ABSTRACT

INTRODUCTION:

‘Metabolic syndrome’ is a constellation of metabolic derangements with a global prevalence is approximately 16%. There is no well defined cut-off point in treating metabolic syndrome, as of now, individual components are treated in addition to the risk factors. Moreover, no single drug is available to treat all the individual components of the syndrome altogether.

AIMS AND OBJECTIVES

To understand the biological basis of the effect of *Momordica charantia* and also its effectiveness as a mono-therapy for metabolic syndrome.

MATERIALS AND METHODS:

After obtaining the ethical approval from IAEC, 34 adult male Sprague-Dawley rats weighing 150-200g aged 3 months were included and divided into Groups I, II, III and IV having 10, 10, 8 and 6 animals respectively. Pellets with 66% fructose was fed to 28 animals [Groups I, II, III] while 6 normal control animals [Group IV] were fed with standard rat chow diet for a period of 6 weeks. Later, for next 6 weeks, Groups I, II, III were treated with *M. charantia* 300 mg/kg/day, *M. charantia* 600mg/kg/day and Standard treatment (Metformin-180mg/kg/day + Telmisartan-2.5mg/kg/day + Rosuvastatin -2.5mg/kg/day) respectively. Serial measurements of body weight, BMI, fasting blood sugar were done at the baseline and every week thereafter, whereas noninvasive blood pressure, serum lipid profile, LDA, SOD and serum NF-κB were assessed at the base line, at the end of 6 weeks of fructose diet (i.e., following induction) and following treatment (12 weeks).Finally, at the end of the study 2 rats
from each of the groups were sacrificed for histopathological examination of liver and heart done.

RESULTS AND DISUSSION:
Paired-t-test was done for analyzing the paired data. One-way ANOVA and post hoc LSD were done to analyze the difference between groups. Following induction, there was statistically significant increase in body weight, BMI, FBS, serum triglycerides, total cholesterol, LDL, lipid derived aldehydes and serum NF-κB while there was reduction in HDL cholesterol and SOD with the exception of BP in comparison to baseline values. Following treatment all the treated groups had shown reversal of the parameters to the level of normalization, while low dose M.charantia 300mg/kg/day did not normalize FBS, serum triglycerides, total cholesterol, LDL, lipid derived aldehydes. Histopathological examination of liver and heart showed fatty infiltration in both these organs and hypertrophy of heart. Following treatment, there was a reduction in micro and macrovesicular steatosis.

CONCLUSION:
M.charantia 600mg/kg/day had exclusively matched the therapeutic efficacy of the standard therapy given in combination. The biological basis of the effects of M.charantia was by inhibiting the inflammatory pathway in metabolic syndrome both at the levels of ROS generation and transcription of pro-inflammatory marker NF-κB. Also, M.charantia is a potentially effective agent in the reversal of detectable pathological changes liver and heart.

KEY WORDS:
Metabolic syndrome, Momordica charantia, Serum NF-κB, Lipid derived aldehydes.