The syndromes of thrombotic microangiopathy

Propositions belonging to the thesis

The syndromes of thrombotic microangiopathy: towards a true etiology–based approach

TMAs cannot be excluded based solely on “normal” hematologic indices without a (kidney) biopsy. (This thesis.)

Massive *ex vivo* C5b9 formation on the resting and activated endothelium can be used to detect acute TMA and risk for C–TMA, respectively. (This thesis.)

Ex vivo C5b9 formation on the endothelium and genotyping categorizes TMAs into different groups, with therapeutic and prognostic implications. (This thesis.)

The atypical HUS type classification is not absolute; the term primary atypical HUS should be replaced by C–TMA. (This thesis.)

Pregnancy is a critical condition in women at risk for C–TMA, but the risks appear lower than formerly believed. (This thesis.)

Recent advances improved the diagnosis, treatment, and prognosis of C–TMA, but the optimal dosing and duration of treatment remains to be established. (Personal note.)

With the current state of knowledge and availability of therapeutic complement inhibition, the central consideration in the management of complement–mediated diseases is the identification of patients who would benefit from such therapies. (Per–sonal note.)

The etiology/pathogenesis–based classification should form the basis of the kidney biopsy report. (Sethi *et al*. J Am Soc Nephrol, 2016.)

Guidelines are not intended to define a standard of care and should not be interpreted as prescribing an exclusive course of management; clinicians should decide how to apply them. (Personal opinion.)

Blind belief in authority is the greatest enemy of truth. (Albert Einstein.)

At times like this, a Dr Pepper helps. (Fats, the house of God.)