Journal of Diabetic Complications & Medicine
ISSN: 2475-3211
2020
Vol.5 No.4

Differential Expression of Vascular Dysfunction of Smaller Diameter Arteries in a Rodent Model of Metabolic Syndrome

* Ibrahim M. Salman*, Omar Z. Ameer & Aiman Y. Alwadi

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Alfaisal University, Riyadh, Saudi Arabia

Abstract

Metabolic syndrome, driven by obesity and diabetes, is a major contributor to cardiovascular disease. While large arteries vascular dysfunction is a well-documented phenomenon of metabolic syndrome, vascular disease of smaller diameter arteries, which are key contributors to peripheral vascular resistance and blood pressure control, remains uncertain. Using in-vitro organ-bath preparation, this study, therefore, investigated functional responses of the superior mesenteric and right iliac arteries in a high fat diet (HFD)/streptozotocin-induced diabetes mellitus rat model. Five-week-old male Wistar albino rats (n=24) were fed with either HFD (45 kcal% fat) or control diet (10 kcal% fat) for 10 weeks. On week 7, 40mg/kg streptozotocin and saline were injected intraperitoneally into the HFD and control groups, respectively. Diabetic HFD rats displayed a time-dependent increase ($p<0.01$) in water intake, urine output and fasting blood glucose. Both mesenteric and iliac vasoconstrictor responses (N/g) to norepinephrine (1×10-9–3×10-5M), but not to the depolarizing signals of high K+ (5–120 mM), were greater ($p<0.01$) in the HFD group relative to controls. Mesenteric, but not iliac, endothelium-dependent vasorelaxation to acetylcholine (1×10-10–1×10-5M) was blunted ($p<0.05$) in the HFD rats compared with controls. In contrast, mesenteric and iliac endothelium-independent vasorelaxation responses to sodium nitroprusside (1×10-11–1×10-6M) remained comparable between groups. In conclusion, vascular functional responses across smaller diameter arteries are differentially expressed in metabolic syndrome, demonstrating upregulated vasoconstriction to adrenergic stimuli and/or impaired endothelium-dependent relaxation. These vascular abnormalities align with those previously described in larger arteries and could therefore further promote the development of cardiovascular disease in metabolic syndrome..

Biography:

Dr. Salman received his PhD in Advanced Medicine from Macquarie University (Sydney, Australia) in 2015, and completed his postdoctoral training in the field of neurovascular regulation of cardiorespiratory and renal function at Case Western Reserve University's School of Medicine (Cleveland, Ohio, United States) in 2017. He is currently an Assistant professor of Physiology and Pharmacology, based in the College of Pharmacy of Alfaisal University (Riyadh, Saudi Arabia). With more than 12 years of research experiences, Dr Salman has contributed more than 50 peer-reviewed publications and has been serving as a reviewer for many leading medical journals in his field.

3rd International conference on Diabetes, Hypertension and Metabolic Syndrome; Webinar- July 02-03, 2020.

Abstract Citation:

Ibrahim M. Salman, Differential Expression of Vascular Dysfunction of Smaller Diameter Arteries in a Rodent Model of Metabolic Syndrome, Diabetes Meet 2020, 3rd International conference on Diabetes, Hypertension and Metabolic Syndrome; Webinar- July 02-03, 2020

(https://diabetesmeet.conferenceseries.com/abstract/2020/differential-expression-of-vascular-dysfunction-of-smaller-diameter-arteries-in-a-rodent-model-of-metabolic-syndrome)