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MORPHOLOGICAL CHANGES IN THE DIABETES MELLITUS IN COVID-19 PATIENTS

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COVID-19, caused by the SARS-CoV-2 virus, continues to have significant global health implications. Among the most vulnerable populations are patients with pre-existing metabolic disorders, particularly type 2 diabetes mellitus (T2D). Autopsy-based investigations and immunophenotypic studies highlight several distinctive morphological and immunological changes in diabetic patients with COVID-19, emphasizing a hyperinflammatory stage, vascular injury, and altered immune cell morphology.

One of the most prominent morphological findings in COVID-19 patients with T2D is the presence of widespread microthrombi, most commonly observed in the lungs, heart, kidneys, and liver. Microvascular thrombosis, a hallmark of severe COVID-19, is significantly more prevalent in individuals with comorbid diabetes, indicating a synergy between hyperglycemia-induced endothelial dysfunction and SARS-CoV-2-induced coagulopathy. Liver histology from autopsies often reveals steatosis (macro- and microvesicular), centrilobular congestion with microthrombi, and focal necrosis. Autopsy findings also emphasize the pulmonary involvement in diabetic patients. Diffuse alveolar damage, pulmonary embolism, and widespread intra-alveolar and capillary microthrombi are frequent. In certain cases, bilateral interstitial pneumonia and pulmonary infarctions were observed. Histologically, hyaline membranes, desquamated pneumocytes, and hyperplastic alveolar epithelium reflect severe viral-induced injury worsened by diabetic endothelial dysfunction. Kidney histopathological findings frequently included acute tubular injury, accompanied by glomerular microthrombi, indicating renal impairment related to both COVID-19 and underlying diabetes. In some cases, pre-existing diabetic damage is exacerbated by ischemic injury from SARS-CoV-2 infection. Cardiac tissue in T2D-COVID-19 patients shows evidence of myocardial fibrosis, microthrombi, and, occasionally, myocarditis. The vascular component of this damage may be partly due to enhanced susceptibility to endothelial injury in diabetics, compounded by SARS-CoV-2's tropism for ACE2-expressing cells.

Conclusively, COVID-19 exacerbates disease severity in diabetic patients by increasing microvascular damage, immune dysfunction, and metabolic stress, primarily through IRF5-driven inflammatory pathways. Additionally, further research is needed to explore the potential forCOVID-19 to induce new-onset diabetes via direct pancreatic effects. Diabetic patients show distinct changes, such as microvascular thrombosis and lung injury, suggesting that pre-existing metabolic issues amplify SARS-CoV-2 pathology. However, whether these outcomes result from direct viral cytopathic effects or systemic metabolic-immune dysregulation remains an open question warranting further exploration.