AKI IN COVID-19 PATIENTS

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Background: Several clinical studies have reported that in the course of COVID-19 kidney involvement is not a rarity. In this contest we analysed the blood tests at the hospital admission as potential predictors of AKI in COVID-19 patients.

Methods: The study population encompassed all the 105 patients admitted at the COVID Unit of the A.O.U. Policlinico “G.Martino” between March 8, 2020 and April 27, 2020 , with the diagnosis of SARS-CoV2 infection

Results: Data shown that in our population 33 of 105 patients (29.5%) developed AKI during their hospitalization. The median of AKI onset is 5 days [5.0-6.75]. The maximum timing of development of AKI compared to hospital entrance is 26 days. Table 1 reports the characteristics of the population divided into 2 groups: AKI and No AKI. Kaplan-Meier analysis shown an increase of mortality in patients that developed AKI (p<0.001).

	No AKI	AKI	p
n	74	31	
Sex, n (%)			0.841
Age, median (min-max)	69 (18-100)	86 (42-92)	<0.001
Chronic kidney disease, n (%)	7 (9.46%)	10 (32.26%)	0.004
Hypertension, n (%)	31 (41.89%)	14 (45.16%)	0.757
Diabetes, n (%)	16 (21.62%)	8 (25.81%)	0.641
Chronic Obstructive bronchopathy, n (%)	2 (2.70%)	5 (16.13%)	0.012
Cardiopathy, n (%)	14 (18.92%)	10 (32.26%)	0.138
Neoplasia, n (%)	4 (5.40%)	1 (3.23%)	0.632
Hemoglobin, (g%)	13.1 (7.4-16.7)	11.8 (8.4-16.9)	0.092
White blood cells (WBC), (mmc)	5700 (2600-13100)	6400 (2200-21200)	0.146
Hematocrit (Hct), (%)	39 (25-50)	37 (26-52)	0.237
Platelet, (mmc)	177000 (30000-443000)	189000 (87000-378000)	0.730
D-dimer, (µg/mL)	0.7 (0.27-4.01)	1.24 (0.31-4.01)	0.011
Creatinine (mg/dL),	0.8 (0.3-2.9)	1.2 (0.4-10.9)	0.003
Azotemia, (mg/dL)	35 (10-117)	62 (18-341)	<0.001
LDH, (U/L)	357 (222-907)	388 (253-1079)	0.249
CPK, (U/L)	64.5 (10-5450)	55 (14-4118)	0.569
Sodium, (mmol/L)	139 (131-153)	144 (136-173)	<0.001
Potassium, (mmol/L)	4.3 (3-5.2)	4.3 (2.7-6.1)	0.626
Calcium, (mg/dl)	8.575 (7.390-9.6)	8.5 (7.1-10)	0.434
Myoglobin, (ng/ml)	40 (21-664)	113.5 (30-15000)	<0.001
PCR (mg/dl)	1.5 (0.05-20.8)	3.650 (0.1-30.63)	0.005

We have hypothesized that at the base of the alterations found there was an unbalance of the RAAS. Higher levels of angiotensin II and lower levels of angiotensin 1-7 could be responsible of oxidative stress damage. In addition patients with AKI have higher myoglobin value. Increased myoglobin could be a trigger for renal injury from angiotensin II.

Conclusion: Further studies are also needed to confirm or disprove the possible role of increased angiotensin II and reduction of angiotensin (1-7) in pathogenesis from COVID-19.