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ORIGINAL ARTICLE

Impact of Momordica charantia (karela) on serum aspartate aminotransferase level in streptozotocin induced diabetic rats

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Abstract

The present experimental study was conducted to investigate whether Momordica charantia (karela) has got any impact on serum aspartate aminotransferase (AST) level in the streptozotocin induced diabetic rats. Sixty healthy young Long Evans rats of male sex weighing 150 to 280 gm aged between 10 to 12 weeks were used in this study. The rats were divided into 4 equal groups depending on their different sorts of dietary feedings and drug treatment. Serum AST level was estimated in all rats up to day 51 from the day of streptozotocin/ vehicle injection. The mean ± SD of final serum AST level as percentage of corresponding initial level (value on 51st day to the value on 7th day) was 95.1 ± 13.4 u/L in healthy rats, 110.0 ± 7.6 u/L in the untreated diabetic rats, 39.4 ± 10.1 u/L in the insulin-treated diabetic rats and 109.5 ± 23.8 u/L in the karela-treated diabetic rats. The AST percentage change value of diabetic rats on 51st day corresponding to the initial on 7th day was significantly higher than that healthy rats ($p < 0.01$). The value in the insulin-treated diabetic rats was significantly lower than that of the untreated diabetic rats ($p < 0.001$) and the karela-treated diabetic rats ($p < 0.001$). There was no significant difference between the values of the untreated diabetic rats and the karela-treated diabetic rats ($p > 0.05$). Karela showed a tendency of acting against hyperglycemic effects of streptozotocin induced diabetes mellitus and also acting against high serum aspartate aminotransferase (AST) level in streptozotocin induced diabetes mellitus. However, further investigations are recommended for establishing karela as a safe and useful effective anti-hyperglycemic agent as well as an agent acting against the rise in serum AST level in streptozotocin induced diabetic rats.

Key words: Diabetes mellitus, Hyperglycemia, Serum aspartate aminotransferase, Momordica charantia (karela)

Introduction

Now a days, diabetes mellitus is a major health problem. In Bangladesh a high proportion of diabetics are registered to different clinics and institutes, among which only Bangladesh Institute of Research and Rehabilitation in Diabetes, Endocrine & Metabolic Disorders (BIRDEM) has registered 193271 cases in 1998, which is higher than that of previous year. Although it

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is understandable that many of the diabetic patient especially in the rural area have not registered themselves to any diabetic clinic or hospital and many other still remain undiagnosed.

In the treatment of diabetes mellitus, synthetic oral hypoglycemics have various side effects and contraindications that have led scientists to search for alternatives including many natural products indigenous to various parts of the world. A large number of herbal products have been used as remedies of polyuria. Some of these are in the usual food list of the people concerned. Scientific studies also revealed the hypoglycemic properties in many of these herbal products. Among these herbal products Momordica charantia ('karela' or bitter gourd) is one of such natural products, cultivated in the many parts of Africa, South America and Asia. The fruit is very popular as a vegetable in Bangladesh. In Sri Lanka the fruit juice of Momordica charantia is considered as an effective hypoglycemic agent in management of diabetes mellitus. In the other parts of the world it is used as the folk medicine to the treatment of diabetes.[1] Karela's hypoglycemic property has also been shown experimentally in the diabetics as well as in normal laboratory animals.[2-5]

As because diabetes mellitus causes rise in serum aspartate aminotransferase (AST) level, it may be hypothesized that karela being an anti-diabetic agent may also be used to minimize the rise of AST level in streptozotocin induced diabetes rats. Thus the aim of the present study was to determine whether karela has any impact on AST level in streptozotocin induced diabetic rats.

Materials and Method

The experiment was carried out on a total number of 60 young rats of male Long Evans strain. They were 10 to 12 wks old, weighing between 150 and 280 gm. Among them 15 rats treated intraperitoneally with vehicle (citrate buffer solution 1 ml/kg body weight) only were used as control rats (Group A) and 45 rats were treated with vehicle and streptozotocin found as diabetes, 15 of which were treated as untreated diabetic control group (Group B), 15 were treated again with insulin at a dose of 1–3 units/kg body weight/day, were treated as the insulin-treated diabetic group (Group C) & 15 were treated with karela at a dose of 10 ml/kg body weight/day orally through tube to control the diabetes mellitus and was called as 'karela'-treated diabetic group (Group D).

In the Research Division of BIRDEM, Dhaka, the AST was estimated for day 7 and on day 51. In this method for sample and blank, one test tube for each was taken and one ml of AST (SGOT) buffer reagent was taken in each test tube. Serum, 0.2 ml, was added to the sample tube. Redistilled water, 0.2 ml, was added to the blank tube. Both of the test tubes were placed in the water bath at 37°C for 30 minutes. After that period, the test tubes were taken out and 1 ml of detection reagent was added to each tube and kept at room temperature for further 20 minutes. Then 10 ml of 0.4 N sodium hydroxide solutions was added to each test tube. After 6 minutes, reading was taken from colorimeter at wavelength 340-365 nm.

Intergroup statistical differences were tested using Student's t-test and the significance level was considered as $p < 0.05$.

Results

Serum AST level was estimated in all rats on day 7 and day 51 from day after streptozotocin/vehicle injection shown in Table 1. The mean ± SD of final serum AST level as percentage of corresponding initial level (value on day 51 day to the value on day 7) was 95.1 ± 13.4 u/L in healthy rats, 110.0 ± 7.6 u/L in the untreated diabetic rats, 39.4 ± 10.1 u/L in the insulin-treated diabetic rats and 109.5 ± 23.8 u/L in the karela-treated diabetic rats. The AST percentage change value of diabetic rats on day corresponding to the initial on day 7 was significantly higher than that healthy rats ($p < 0.01$). The value in the insulin-treated diabetic rats was significantly lower than that

Table 1. Serum aspertate aminotransferase (AST) level in different groups of rats (n=15 in each group

Group	Serum AST Level (u/L)		
	Initial level (on day 7) Mean±SD	Final level (on day 51) Mean±SD	Final level as % of initial level Mean±SD
A (Healthy group)	30.5±2.7	28.8±2.8	95.1±13.4
B (Untreated diabetic)	89.9±16.0	99.0±14.6	110.0±7.6*
C (Insulin-treated diabetic)	90.2±22.7	33.0±2.5	39.4±10.1#
D (Karela-treated diabetic)	99.5±13.5	107.2±22.0	109.5±23.8$

*: Group A vs Group B ($p < 0.01$); #: Group B vs Group C ($p < 0.001$); $: Group B vs Group D ($p > 0.05$) by Student's t-test.

of the untreated diabetic rats ($p < 0.001$) and the karela-treated diabetic rats ($p < 0.001$). There was no significant difference between the values of the untreated diabetic rats and the karela-treated diabetic rats ($p > 0.05$).

Discussion

Untreated diabetic rats showed a significant rise serum AST level than the healthy rats. Higher serum AST level has been found to be a feature of experimentally induced diabetic rats. The higher serum AST level of the diabetic rats was decreased by the treatment with insulin. The decrease in serum AST level was significantly higher in insulin treated diabetic rats than that in the untreated diabetic rats. There was no significant difference in the decrease of AST between the untreated diabetic rats and the karela treated diabetic rats in this regard. However, in other study it was found that the serum AST level in karela treated diabetic rats was significantly lower than that in the untreated diabetic rats and this impact was similar to that of the effect of insulin on Streptozotocin- induced diabetic rats.[1] Doi et al. found the higher serum AST level in Streptozotocin-induced diabetic rats than that in the control mice.1 In case of diabetes mellitus serum AST level is increased and thus serum AST level may be an important landmark to detect the diabetic status.[6,7] However, the present study did not show that karela has any AST level lowering effect. The reason may be that the other study was conducted on male Sprague-Dawley rats instead of male Long Evans strain.

Conclusion

Momordica charantia (karela) showed a tendency of lowering the rise in serum AST level in diabetes mellitus. However, further investigations are recommended for establishing the active ingredient(s) of karela as a safe, useful and effective anti-hyperglycaemic agent as well as an agent against the rise in serum AST level.

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