Table 1. Analytical evolution of the patients included in the study. ANOVA test for independent samples

Analytical parameters (SD, 95% CI)	Total	KDIGO 1	KDIGO 2	KDIGO 3	P value
Baseline creatinine	1.22 (0.71, 1.03-1.37)	1.56 (1.24, 0.67-2.45)	1.01 (0.27, 0.88-1.15)	1.19 (0.68, 0.98-1.40)	0.46
Admission creatinine	1.84 (1.68, 1.45-2.24)	1.97 (1.33, 1.01-2.92)	1.58 (0.74, 1.21-1.96)	1.92 (2.01, 1.31-2.53)	0.49
Maximum creatinine	1.82 (2.03, 3.34-4.3)	2.17 (1.44, 1.13-3.20)	2.40 (0.84, 1.98-2.82)	4.77 (1.91, 1.05-1.77)	< **0.0001**
Discharge or death creatinine	1.14 (1.03, 1.13-1.62)	1.70 (0.89, 1.06-2.34)	1.11 (0.63, 0.80-1.42)	1.41 (1.18, 1.05-1.77)	0.12
Previous/ discharge or death creatinine difference	0.17 (0.68, 0.12-0.33)	0.14 (-0.63, 0.20-0.48)	0.09 (0.63, -0.21-0.41)	0.21 (0.75, -0.11-0.44)	**0.03**
Posthospitalization creatinine	1.35 (0.82, 1.16-1.55)	1.59 (0.99, 0.88-2.30)	1.14 (0.52, 0.88-1.40)	1.39 (0.87, 1.12-1.65)	0.44
Baseline/posthospitalization creatinine difference	0.15 (0.44, 0.95-0.26)	0.02 (0.42, -0.27-0.32)	0.12 (0.51, -0.12-0.37)	0.19 (0.41, 0.07-0.32)	0.61

with 57% in the prerenal group ($P < .001$). Patients with prerenal AKI had greater comorbidity. On the other hand, patients with ATN AKI developed more serious COVID-19 infection: higher percentage of severe pneumonia, admission to the intensive care unit and need for orotracheal intubation. The analytical parameters were more extreme in patients with ATN AKI, except for creatinine and urea upon admission, which were higher in the prerenal AKI group.
A total of 89 patients died during the study; 65% of ATN AKI patients versus 31% of prerenal-AKI patients ($P < .001$). The ATN was a mortality risk factor, whit a hazard ratio 2.74 [95% confidence interval (95% CI)1.29–5.7] ($P = .008$) compared with the prerenal AKI.
CONCLUSION: AKI in hospitalized patients with COVID19 presented with two different clinical patterns. Prerenal AKI more frequently affected older, more comorbid patients, and with a mild COVID19 infection. The NTA AKI affected younger patients, with criteria of severity of infection and multiplying mortality almost three times. In analytical control 1-month post-hospitalization, most of the patients recovered their kidney function. Although the implications of AKI associated with COVID-19 in the development of chronic kidney disease are still unclear, our data suggest that most patients will recover kidney function in a medium term.

MO329 **ACUTE KIDNEY INJURY (AKI) IS ASSOCIATED WITH INCREASED IN-HOSPITAL MORTALITY AND WITH IMPAIRMENT OF RENAL, LUNG, MOTOR AND IMMUNE FUNCTION 1 YEAR AFTER DISCHARGE FOR COVID-19**

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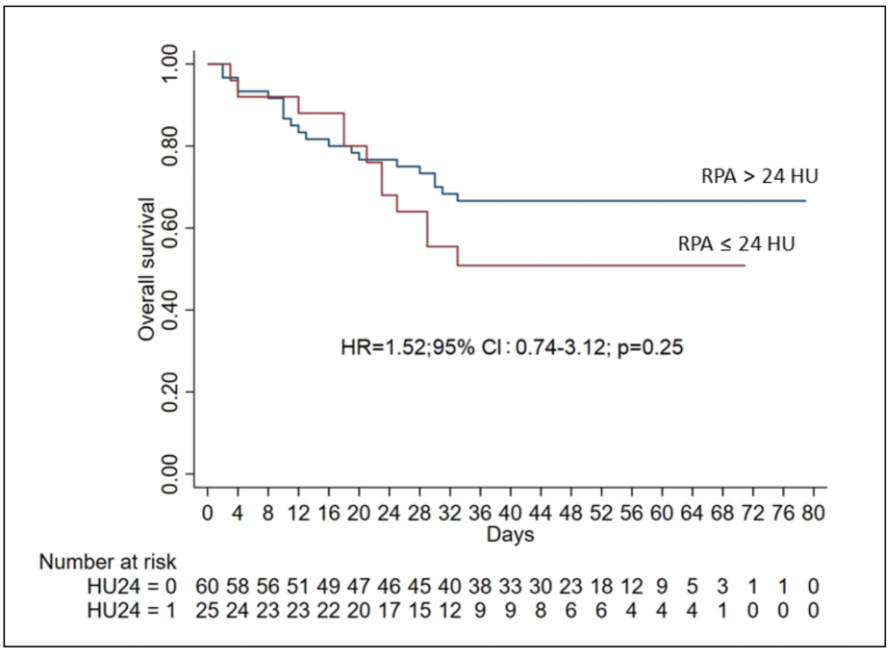
BACKGROUND AND AIMS: AKI is the most frequent complication after respiratory failure in COVID-19. AKI increases mortality risk, length of hospital stay and healthcare costs, with possible progression towards CKD. Study aims: (1) evaluation of AKI incidence in 1020 COVID-19 hospitalized patients; (2) comparison of AKI incidence in COVID-19 versus pre-pandemic period; (3) establishment of out-patient follow-up for monitoring kidney, lung, motor and immune function; (4) creation of a biobank for biomarker discovery studies.
METHOD: AKI incidence was calculated matching laboratory and administrative data of 26 214 hospitalized patients in 2018–2019 and in 1020 COVID-19 patients in 2020–2021: KDIGO algorithms were applied for AKI grading. After 12 months from discharge, 232 COVID AKI patients and relative controls matched for age and gender were evaluated for kidney (eGFR, biomarkers of tubular damage NGAL, CCl-14, DKK-3), lung (DLCO, CT scan) and neuro-motor (SPPB, 2-min walking test, post-traumatic stress test-IES) function.
RESULTS: Before the pandemic, in-hospital AKI incidence was 18% (10% KDIGO 1, 5% KDIGO 2, 3% KDIGO 3): median age of AKI patients was 69. In-hospital mortality was 3.5% in non-AKI group versus 15% in AKI group in accordance with KDIGO stages. In COVID patients, AKI incidence increased to 37% (20% KDIGO 1.11% KDIGO 2, 6% KDIGO 3): median age of patients was 54. In-hospital mortality was 31% in the AKI group; AKI is an independent risk factor for death. After 12 months from hospital discharge, COVID AKI patients showed a persistent reduction of respiratory function (severe DLCO impairment < 60%) related to the extent of CT scan abnormalities. AKI patients also presented the motor function impairment and a worse post-traumatic stress response. GFR reduction was 1.8 mL/min in non-AKI patients versus 9.7 mL/min in AKI COVID patients not related to age. Urinary DKK-3

Table 1. Univariate and multivariate logistic regression analyses for AKI in hospitalized patients with COVID-19

	UNIVARIATE MODEL			MULTIVARIATE MODEL		
	Odds ratio	95% CI	*P*	Odds ratio	95% CI	*P*
Age, years	1.00	0.97–1.03	.960	0.96	0.91–1.01	.146
Male gender	0.87	0.32–2.33	.777	0.26	0.05–1.23	.090
Charlson index	1.11	0.90–1.38	.325	1.16	0.89–1.53	.266
Hypertension	2.79	1.13–6.88	**.025**	1.25	0.30–5.30	.759
Diabetes	0.56	0.14–2.29	.422			
Heart failure	1.19	0.25–5.70	.828			
Creatinine, mg/dL	4.84	1.23–29.13	**.026**	50.7	4.62–556.01	**.001**
RPA < 24 HU	2.22	0.86–5.74	.100	4.56	1.27–16.44	**.020**



Abbreviations: HR, hazard ratio; CI, confidence intervals; RPA, renal parenchyma attenuation; HU, Hounsfield units.

FIGURE 1: Kaplan–Maier curves of survival for hospitalized COVID-19 patients on the basis of AKI-specific cut off of RPA.

and CCL-14 were also higher in the AKI group. Last, IgG response after SARS-CoV-2 vaccination was significantly lower in the AKI group.
CONCLUSION: AKI incidence was significantly increased during COVID-19 in respect to the pre-pandemic period, with an association with higher mortality in class 2–3 KDIGO. In the post-COVID follow-up, AKI was associated with lung and neuromotor function impairment, a defective antibody response and a sudden GFR decline concomitant to the persistence of tubular injury biomarkers. These results suggest the importance of nephrological and multidisciplinary follow-up of frail patients who developed AKI during hospitalization for COVID-19.

MO330 **AKI AND RENAL PARENCHYMA ATTENUATION IN HOSPITALIZED PATIENTS WITH COVID-19**

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BACKGROUND AND AIMS: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) had a worldwide spread since early 2020 and a lot of studies concerning the diagnostic and prognostic role of chest computed tomography (CT) on coronavirus disease (COVID-19) has been published. Renal involvement might be present in up to 75% of cases, significantly impacting on the prognosis.
The aim of this study is to clarify the role of opportunistic kidney assessment on non-enhanced chest CT and to evaluate if radiological findings could be associated with relevant clinical information regarding kidney function and patient's prognosis in hospitalized patients with COVID-19.

METHOD: We collected data on patient demographics, comorbidities, chronic medications, vital signs, baseline laboratory test results and in-hospital treatment in patients with COVID-19 consecutively admitted to our Institution who underwent chest CT. The standard chest CT-scan acquired in full inspiration include large part of both kidneys as per protocol. Three regions of interest (ROI) of 0.5–0.7 cm^2 were positioned in every kidney, right and left to include both the cortex and the medulla. The mean values of attenuation of kidney regions were analysed. The primary and secondary outcomes were the occurrence of acute kidney injury (AKI), in-hospital and 9 months of death for all causes.
RESULTS: A total of 86 patients with COVID-19 and unenhanced chest CT were analyzed splitting the cohort into CT renal parenchyma attenuation (RPA) quartiles. Patients with a CT RPA below 24 Hounsfield unit (HU) were more likely to develop AKI when compared with other patients ($\times 2 = 2.77$, $P = .014$). An AKI-specific cut-off point of RPA was identified by performing a survival receiver operating characteristic (ROC) curve. At multivariate logistic regression analysis, being in the first quartile of CT RPA was associated with a four-times higher risk of AKI (Table 1) after adjustment for age, gender, hypertension, kidney function at admission and other comorbidities. During a mean 22 ± 15 days of admission, 32 patients died (37.2%). Patients with lower values of RPA at CT (first quartile, <24 HU) were not at a higher risk of death compared with patients with RPA ≥ 24 HU, as shown by Kaplan Maier curve (Fig. 1) and by multivariate Cox regression analysis [HR 1.84 (95% CI 0.82–4.13); $P = .14$].
CONCLUSION: The association between AKI and RPA < 24 HU was independent of age, gender, creatinine and comorbidities. RPA values seemed to be predictive of AKI development in COVID-19 patients who underwent chest CT, suggesting RPA values could significantly improve patients' care. The opportunistic measure of RPA could help physicians identifying patients with a higher risk of AKI, and this increased awareness could guide choices for diagnostic and therapeutic procedures.