MORPHOLOGICAL AND IMMUNOHISTOCHEMICAL FEATURES OF ENDOTHELIAL DYSFUNCTION IN DIABETIC ANGIOPATHY ASSOCIATED WITH POST-COVID-19 SYNDROME

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Abstract

Diabetic angiopathy is characterized by progressive damage to the vascular endothelium, impaired microcirculation, and structural remodeling of blood vessel walls. Persistent hyperglycemia contributes to oxidative stress, endothelial dysfunction, and thickening of capillary basement membranes. Following SARS-CoV-2 infection, prolonged inflammatory responses and disturbances in vascular regulation may further aggravate pre-existing diabetic vascular abnormalities. Therefore, investigating the morphological and immunohistochemical manifestations of endothelial dysfunction in patients with diabetic angiopathy and post-COVID-19 syndrome is of considerable scientific interest.

Objective. To analyze the morphological and immunohistochemical features of endothelial dysfunction in diabetic angiopathy associated with post-COVID-19 syndrome, with particular attention to vascular remodeling and microcirculatory abnormalities.

Materials and Methods. A comparative analysis of five published scientific studies concerning diabetic microangiopathy, post-COVID-19 endothelial dysfunction, and vascular histopathology was performed. The assessment focused on endothelial integrity, vascular wall remodeling, capillary basement membrane alterations, and

microvascular thrombosis. The potential diagnostic significance of CD31, CD34, von Willebrand factor (vWF), and vascular endothelial growth factor (VEGF) immunohistochemical markers was also considered.

Results and Discussion. Published evidence demonstrates that diabetic angiopathy is associated with capillary basement membrane thickening, reduced capillary density, and narrowing of vascular lumina. Fiordaliso et al. reported a 22.2% reduction in dermal capillary density in patients with diabetic foot ulcers compared with non-diabetic controls.

Histopathological investigations of acute COVID-19 have demonstrated endothelial injury, inflammatory infiltration, microvascular thrombosis, and abnormal angiogenesis. Ackermann et al. identified extensive pulmonary endothelial damage and reported that alveolar capillary microthrombi were approximately nine times more prevalent in COVID-19 than in influenza-associated lung injury [4].

Studies of post-COVID-19 syndrome have also demonstrated persistent endothelial dysfunction. Kuchler et al. reported reduced venular flicker-induced dilation in patients with post-COVID-19 syndrome compared with healthy controls ($3.42 \pm 1.77\%$ versus $4.64 \pm 2.59\%$; $p = 0.02$), suggesting prolonged microvascular dysfunction following infection.

From an immunohistochemical perspective, CD31 and CD34 may be used to assess endothelial distribution and microvascular density, while vWF may provide information about endothelial phenotype and vascular injury. VEGF expression may help characterize angiogenic responses. However, these markers require direct investigation in lower-extremity vascular tissues before specific expression patterns can be attributed to post-COVID-19 diabetic angiopathy.

Conclusion. Diabetic angiopathy and post-COVID-19 syndrome share mechanisms involving endothelial dysfunction, oxidative stress, inflammation, and disturbances in microvascular regulation. Existing morphological evidence supports

the importance of endothelial injury and vascular remodeling in both conditions. Further histological and immunohistochemical investigations of lower-extremity vessels are necessary to establish the specific contribution of post-COVID-19 syndrome to the progression of diabetic angiopathy.

Keywords: post-COVID-19 syndrome, diabetic angiopathy, endothelial dysfunction, immunohistochemistry, CD31, CD34, VEGF, microcirculation, vascular remodeling.

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