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DIAGNOSIS OF THROMBOTIC MICROANGIOPATHY IN KIDNEY BIOPSY SPECIMENS

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Relevance. Thrombotic Microangiopathy (TMA) is a pathological process that is characterized by microvascular thrombosis, vessel wall thickening, and endothelial cell swelling, resulting in thrombocytopenia, ischemic organ damage and particularly acute kidney injury. TMA represents a vast spectrum of disorders, from primary syndromes such as atypical hemolytic uremic syndrome (aHUS) and thrombotic thrombocytopenic purpura to secondary forms associated with malignant hypertension, systemic sclerosis, the postpartum period and infection. TMA has an annual incidence of 5.9-14.4 cases per million persons per year.

Aim: to characterise the renal histopathological features of TMA across various biopsy samples and correlate these findings with clinical data.

Material and methods. Analysis of 14 kidney biopsies from patients diagnosed with TMA were obtained. Biopsies were obtained via ultrasound-guided percutaneous needle biopsy, fixed in 10% formalin, paraffin embedded and sectioned at 3μm. These sections were stained with hematoxylin and eosin (H&E), periodic acid-Schiff (PAS), Masson's trichrome, and Jones silver stain for histopathological evaluation. Slides were examined under light microscopy. Immunofluorescence was performed to assess immune deposits (IgA, IgG, IgM, C3, C1q). Clinical data was obtained from referral charts.

Results and their discussion. The study included 14 patients, 7 males (50%) and 7 females (50%). (M:F 1:1). The age of patients ranged from 23 to 59 years. The mean age was 38 ± 10.3 years. Whilst examining histopathology, all cases exhibited features that are consistent with TMA, such as endothelial swelling, microthrombi, focal segmental glomerulosclerosis and vascular sclerosis. However, clinical presentations did vary. Two female patients (14.3%) presented with postpartum TMA and complications which include placental disruption, aHUS and disseminated intravascular coagulation syndrome. Histopathology revealed acute changes such as microthrombi, acute tubular necrosis and glomerular necrosis. Three male patients (21.4%) presented with malignant hypertension. Histopathology revealed characteristics that would suggest malignant hypertension due to vascular sclerosis, fibrointimal proliferation along with variable glomerular ischemia and segmental sclerosis. Two other patients (21.4%) presented with severe hypertension but also systemic sclerosis. These cases showed chronic TMA changes with significant vascular sclerosis and interstitial fibrosis.

Conclusions. A detailed analysis of vascular, glomerular, and interstitial changes is vital for distinguishing primary TMA forms, such as aHUS, from secondary forms, leading to targeted and specific therapy but also to predict long-term renal outcomes.