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VASCULAR MORPHOMETRIC REMODELING IN METABOLIC SYNDROME: STRUCTURAL ALTERATIONS OF LARGE AND SMALL BLOOD VESSELS ASSOCIATED WITH OBESITY AND INSULIN RESISTANCE

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Abstract. Metabolic syndrome is characterized by a combination of central obesity, insulin resistance, dyslipidemia, arterial hypertension, and impaired glucose metabolism. In addition to functional endothelial dysfunction, these metabolic abnormalities are associated with progressive structural remodeling of both large arteries and microvascular vessels. The aim of this thesis is to summarize the principal morphometric changes in large and small blood vessels associated with obesity and insulin resistance. Current experimental and clinical data indicate that metabolic syndrome is accompanied by increased intima-media thickness, medial hypertrophy, adventitial remodeling, extracellular matrix accumulation, and increased arterial stiffness. In small resistance arteries, hypertrophic remodeling is commonly observed, characterized by increased wall thickness and wall-to-lumen ratio with relative reduction of the vascular lumen. At the microcirculatory level, obesity and insulin resistance are associated with impaired capillary recruitment, reduced capillary density, and structural microvascular rarefaction. These alterations may impair tissue perfusion and contribute to the progression of insulin resistance, hypertension, and target-organ damage.

The pathogenesis of vascular remodeling in metabolic syndrome is multifactorial. Chronic low-grade inflammation, oxidative stress, reduced nitric oxide bioavailability, compensatory hyperinsulinemia, activation of the renin–angiotensin–aldosterone system, sympathetic hyperactivity, adipokine imbalance, and dysfunction of perivascular adipose tissue all contribute to structural changes in the vascular wall. Endothelial dysfunction is considered an early event, while persistent metabolic and hemodynamic stress promotes vascular smooth muscle hypertrophy, extracellular matrix deposition, and progressive narrowing of resistance vessels. Large arteries demonstrate increased intima-media thickness and stiffness, whereas the microvascular network may undergo both functional and structural rarefaction.

From a morphological perspective, several parameters are particularly informative for evaluating vascular injury in metabolic syndrome: intima-media thickness, total vascular wall thickness, medial thickness, lumen diameter, wall-to-lumen ratio, medial cross-sectional area, adventitial thickness, and capillary or microvessel density. Combined assessment of these parameters allows simultaneous characterization of macrovascular remodeling and microvascular rarefaction.

Keywords: metabolic syndrome, obesity, insulin resistance, vascular remodeling, morphometry, intima-media thickness, wall-to-lumen ratio, microcirculation, capillary rarefaction.

Conclusion. Obesity and insulin resistance induce coordinated structural changes throughout the vascular system. Large-vessel remodeling is characterized by wall thickening, increased intima-media thickness, fibrosis, and stiffness, whereas small arteries demonstrate hypertrophic remodeling and

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increased wall-to-lumen ratio. At the microvascular level, reduced capillary density and structural rarefaction may further aggravate tissue hypoperfusion and metabolic dysfunction. Comprehensive histomorphometric assessment of large and small vessels may therefore serve as an objective morphological approach for evaluating vascular damage associated with metabolic syndrome.

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