

PATHOLOGY OF THROMBOTIC MICROANGIOPATHY

Terry Cook
Imperial College London

Disclosure of Interests

- GlaxoSmithKline consultancy
- Biogen Idec consultancy
- Achillion Pharmaceuticals consultancy



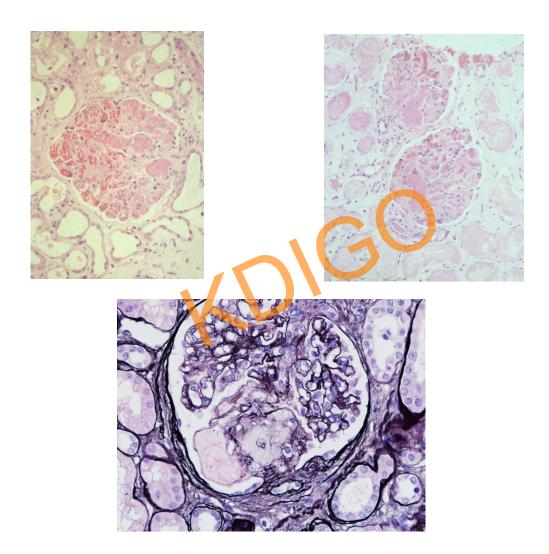
TMA - morphology

Range of morphological changes in the kidney vasculature in response to endothelial injury

May involve glomeruli, arterioles and arteries

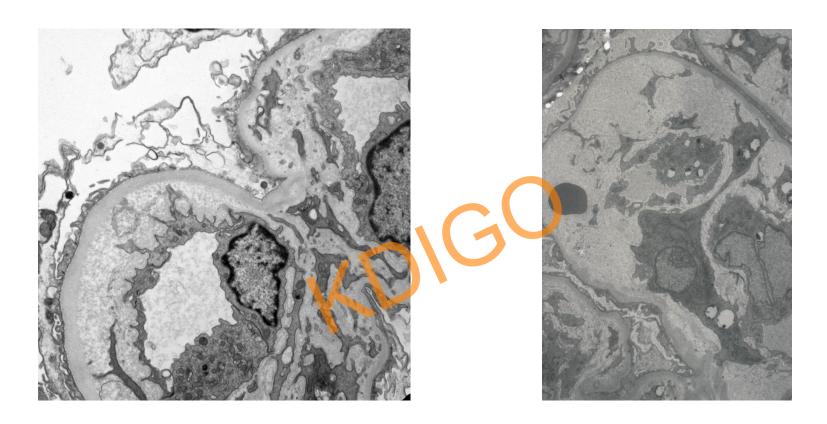


Glomeruli – acute lesions



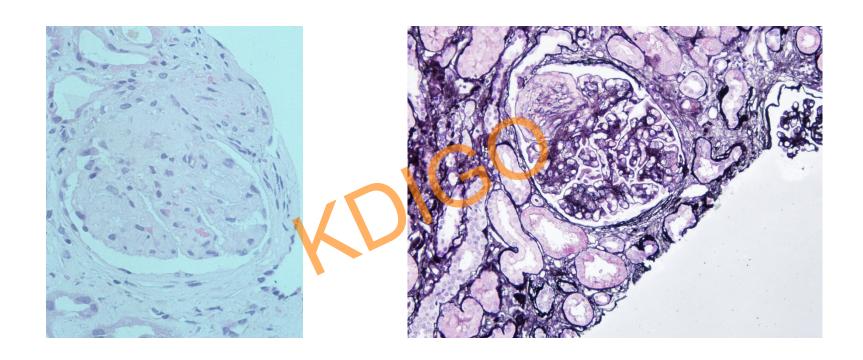


Glomeruli – acute lesions



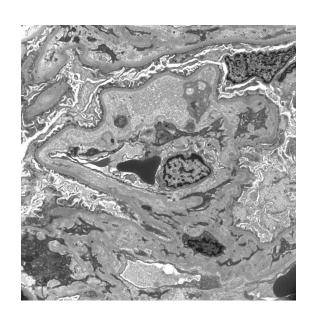


Glomeruli- chronic lesions

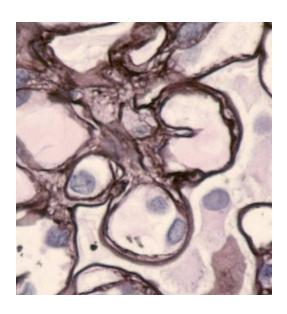




Glomeruli- chronic lesions

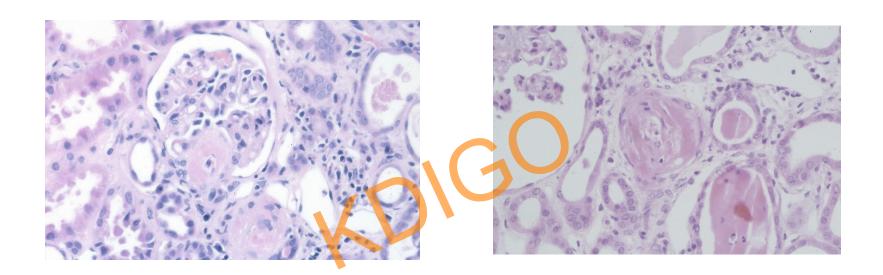






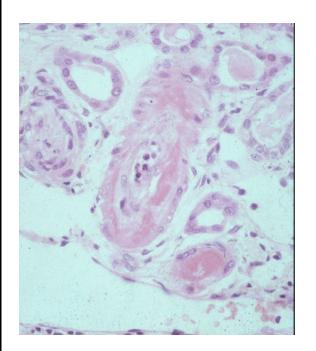


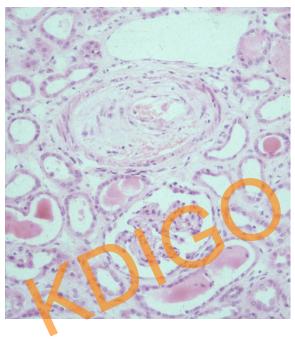
Arterioles

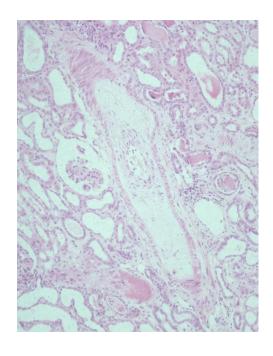


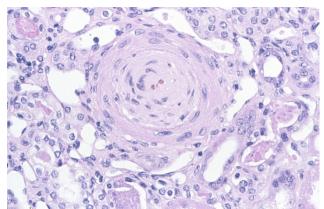


Arteries











• 1. Nomenclature of TMA: is it a problem that TMA encompasses changes that are not 'thrombotic' (e.g., glomerular subendothelial expansion, mesangiolysis and myxoid arterial intimal thickening)?



 2. What are the morphological differences between TMAs of different etiology?





2. What are the morphological differences between TMAs of different etiology?

- Shiga toxin associated HUS shows only glomerular involvement
- TTP has platelet rich thrombi
- TMA due to malignant hypertension shows predominant vascular involvement
- Does C5b-9 staining differentiate causes due to primary complement activation?



 3. What is the distinction between TMA associated with malignant hypertension as opposed to TMA due to other causes with secondary hypertension?



3. What is the distinction between TMA associated with malignant hypertension as opposed to TMA due to other

causes with secondary hypertension?

- Possibly more chronic vascular changes in TMA due to malignant hypertension
- Can C5b-9 staining help?



- 4. What are the morphological features of acute and chronic TMA lesions?
- Is there an entity of chronic TMA due to complement abnormalities that leads to chronic renal impairment without acute episodes?

