The first cases of the disease were identified in Wuhan, China, which occurred in early December 2019. Initially, it was called pneumonia of unknown origin, severe acute (SARS-CoV-2) [2]. In addition to being a target of the virus, the kidney also seems to have a substantial influence on the outcomes of the disease [3]. Underlying kidney disease is an emerging risk factor for more severe coronavirus disease 2019 (COVID-19) illness [4]. Identifying patients who require a palliative care approach is challenging for family physicians, even though several identification tools have been developed for this purpose. The Supportive and Palliative Care Indicators Tool (SPICT^{TM}) aims at facilitating this identification [5]. The aim of this study is to compare the outcomes presented between patients with nephropathy and lung disease in the first wave of COVID-19 assisted by a reference center in Brazil.

METHOD: This study is a data analysis from patients assisted by a reference center in São Paulo, Brazil, which includes 2013 patients from March to July of 2020. This period consists on the first wave of COVID-19 infection in this country. In addition, a literature review was conducted, papers were selected searching PubMed/Medline, SciELO and LILACS databases using the terms (COVID-19) AND (nephropathy) AND (pneumopathy) AND (outcomes) in January 2022. There were no language or publication date restrictions. Also, we used the (SPICT^{TM} to classify the patients for treatment with palliative care.

RESULTS: Among 2013 patients included in our study, 223 had nephropathy, 127 males and 96 females. As for pulmonary disease, there were 155 patients, 93 males and 62 females. Furthermore, among pneumopathy patients, 65% were >60 years old, and, among renal patients, 47% approximately were >60 years old. In addition, the mean age of the renal patients' group was lower than the group of patients with lung disease. The outcomes in the nephropathy group consisted in 109 discharges and 114 deaths. In the group of pulmonary patients, 90 were discharged and 59 died; the other patients were transferred to specialized services. Renal patients presented worse outcomes than pulmonary patients, even though COVID-19 mainly affects the lung, our patients had substantial impact of its infection. Despite the fact that SPICT^{TM} identified 41 renal patients eligible for palliative care, only 31 were referred to this service at the hospital where the study was conducted. When compared with the total population of this study, the renal patient's group was the third with the highest number of hospitalizations due to COVID-19.

CONCLUSION: Renal patients had a worse prognosis when compared with patients with lung disease and the total population. The outcomes could have been different if all renal patients had been referred and treated with palliative care.

MO305 COVID-19 IN ELDERLY PATIENTS WITH ACUTE KIDNEY INJURY

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BACKGROUND AND AIMS: Coronavirus disease 2019 (COVID-19), which started in China in December 2019 and spread all over the world, is more progressive in patients who are elderly and with chronic diseases. Especially, kidney involvement affects the survival of patients. In this study, we analysed COVID-19 patients who developed acute kidney injury treated in our unit, retrospectively.

METHOD: The clinical and laboratory data of 610 patients who were hospitalized due to COVID-19 pandemic between 1 June 2020 and 30 June 2021 in the intensive care and other clinics of our hospital were evaluated from the records, retrospectively. A total of 140 patients were diagnosed with AKI according to the criteria of Kidney Disease Global Outcomes (KDIGO). The patients were divided into two groups as KDIGO stages 1 and 2 and 3.

RESULTS: The median age in both groups was 70 (35–92) and 73 (35–90) years. Approximately 70% of them were >65 years old. Almost all of the patients had hypertension. Most of the patients were using angiotensin converting enzyme inhibitors (ACE inh) or angiotensin receptor blockers (ARB) (84%). AKI was present at the time of admission (61.9%) in the KDIGO 1 group and at the time of hospitalization (64.3%) in the KDIGO 2, 3 groups. The mortality rate was higher in stage 2–3 AKI patients (35.7%). Ferritin and fibrinogen levels were high in the KDIGO 2, 3 group, while lymphocyte levels were low.

CONCLUSION: AKI can be seen at the time of admission and during treatment in patients who are hospitalized and treated due to COVID-19. COVID-19 is more mortal in patients with advanced AKI.

Table 1. Characteristics and laboratuary findings in both groups

	KDIGO stage 1 ($n = 112$)	KDIGO stage 2 and 3 ($n = 28$)	P
Age, median (min–max)	70 (35–92)	73 (35–90)	.630
Age ≥ 65, *n* (%)	76 (67.9)	76 (67.9)	1
Diabetes mellitus, *n* (%)	44 (39.3)	8 (28.6)	.294
Hypertension, *n* (%)	109 (97.3)	27 (96.4)	.800
Chronic kidney disease, *n* (%)	26 (23.2)	6 (21.4)	.840
Obesity, *n* (%)	2 (1.8)	1 (3.6)	.491
Chronic obstructive pulmonary disease, *n* (%)	16 (14.3)	3 (10.7)	.765
Coronary artery disease, *n* (%)	46 (41.1)	11 (39.3)	.863
Heart failure, *n* (%)	33 (29.5)	4 (14.3)	.103
Cerebrovascular disease, *n* (%)	6 (5.4)	3 (10.7)	.383
Malignity, *n* (%)	13 (11.6)	7 (25)	.126
Chronic liver disease, *n* (%)	2 (1.8)	0 (0)	.476
Medications			
-ACE inh, *n* (%)	54 (48.2)	13 (46.4)	.866
-ARB, *n* (%)	41 (36.6)	10 (35.7)	.930
-CCB, *n* (%)	42 (37.5)	10 (35.7)	.861
-BB, *n* (%)	72 (64.3)	13 (46.4)	.084
-Insulin, *n* (%)	32 (28.6)	3 (10.7)	.051
-OAD, *n* (%)	13 (11.6)	5 (17.9)	.359
-Antiagregan, *n* (%)	78 (69.6)	16 (57.1)	.208
-Anticoagulan, *n* (%)	9 (8)	2 (7.1)	.875
Duration of acute kidney injury, *n* (%)	5 (2–25) ($n = 39$)	7 (2–25) ($n = 16$)	.386
Acute renal failure			
-during hospitalization, *n* (%)	38 (33.9)	18 (64.3)	**.003**
-at admission, *n* (%)	69 (61.9)	10 (35.7)	**.013**
AKI on CKD	26 (23.2)	6 (21.4)	.626
AKI progression, *n* (%)	10 (8.9)	11 (39.3)	**<.001**
Mortality, *n* (%)	9 (8)	10 (35.7)	**.001**
Duration of intensive care unit, median (min–max)	8 (2–45)	6 (1–21)	.546
Ferritin (μg/L)	304.10 (23.40–2000)	517.50 (74.10–2000)	**.042**
Lenfosit (10^3/μL)	1.32 (0.24–4.92)	0.89 (0.06–2.13)	**<.001**
D dimer (μg/mL)	9.50 (1.5–92.2)	10.2 (2–91.6)	.306
Fibrinogen (mg/dL)	424 (148–900)	511.50 (189–900)	**.017**
Serum creatinine (mg/dL)	1.47 (0.56–5.35)	1.43 (0.56–8.38)	.942
Serum Na (mmol/L)	137 ± 4.9	136 ± 4.9	.206
Serum albumin (g/dL)	3.45 ± 0.47	3.32 ± 0.44	.166
CRP level, ≥5, *n* (%)	98 (87.5)	27 (96.4)	.304
Oxygen saturation > 90, *n* (%)	97 (86.6)	17 (60.7)	**.002**

MO306 **PHARMACOLOGIC VERSUS IMMUNOLOGIC ACUTE INTERSTITIAL NEPHRITIS LONG TERM OUTCOME. ARE THERE ANY DIFFERENCES**

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BACKGROUND AND AIMS: Acute interstitial nephritis (AIN) is a common cause of acute renal failure affecting between 5% and 27%. It is characterized by the presence of interstitial inflammatory infiltrate, tubulitis and edema. A considerable number of cases develop chronic kidney disease (CKD), with a linear relationship observed with the diagnosis delay. There are different etiologies, most importantly those secondary to drug exposure and those related to immunological diseases. To the date, there are few studies comparing both etiologies. In this regard, the aim of this study is to determine difference between both etiologies by comparing clinical, histological and renal prognosis.

METHOD: A retrospective, observational, single-center study was performed. Clinical and histological characteristics of patients with a first episode of pharmacological or immunological AIN, confirmed by renal biopsy between 1998 and 2020, were compared. Histological analysis was measured by semi-quantitative methods.

RESULTS: A total of 67 patients have been described, with a mean age of 68 ± 13 years, 56% were women. The baseline creatinine was 93.8 umol/L with an eGFR (CKD-EPI) of 67 mL/min/1.73 m^2; mean follow-up time was 63 ± 55 months. A total of 46 patients presented pharmacological AIN, being NSAIDs and antibiotics the most frequent agents, 21 patients with AIN associated to immunological diseases, being Sjögren's syndrome the most frequent. Previous CKD was present in both groups, with 42% in immunological AIN versus 32% in pharmacological AIN, of which stage 3a CKD was more frequent in drug-mediated AIN (69% versus 33%) as opposed to stage 3b CKD was more frequent in immunological AIN (67% versus 19%). However, no significant differences were observed in the estimated glomerular filtration rate when comparing both groups. A longer diagnostic delay was observed in patients with immunological, defined as the elapsed time from renal function deterioration to the performance of renal biopsy, 93 ± 210 versus 13 ± 15 days ($P = .021$). However, creatinine at the time of diagnosis was higher in patients with pharmacological cause (380 ± 199 versus 252 ± 246 umol/L; $P = .0002$), leukocyturia was more frequent in the pharmacological AIN group without differences in the percentage of hematuria or proteinuria. In histological analysis, there were no significant differences in interstitial infiltrate, renal fibrosis or tubular atrophy.

Once the biopsy was performed, treatment was started in all patients before the 5th day. In pharmacological AIN, the causative drug was withdrawn and corticosteroids were administered to the majority of patients. Out of 46 patients, 7 (15%) patients of the pharmacological AIN group, the drug was withdrawn with no additional treatment, 4 of them progressed to end-stage kidney disease (ESKD). In the immunological AIN group, most of patients were treated with corticosteroids or immunosuppressants. Out of 21 patients, 3 patients (14%) were not treated