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Renal pathologic findings and clinical associations in patients with infective endocarditis

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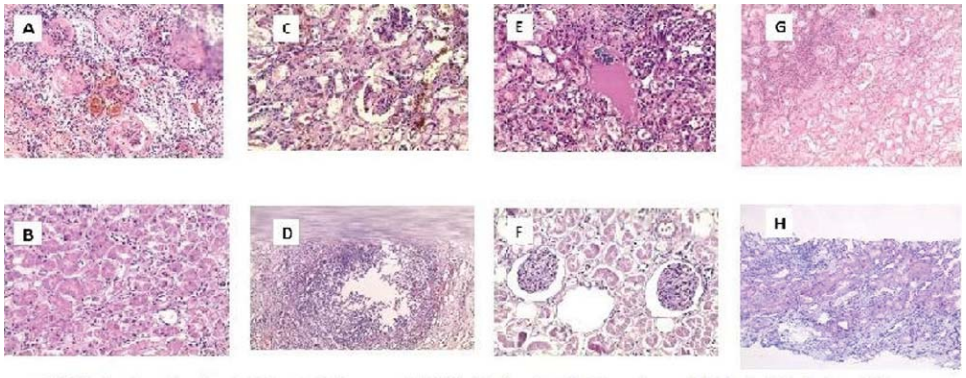
Background: Kidney involvement in infective endocarditis (IE) has a huge contribution in failure of the antimicrobal therapy and in rejection of the surgical treatment. Frequency of kidney lesions is still high and is diagnosed in 50–80% cases intravitaly, and in 91,6% post-mortem.
Materials/Methods: 28 patients with verified IE (DUKE 2009, 2015), hospitalized and treated in clinical hospital from 2010 to 2018, were included in the study. Kidney function was assessed using CKD-EPI formula. Acute kidney injury (AKI) was diagnosed according to current guidelines (KDIGO 2012). Intravital nephrobiopsy was performed in 2 (7,1%) patients, in 26 patients (92,9%) morphological assessment was made on the autopsy tissue specimens. Autopsy material was assessed grossly and microscopically with H&E staining. Morphological changes in kidney were estimated based on standart histological criteria.
Results: Majority of patients with IE (92.9%) had structural changes in kidney tissue: 3 (12%), 16 (64%) and 6 (24%) patients had respectively isolated glomerular, tubular and mixed lesions. Significantly higher is rate of tubular lesion than glomerular (p=0.014). Main pattern in glomerular damage was mesangial proliferation, we didn't found any cases of crescentic GN. Herewith, proliferation was mainly diffuse (87.5%) rather than focal (12.5%). Glomerular damage in subacute IE (>56 days) appears more often than in acute IE (<56 days) (p=0.057 χ^2=3.63). When studying the influence of various factors on the involvement of the glomeruli or tubules, no statistically significant group differences were obtained, except for the gram-negative flora as the causative agent, in which the tubules are significantly more likely to be affected (p=0.019)
Conclusions: A morphological study of patients with IE revealed a wide range of kidney damage, however, the frequency of tubular lesions, mediated by nephrotoxic drugs and hemodynamic disorders significantly exceeds the frequency of immune complex glomerular lesions, as previously thought.

Morphological diagnosis	n (%)	Morphological diagnosis	n (%)
Glomerulonephritis	8 (28.6)	Tubulointerstitial nephritis	3 (10.7)
Membranoproliferative glomerulonephritis	3 (10.7)	Allegrgic tubulointerstitial nephritis	2 (7.2)
Mesangioproliferative glomerulonephritis	5 (17.9)	Infective tubulointerstitial nephritis	1 (3.6)
Extracapillar crescentic glomerulonephritis	0 (0)	Kidney abscess	3 (10.7)
Acute tubular necrosis	22 (78.6)	Kidney infarction	1 (3.6)
Acute tubular necrosis (ischaemic)	11 (39.3)	Renal artery embolism	1 (3.6)
Acute tubular necrosis (toxic)	11 (39.3)	Shock kidney	4 (14.3)



A) Diabetic glomerulosclerosis B) Acute tubular necrosis C) Emboli in the artery D) Kidney abscess E) Allergical tubulo-interstitial nephritis F) Mesangioproliferative glomerulonephritis G) Kidney infarction H) Acute tubular necrosis (intravital biopsy)