Novel developments in thrombotic microangiopathies: is there a common link between hemolytic uremic syndrome and thrombotic thrombocytic purpura?

Zipfel PF, Wolf G, John U, Kentouche K, Skerka C (2011) Novel developments in thrombotic microangiopathies: is there a common link between hemolytic uremic syndrome and thrombotic thrombocytic purpura? *Pediatr Nephrol* 26(11), 1947-1956.

Abstract

Thrombotic microangiopathies (TMA) represent a spectrum of related disorders associated with newly formed thrombi that block perfusion and thus affect the function of either renal or neurological organs and tissue. Recent years have seen a dramatic development in the field of TMA and for the two major forms hemolytic uremic syndrome (HUS) and thrombocytopenic purpura (TTP), new genetic causes and also autoimmune forms have been identified. This development indicates a similar pathophysiology and suggests that the two acute disorders are based on common principles. HUS is primarily a kidney disease and TTP also develops in the kidney and at neurological sites. In HUS thrombi formation is likely due to a deregulated complement activation and inappropriate platelet activity. In TTP thrombi formation occurs because of inappropriate processing of released multimers of von Willebrand Factor (vWF). Defining both the similarities and the unique features of each disorder will open up new ways and concepts that are relevant for diagnosis, for therapy, and for the prognostic outcome of kidney

transplantations. Here we summarize the most relevant topics and timely issues that were presented and discussed at the 4th International Workshop on Thrombotic Microangiopathies held in Weimar in October 2009 (www.hus-ttp.de).

Beteiligte Forschungseinheiten

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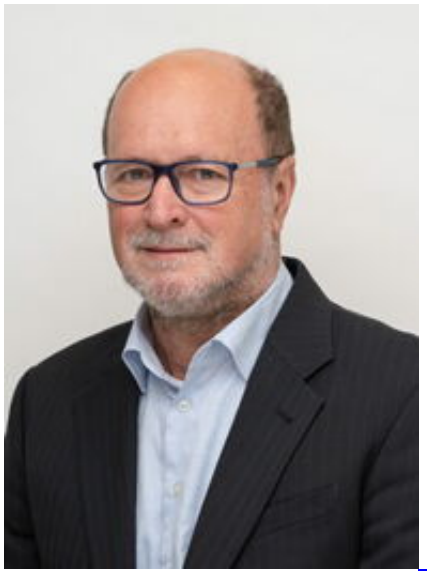
Details



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Identifier

doi: 10.1007/s00467-011-1923-9

PMID: 21671028