Potential role of Nicorandil, Telmisartan and Fenofibrate on hypertension, hyperlipidemia, insulin resistance, adiponectin, and metalloproteinase levels

in fructose-induced experimental cardiometabolic syndrome in rats.

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Abstract

The aim of the present work was to study the effect of nicorandil, telmisartan and fenofibrate on fructose - induced cardiometabolic syndrome in rats. Cardiometabolic syndrome was induced in male albino rats by 65% fructose in the drinking water orally for 5 weeks. Rats were divided into six groups; control group, fructose group, fructose and nicorandil group, fructose and telmisartan group, fructose fenofibrate group and fructose and the combined telmisartan and fenofibrate group. Nicorandil (2 mg/kg/day), Telmisartan (10 mg/kg day) and Fenofibrate (200 mg/kg/day) were administered orally daily for 5 weeks to study their effects on body weight, blood pressure, heart rate, serum glucose level, serum insulin, serum lipid profile, serum nitric oxide (NO), serum glutathione (GSH), serum superoxide dismutase (SOD), cardiac level of adiponectin and matrix metalloproteinase enzyme (MMP-9). Histopathological analysis of aorta and epidydimal fat were also performed. Results revealed that the three selected drugs significantly decrease the body weight, heart rate (HR), serum insulin, homeostasis model assessment index (HOMA), serum lipid profile and cardiac level of matrix metalloproteinase enzyme (MMP-9). On the other hand the drugs produced significant increase in high-density lipoprotein cholesterol (HDL-C), serum NO, serum glutathione, serum superoxide dismutase and cardiac level of adiponectin. Histopathological examination revealed that rats treated with nicorandil, telmisartan and fenofibrate show less infiltration, vacuolization and necrosis of aortic tissue and downsizing of adipocyte cells compared to fructose group.

<u>Key Words:</u> Metabolic syndrome, Fructose, Nicorandil, Telmisartan, Fenofibrate.