mL; normal range 0–10 U/mL) and mild positivity for pancreatic islet cell autoantibodies (ICAs). C-peptide level was not measured at this time. He was diagnosed with T1DM. He was put on insulin replacement therapy (38 IU of insulin) and standard conventional diabetes diet with six meals containing 240 grams carbohydrate daily. He followed this regime for 20 days. While on this regime his glucose levels fluctuated between 68–267 mg/dL (Figure 1).

Table 1: Laboratory data at the time of diagnosis on a normal diet (on November 25, 2013) and at 10 weeks after diet initiation, on the paleolithic-ketogenic diet without insulin (on March 14, 2014). Note the normal level of glucose, HbA1c and low level of triglyceride while on the paleolithic-ketogenic diet. Dashes indicate that the given parameter was not measured.

Normal diet		Paleolithic-ketogenic diet	
WBC	5.9	5.4	G/l
RBC	5.7	5.3	T/l
Hemoglobin	16	15.2	G/dL
Hematocrit	48	45	%
Iron	136.3	98.9	µg/ dL
Thrombocyte	230	150	G/l
Sodium	134	139	mEq/l
Potassium	3.9	3.8	mEq/l
Calcium	9.52	10	mg/dL
Magnesium	—	2.02	mg/dL
Carbamide	10.6	14.8	mg/dL
Creatinine	0.88	0.97	mg/dL
eGFR	>90	>90	
Glucose	218	88	mg/dL
Hb1Ac	9.2	5.5	%
Total cholesterol	143	301	mg/dL
HDL cholesterol	—	54.8	mg/dL
LDL cholesterol	—	224	mg/dL
Triglyceride	168	111	mg/dL
Uric acid	6.12	—	mg/dL
GOT	31	19	U/l
GPT	44	18	U/l
GGT	17	16	U/l
Total bilirubin	0.94	—	mg/dL
TSH	3.53	—	mIU/l
CRP	1.1	—	mg/L

Abbreviations: WBC - white blood cell count, RBC - red blood cell count, eGFR - estimated glomerular filtration rate, HbA1c - glycated hemoglobin, HDL - high density lipoprotein, LDL - low density lipoprotein, TSH - thyroid stimulating hormone, CRP - C-reactive protein

Carbohydrate he consuming before was less than 240 grams. Since his malaise did not improve the patient consulted the first author in December 2013. To ascertain T1DM laboratory examination of C-peptide was carried out. C-peptide level of 0.6 ng/mL measured on January 08, 2014 indicated subnormal insulin secretion (Figure 2). The patient was suggested to switch to the paleolithic-ketogenic diet which he initiated on December 21, 2013. From this time, he was also taking 5,000 IU of vitamin D3 but nothing else as supplement. His diet consisted of meat, organ meat, fat and eggs. In his diet, red and fat meats dominated over lean meats. He was eating vegetables in insignificant amounts. His diet had a ketogenic ratio (fat : protein + carbohydrate) of at least 2:1. No oil of plant origin or artificial sweeteners were allowed. The patient was under our close control and reported daily food records and blood glucose levels. Upon shifting toward the paleolithic ketogenic diet glucose levels returned to normal and no major elevations were seen postprandially either. Insulin was therefore discontinued. The tapering of insulin was done promptly: following the first paleolithic-ketogenic meal glucose level was only 86 mg/dL thus there was no need for external insulin. Similar blood glucose levels were measured on subsequent meals on the diet. Thus, the patient required no insulin subsequently either. Home glucose monitoring was carried out preprandially as well as postprandially and tracked once a day for consecutive meals (that is on consecutive days measures were taken for breakfast, lunch and dinner, respectively). Average blood glucose level while on the standard diabetes diet with insulin was 119 mg/dL while 85 mg/dL on the paleolithic-ketogenic diet without insulin. Fluctuations in glucose levels decreased as indicated by a reduction of standard deviation values from 47 mg/dL on the standard diabetes diet to 9 mg/dL on the paleolithic-ketogenic diet. Average postprandial glucose elevation on the standard diabetes diet was 23 mg/dL while only 5.4 mg/dL on the paleolithic-ketogenic diet (Figure 1).

C-peptide measurement was repeated on the 10th week of the diet (on March 06, 2014). This indicated an elevation to a value of 2.2 ng/mL (Figure 2). A comprehensive laboratory workup carried out on March 14, 2014 indicated normal laboratory parameters with the exception of total cholesterol and LDL cholesterol which were slightly elevated. Glucose level was 88 mg/dL while HbA1c was 5.5% (for all laboratory values see Table 1). Urinary ketone was positive. On March 21, 2014 antibody testing for ICA showed no change in the mild

positivity measured before and some elevation in GAD antibodies (86 U/mL). At the time of writing this case report, the patient is on the paleolithic ketogenic diet for 6.5 months and still exhibit low glucose levels. No side effects emerged and he is completely free of symptoms.

The patient gave written informed consent for writing this case study.

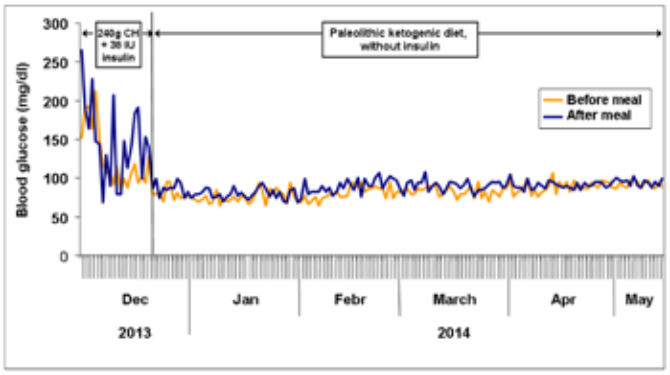

Figure 1: Blood glucose levels while on the standard diabetes diet containing 240 g carbohydrate with insulin therapy and while on the paleolithic-ketogenic diet without insulin. Glucose was measured preprandially and postprandially once a day for consecutive meals (that is on consecutive days measures were taken for breakfast, lunch and dinner, respectively). Note low glucose levels and the absence of major postprandial elevations while on the paleolithic ketogenic diet. Due to stable glucose levels through five months, from May 15, 2014 the patient switched to self-monitoring his glucose levels only once a week.

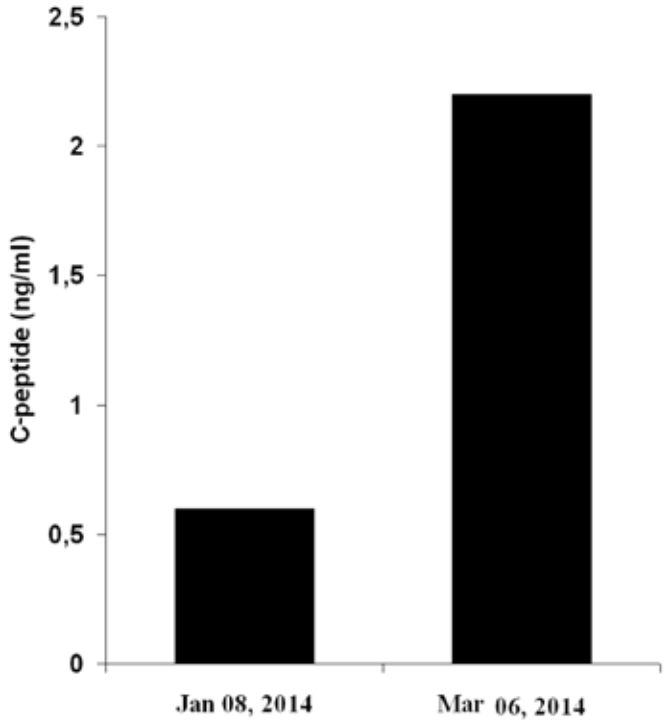

Figure 2: C-peptide levels shortly after diet initiation (on the 18th day of the paleolithic ketogenic diet) and two months later. Note the more than three-fold increase in C-peptide within two months.

DISCUSSION

This is a first report of T1DM being successfully managed with the paleolithic-ketogenic diet. In literature, a few studies are available on the use of carbohydrate restricted diet in the treatment of T1DM [6, 7]. Low carbohydrate diet in these studies resulted in reduced number of hypoglycemic episodes and also lowered the need for insulin. In our case, however, insulin replacement was not just reduced but could be stopped. Importantly, insulin discontinuation was paralleled by a marked increase in C peptide level indicating restored pancreatic insulin production.

Shortly before diabetes onset our patient consumed muscle boosting protein which contained bovine milk protein. Consumption of cow's milk has repeatedly been shown to increase risk of T1DM [13, 14]. It is suggested that bovine milk protein may promote autoimmune processes giving rise to T1DM [15]. Also in two case reports from literature, where epilepsy was treated with the classical ketogenic diet, which contains large amount of dairy, T1DM developed subsequently [9, 11]. A major difference between the classical ketogenic diet and the paleolithic ketogenic diet is that milk and dairy are excluded in the latter. We suggest that the paleolithic ketogenic diet not only normalize glucose levels but may also halt autoimmune processes mediated by non-paleolithic substances including milk protein [16].

While on the paleolithic-ketogenic diet glucose levels remained low both during preprandially and postprandially. Follow-up laboratory assessment indicated laboratory parameters remaining in the normal range except for elevations in total cholesterol and LDL cholesterol. In fact, these elevations are expected on a diet rich in animal fat and cholesterol and were also reported in studies with the classical ketogenic diet [17] as well as in our previous case of childhood absence epilepsy treated with the paleolithic ketogenic diet [12]. Moreover, it is now acknowledged that neither dietary nor serum cholesterol represent a risk factor for cardiovascular disease [18]. On follow-up antibody testing ICA remained mildly positive while GAD antibodies elevated to some extent. Although these parameters are frequently associated with T1DM they do not seem to be specific nor indicate progression of disease [19].

Type 1 diabetes mellitus is considered as a lifelong metabolic condition due to the exhaustion of insulin-secretory cells of the pancreas. Therefore, T1DM is generally believed to be untreatable by any diet. There are indications, however, that residual pancreatic beta cell functioning may extend well beyond the time of diagnosis [20]. Nevertheless C-peptide levels decrease monotonically through years after diagnosis [20]. We are not aware of any data from literature indicating elevation of C-peptide resulting from a dietary intervention. A recent case study of a child with T1DM reported remission without insulin on gluten-free diet [21]. However, in that case C-peptide continued to decline while on the gluten-free diet.

In the standard care of T1DM insulin is a cornerstone. It is important to emphasize that the paleolithic-ketogenic diet as a standalone therapy may be applied only in those cases with residual insulin secretion. In cases with no internal insulin secretion the paleolithic-ketogenic therapy may be only used as an adjunct to insulin replacement.

CONCLUSION

We suggest that an intervention with the paleolithic ketogenic diet in an early stage of the disease with residual insulin secretion may halt or reverse type 1 diabetes mellitus (T1DM). Follow-up at sixth month in the case of our patient is relatively short and the positive results may appear as a honeymoon effect. However, this term is used in relation to the beginning of insulin therapy not the end of it. We believe that with normalized insulin secretion and a further adherence to the diet the patient may be managed on the long-term.

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Author Contributions

Tóth Csaba – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Zsófia Clemens – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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