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ASSOCIATION BETWEEN CAUSATIVE MICROORGANISMS OF INFECTIVE ENDOCARDITIS AND CLINICAL SPECTRUM OF RENAL LESIONS

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Background and Aims: The relation between microorganisms and renal lesions in IE has bilateral nature: urinary tract infections may be source of systemic invasion that will cause IE, IE causative microorganisms may cause inflammatory changes in glomeruli or be disseminated in renal parenchyma hematogenously with the formation of the abscesses in case of fragmentation or migration of the vegetations. IE-associated GN - is an immune-mediated GN caused by bacterial infection. More commonly GN presented in cases of streptococcal and staphylococcal IE. For renal embolism is typical association with staphylococcal infection, and there are documented cases of renal infarctions in IE caused by S. pneumoniae, and C. burnetii. The effect of microorganisms on other clinical manifestations of kidney damage in IE has not been studied.

Method: The study included 209 patients with verified IE. All patients were performed bacteriological and 81 (38,8%) parallel one-moment biomolecular (PCR or PCR with follow-up sequencing) tests. In case of death or cardiac surgery, affected valves (n=15) were tested with the same technique. To assess renal function we calculated glomerular filtration rate with CKD-EPI formula. AKI, AKD and CKD were diagnosed in accordance with present guidelines [KDIGO, 2012].

Results: Renal involvement into the pathological process trended higher in patients with Gr- (p=0,016) and few causative microorganisms (p=0,039). There are significant differences in incidence of different renal syndromes in patients with MRSA, Gr- and few causative microorganisms - these patients more often had acute nephritic syndrome (respectively p=0,036; p=0,005; p=0,003) (tab. 1). Impairment in the renal function was significantly associated with MRSA (p=0,012), Gr- (p=0,01) and few causative microorganisms (p=0,003). In addition long-lasting renal impairment was observed: patients with MRSA had median time of worsening of the renal function 9 days (IQR 4-14), with Gram-negative microorganisms median time was 9 days (IQR 5-12), with polyflora 10 days (IQR 7-14). But there were no significant differences in median time of renal function worsening in patients with other causative microorganisms.

Incidence of AKI and AKD depending on causative organism did not significantly differ.

Conclusion: Kidney lesions in IE significantly associated with Gr- microorganisms, MRSA and with the presence of 2 or more causative microorganisms. Clinical spectrum of renal lesions is diverse: from the changes in the urine sediment to longstanding kidney dysfunction.

Table 1. Incidence of renal syndromes depending on the causative agent of IE (n=160).

Causative microorganism	Urinary syndrome (n=105)	Acute nephritic syndrome (n=40)	Nephrotic syndrome (n=15)	χ^2
Gr+ microorganisms (n=140), n(%)	75(71,4)	31(77,5)	7(46,7)	8,9*
Staphylococcus (n=96), n(%)	46(43,8)	23(57,5)	6(40,0)	
S. aureus (n=72), n(%)	34(32,4)	18(45,0)	5(33,3)	
MRSA (n=51), n(%)	28(26,7)	9(22,5)	3(20,0)	
MRSA (n=16), n(%)	4(3,8)	7(17,5)	2(13,3)	8,5*
CoNS (n=25), n(%)	12(11,4)	7(17,5)	2(13,3)	
Streptococcus (n=11), n(%)	6(5,7)	3(7,5)	0(0,0)	
Enterococcus (n=37), n(%)	23(21,9)	9(22,5)	0(0,0)	7,0
Gr- microorganisms (n=40), n(%)	17(16,2)	14(35,0)	5(33,3)	12,9**
Fungi (n=8), n(%)	2(1,9)	3(7,5)	1(6,7)	
Few causative microorganisms (n=58), n(%)	25(23,8)	20(50,0)	5(33,3)	14,1**

*p<0,05, **p<0,01, ***p<0,001 – significance of the differences between the groups.

Figure: