MORPHOLOGICAL CHARACTERISTICS OF THE BLOOD VESSELS OF THE RENAL CORTEX IN EXPERIMENTAL METABOLIC SYNDROME

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ABSTRACT

This thesis is dedicated to the study of morphological characteristics of the renal cortical blood vessels under conditions of experimental metabolic syndrome. Using a rat model of metabolic syndrome induced through high-fat diet and fructose supplementation, structural changes in the afferent and efferent arterioles, glomerular capillaries, and peritubular capillary network of the renal cortex were assessed using histological, morphometric, and electron microscopic methods. The study provides new data on the early vascular remodeling processes in the kidney under metabolic dysfunction, with practical relevance for nephrology, pathological anatomy, and endocrinology.

KEYWORDS: metabolic syndrome; renal cortex; blood vessels; morphological characteristics; microcirculation; arterioles; glomerular capillaries; endothelial dysfunction; hyalinosis; basement membrane thickening; experimental model; morphometry; nephropathy.

INTRODUCTION

Metabolic syndrome (MetS) is one of the most clinically significant and rapidly spreading pathological conditions of the 21st century. According to WHO data, MetS affects 20–25% of the global adult population and is closely associated with the development of type 2 diabetes mellitus, cardiovascular diseases, and chronic kidney disease. The condition is characterized by a cluster of interrelated metabolic disturbances including central obesity, insulin resistance, hyperglycemia, arterial

hypertension, and dyslipidemia — all of which collectively impair the structural and functional integrity of the vascular system. The kidneys, given their rich microvascular architecture, are particularly vulnerable to metabolic dysregulation. The renal cortex contains a dense network of afferent and efferent arterioles, glomerular capillaries, and peritubular capillaries that are directly exposed to hemodynamic and metabolic stress under MetS conditions. However, despite the growing prevalence of MetS-associated nephropathy, the detailed morphological changes in renal cortical vessels remain insufficiently characterized.

Most existing research focuses on cardiac or endocrine complications of MetS, while the renal microvascular morphology has received comparatively less attention. This gap underscores the scientific and practical relevance of the present study, which aims to provide a comprehensive morphological characterization of renal cortical vessels in experimental metabolic syndrome.

MATERIALS AND METHODS

The study was conducted on 30 male Wistar rats divided into two groups: a control group (n=15, standard diet) and an experimental group (n=15, high-fat diet supplemented with 20% fructose solution for 12 weeks) to induce metabolic syndrome. Metabolic syndrome was confirmed by measuring body weight, blood glucose levels, insulin resistance (HOMA-IR index), serum lipid profile, and arterial blood pressure. After the experimental period, animals were euthanized under anesthesia, and renal tissue samples were collected and processed for histological examination. Serial sections (5–7 μm) were stained with hematoxylin and eosin (H&E), Masson's trichrome, and periodic acid–Schiff (PAS) to evaluate general tissue morphology, fibrosis, and basement membrane changes, respectively. Morphometric analysis was performed using an Olympus BX51 microscope with digital image analysis software (Image-Pro Plus 6.0). Transmission electron microscopy (TEM) was used to examine ultrastructural changes in endothelial cells and vascular basement membranes. All data

were analyzed statistically using SPSS 23.0; differences were considered significant at $p<0.05$.

RESULTS AND DISCUSSION

Animals in the experimental group demonstrated reliable signs of metabolic syndrome after 12 weeks: body weight increased by 35.2% compared to controls ($p<0.01$), fasting blood glucose was elevated to 8.4±0.6 mmol/L (control: 4.9±0.3 mmol/L), HOMA-IR index was significantly higher (5.8±0.4 vs. 1.9±0.2, $p<0.001$), and systolic blood pressure was raised (148±6 mmHg vs. 112±4 mmHg, $p<0.001$). Histological examination of the renal cortex revealed pronounced morphological changes in the microvascular bed of the MetS group. The afferent arterioles showed significant wall thickening with hyaline deposits replacing smooth muscle cells — a classic feature of hyaline arteriolosclerosis. The lumen of afferent arterioles was narrowed by an average of 42.3% compared to controls ($p<0.001$). Efferent arterioles also demonstrated wall thickening, though to a lesser degree (23.7% lumen reduction, $p<0.01$).

Glomerular capillaries exhibited mesangial matrix expansion and basement membrane thickening. PAS staining confirmed a 1.8-fold increase in basement membrane width compared to controls ($p<0.001$). Focal segmental glomerulosclerosis was detected in 38% of glomeruli in the MetS group. Peritubular capillaries showed rarefaction — a reduced capillary density of 31.6% compared to controls ($p<0.05$), accompanied by pericyte detachment. Electron microscopy revealed endothelial cell swelling, loss of fenestrations, subendothelial electron-dense deposits, and irregular thickening of the glomerular basement membrane. These ultrastructural findings indicate early endothelial dysfunction and impaired filtration barrier integrity. The observed changes are consistent with previously reported data on diabetic nephropathy but represent an earlier, metabolic syndrome-specific vascular remodeling pattern.

The morphological changes identified in this study are attributable to the combined effects of the major MetS components: insulin resistance promotes endothelial dysfunction through decreased nitric oxide bioavailability; chronic hyperglycemia triggers advanced glycation end-product (AGE) formation, contributing to basement membrane thickening; arterial hypertension induces mechanical shear stress on vessel walls promoting hyalinosis; and dyslipidemia with elevated LDL and triglycerides accelerates oxidative stress and lipid peroxidation in vascular endothelium. These findings highlight the complex, multifactorial pathogenesis of MetS-associated nephrovascular damage.

CONCLUSION

Experimental metabolic syndrome induces significant and progressive morphological changes in the blood vessels of the renal cortex. Key findings include afferent arteriole hyaline arteriolosclerosis with luminal narrowing, glomerular basement membrane thickening, mesangial expansion, focal segmental glomerulosclerosis, endothelial dysfunction at the ultrastructural level, and peritubular capillary rarefaction. These changes collectively impair renal microcirculation and represent an important early morphological substrate for MetS-associated nephropathy. The results of this study contribute to a deeper understanding of the pathogenesis of renal vascular damage in metabolic syndrome and provide a morphological basis for early diagnosis of nephropathy. The data obtained have practical value for nephrology, pathological anatomy, endocrinology, and morphology, and may serve as a foundation for developing targeted preventive and therapeutic strategies against MetS-related renal complications.

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