C5b-C9 MEMBRANE ATTACK COMPLEX PLASMATIC LEVELS IN DIFFERENT PATTERNS OF ACUTE KIDNEY INJURY

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BACKGROUND

Complement system is involved in the patophysiology of several kidney diseases.

The final component of complement system, membrane attack complex (MAC or C5b-C9) has been demonstrated to be releated with Membranous nephropaty and animal models of ischemia-reperfusion among others.

AIM

To assess if after injury, complement is activated leading to the production of proinflammatory cytokines such interleukin-6 generating neutrophil recruitment with NGAL (Neutrophil Gelatinase-Associated Lipocalin) release.

METHODS

77 patients in a tertiary hospital classified according to different AKI patterns:

1) Septic (n=26, 47% AKI)
2) Ischemia-reperfusion (renal transplants, n=23, 70% AKI)
3) Nephrotoxic pattern (patients under colistin treatment, n=15, 46% AKI)
4) Multifactorial model of AKI (n=13, 50% AKI)

Overall, 61% (n=47) had AKI and 38% (n=30) normal renal function.

Samples were tested for IL-6 and MAC using ELISA kit (R&D Systems®) and NGAL were tested by means to inmunofluorescence assay (Alere®)

RESULTS

Plasmatic MAC level was statistically different in patients with AKI as compared to normal renal function controls, regardless of the etiology of AKI (501±247 mAU/mL vs 388±150 mAU/mL; *p* 0.015).

Plasmatic IL-6 levels were significant higher in AKI patients compared to normal kidney function (10,47±2,8 pg/mL vs 7,37±3,0 pg/mL *p*=0,02)

NGAL levels were also significantly higher in AKI patients (570,5±305 ng/mL vs 292,5±233 ng/mL *p*< 0,001).

No relevant differences in the three biomarkers were detected in the different etiological subgroups.

	AKI	Non-AKI	*p*
MAC	**501 ±247** mAU/mL	388 ±150 mAU/mL	0.015
IL-6	**10,47 ±2,8** pg/mL	7,37 ±3 pg/mL	0.02
NGAL	**570,5 ±305** ng/mL	292,5 ±233 ng/mL	<0.001

CONCLUSIONS

Our data show that in AKI, regardless etiology, the complement system is activated and could lead pro-inflammatory cytokine stimulation (IL-6) and could produce releasing of NGAL from neutrophils.

REFERENCES

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