Thrombotic Microangiopathy Update

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Abstract

Thrombotic microangiopathy (TMA) is a manifestation of endothelial cell injury that is associated with many different clinicopathologic settings, which seem disparate at first glance. Features of active TMA include endothelial cell swelling and loss of fenestrations, but these can also be observed in tissues that are not optimally processed or handled. If sampled, red blood cell fragments and the fibrin tactoids of thrombi may be seen by electron microscopy. Detachment of endothelial cells from the glomerular basement membrane (GBM) is a more robust early pathologic feature of TMA and duplication or multilayering of the GBM indicates chronicity or repetitive microangiopathic episodes. Mesangiolysis may represent an injury somewhere between acute and chronic. Multilayering of peritubular capillary basement membranes has also been observed in native kidneys, but has been much less studied and underappreciated compared with kidney allografts. The overactivation of the complement cascade is emerging as a major contributor of TMA, and we will discuss the common thread that links this mechanism to autoimmunity, pregnancy, and transplantation.

References

1. #NephMadness 2019: Complement – Missing the Forest for the Trees – AJKD Blog
2. Gallan AJ, Chang A. A new paradigm for renal thrombotic microangiopathy. Sem Diag Pathol 2020, 37:121-126. PMID: 32085935