

# Focus on renal blood flow in *mechanically ventilated* patients with SARS-CoV-2

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## Research Article

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# Abstract

**Background:** Patients with ARDS due to the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) seem particularly susceptible to AKI. Our hypothesis was that the renal blood flow could be more compromised in SARS-CoV-2 patients than in patients with “traditional” ARDS. We compared the renal resistivity index (RRI) and the renal venous flow (RVF) in ARDS patients with SARS-CoV-2 and in ARDS patients due to other etiologies.

**Materials and Methods:** Prospective, observational study performed on 30 mechanically ventilated patients (15 with SARS-CoV-2 ARDS and 15 with ARDS). Ultrasound Doppler measurements of RRI and RVF pattern were performed in each patient.

**Results:** Patients with SARS-CoV-2 ARDS had higher RRI than patients with ARDS (0.71[0.67–0.78] vs 0.64[0.60–0.74],  $p=0.04$ ). RVF was not-continuous in 9/15 patients (71%) in the SARS-CoV-2 ARDS group and in 5/15 (33%) in the ARDS group ( $p=0.27$ ). A linear correlation was found between PEEP and RRI in patients with SARS-CoV-2 ARDS ( $r^2=0.31$ ;  $p=0.03$ ) but not in patients with ARDS. Occurrence of AKI was 53% in patients with SARS-CoV-2 ARDS and 33% in patients with ARDS ( $p=0.46$ ).

**Conclusions:** We found a more pronounced impairment in renal blood flow in mechanically ventilated patients with SARS-CoV-2 ARDS, compared with patients with “traditional” ARDS.

## Introduction

The occurrence of acute kidney injury (AKI) in mechanically ventilated patients with acute respiratory distress syndrome (ARDS) due to the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) ranges between 15 and 23% being as high as 50% in non-survivors. [1,2] Besides COVID-19 may *per se* induce a kidney disease, [3] mechanical ventilation could play a role. Recent guidelines recommend the use of lung-protective mechanical ventilation in ARDS, consisting in low tidal volumes and relatively high levels of positive end-expiratory pressure (PEEP). [4] although this strategy could have a relevant hemodynamic impact. [5,6] In particular, positive pressure ventilation associated with relatively high PEEP levels has been shown to increase arterial resistances and decrease venous return, leading to glomerular congestion and increase in kidney interstitial hydrostatic pressures. [5-8] Furthermore, it seems that in SARS-CoV-2 ARDS respiratory system compliance ( $C_{RS}$ ) is atypically high compared to the degree of arterial hypoxemia, [9-10] and this could lead to even more pronounced impact of PEEP on systemic and renal perfusion. [9-10] This mechanism of impairing renal function should be considered as additional to the kidney damage due to COVID-19 infection by itself. [3] It might be then hypothesized that the levels of PEEP needed in patients with COVID-19 infection could result in greater extent of kidney damage compared to those patients with ARDS due to different etiologies. Hence the aim of our study was to document a potential different involvement of the kidney function by evaluating the degree of renal blood flow impairment in SARS-CoV-2 ARDS compared to those with the “classic” ARDS. Accordingly, we performed an ultrasound evaluation of the renal resistivity index (RRI) and renal venous flow pattern

(RVF) [11-13] in two groups of patients, i.e. with SARS-COV-2 ARDS and with ARDS, ventilated with the same lung-protective protocol.

## Materials And Methods

### *Population*

Prospective, observational study performed in a COVID-19 Intensive Care Unit (ICU) and a mixed medical-surgical ICU at University Hospital of Ferrara, Italy. The study was approved by the ethic committee (approval number 339/2020). We included consecutive patients with SARS-COV-2 ARDS or ARDS undergoing mechanical ventilation. Exclusion criteria were: diagnosis of AKI before ICU admission, unsatisfactory ultrasound visualization, arrhythmia, renal replacement therapy and denied consent. All patients included in the SARS-COV-2 ARDS group had laboratory-confirmed COVID-19.

### *Mechanical ventilation settings*

At the time of renal blood flow assessment, all patients were deeply sedated and paralyzed with continuous infusion of cis-atracurium 1–3 mcg/kg/min for clinical reasons. Mechanical ventilation settings included constant-flow controlled ventilation, a tidal volume of 6 ml/kg of ideal body weight and the PEEP level titrated to the lowest driving pressure. [14] Briefly, PEEP was increased by 2 cmH<sub>2</sub>O step starting from 6 cmH<sub>2</sub>O, up to the PEEP level leading to a static end-inspiratory plateau pressure ( $P_{PLAT}$ ) of 28-30 cmH<sub>2</sub>O; then, the PEEP level corresponding to the lowest driving pressure was chosen.

The driving pressure was measured as:

$$P_{PLAT} - PEEP_{TOT}$$

Where  $PEEP_{TOT}$  is the total static positive end-expiratory pressure.  $P_{PLAT}$  and  $PEEP_{TOT}$  were measured through the occlusion technique (i.e. a 4 sec airway opening occlusion maneuver at end-inspiration and end-expiration, respectively)

### *Renal blood flow assessment*

Ultrasound evaluation of renal blood flow was performed within 24 hours from starting of MV. All the measurements were taken on the right kidney with the patient in semi-recumbent position (30 degrees), through a posterolateral approach. Color Doppler images were used to identify the interlobar vessels. An interlobar or arcuate artery was selected for pulse wave Doppler measurements and RRI was calculated as:

peak systolic velocity-end diastolic velocity/peak systolic velocity.

An RRI >0.7 was deemed pathological. [11-13]

The RVF pattern was classified as continuous, biphasic or monophasic. [11] Under physiological conditions, the RVF pattern is continuous, whereas during renal congestion it becomes biphasic or even monophasic. [11]

For both RRI and RVF three measurements were obtained over 3 cardiac cycles and the mean value was recorded for analysis.

### *Secondary outcomes*

The occurrence of AKI, as well as need for renal replacement therapy (RRT), was recorded. Further, we investigated the ability of RRI to predict the occurrence of AKI and the need of RRT. AKI was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines as an increase in serum creatinine of 0.3 mg/dL within 48 h, an increase in serum creatinine to 1.5 times the baseline value present within the previous 7 days, or a urinary output < 0.5 mL/kg/h for 6 hours. [15] Indications for RRT were serum urea >150 mg/dL, severe hyperkalemia (over 6 mmol/L or with sign of electrocardiogram abnormalities), urine output <200 mL/die, fluid overload despite diuretic treatment. [16]

### *Statistical analysis*

Categorical data are presented as frequencies and percentages, while continuous variables as mean  $\pm$  standard deviation or medians with interquartile range, as appropriate. The Shapiro-Wilk test was used to assess the assumption of normality. Categorical data were compared using the  $\chi^2$  test or Fisher exact test as appropriate. Mann-Whitney U tests was used to compare continuous variables. Pearson correlation with R square was used to analyze the correlation. Receiver operator characteristic (ROC) curves were used to analyze the ability of RRI to predict AKI. ROC curve analyses are reported as AUROC with 95% confidence interval (95% CI). Optimal cut-off scores were determined using Youden's Index. Differences in repeated measurements in the two groups were analyzed using Friedman's rank analysis. Statistical analyses were performed using SPSS 25.0 statistical software (SPSS Inc., Chicago, IL). For each statistical test a 2-tailed test was performed and a p value of 0.05 was considered statistically significant.

### *Sample size*

Due to the lack of clinical study in this field, we were unable to perform an accurate power analysis. Nonetheless, using the upper confidence interval for the population variance approach to the sample size calculation a pilot sample size between 20 and 40 correspond to standardized effect sizes of 0.4 and 0.7 (for 90% power based on a standard sample size calculation). According to this issue, 30 patients (15 for each group) were included in the study.

## **Results**

### *Population and renal blood flow evaluation*

The renal blood flow evaluation was performed after 1 [0–1] days of MV. As shown in Table 1, compared with patients with ARDS, patients with SARS-CoV-2 ARDS had higher RRI (0.71 [0.67–0.78] vs 0.64 [0.60 – 0.74],  $p=0.04$ ). (Figure 1) In patients with SARS-CoV-2 ARDS, but not in patients with ARDS, there was a linear correlation between PEEP and RRI ( $r^2=0.31$ ;  $p=0.03$ ) (Figure 2). The RVF was not-continuous in 9/15 patients (71%) in SARS-CoV-2 ARDS group and 5/15 (33%) in not-CoV-2 ( $p=0.27$ ).

### *Secondary outcomes*

AKI occurred in 8/15 of patients (53%) in SARS-CoV-2 ARDS and in 5/15 (33%) in ARDS ( $p=0.46$ ). AKI was detected after 2 [1-2] days (range 1-4) from renal blood flow evaluation. Four patients (4/15, 27%) in SARS-CoV-2 ARDS group needed RRT versus one in the ARDS group (1/15, 6%;  $p=0.33$ ). Additional data regarding SARS-CoV-2 ARDS patients are given in Table S1 and Figure S1. In particular, SARS-CoV-2 ARDS patients with AKI were more likely to experience a subsequent impairment in respiratory system compliance (Figure S1). Taking into account the whole population, patients who developed AKI had higher RRI (0.77 [0.73–0.80] vs 0.66 [0.60–0.69];  $p=0.001$ ) as well as higher percentage of impaired RVF (13/13 vs 1/17;  $p<0.001$ ). The RRI was associated with the subsequent occurrence of AKI (AUROC 0.938 [0.93–0.99];  $p<0.001$ ). The Youden index analysis showed that  $RRI>0.71$  was the best cut-off value to predict AKI, with 90% sensitivity and 100% specificity. The RRI was also a predictor for RRT (AUROC=0.896 [0.73 – 0.98]; best cut-off 0.77; sensitivity 80% specificity 92%).

## **Discussion**

The main finding of our study is that the renal blood flow is more compromised in mechanically ventilated patients with SARS-CoV-2 ARDS than in patients with ARDS due to other etiologies.

The pathogenesis of renal dysfunction in patients with COVID 19 is multifactorial. [2,3,17] A recent observational study including 116 hospitalized patients shows that non ventilated patients with SARS-CoV-2 pneumonia seldom develop AKI [18]; conversely, in studies including patients requiring respiratory support, incidence of AKI was up to 23%. [1] Despite the increasing severity of the disease could explain these findings, positive pressure ventilation can also play an important role. [5-8]. The underlying mechanism could be the well-known impact of positive pressure on the renal blood flow. Mechanical ventilation could induce glomerular congestion, by increasing the pericardial pressure, leading to a critical increase in interstitial hydrostatic pressure [6-7], which increases arterial resistances [6-7]. Interestingly, Gattinoni and Marini suggested that the SARS-CoV-2 induced ARDS is as a peculiar ARDS form characterized by an atypically high respiratory system compliance ( $C_{RS}$ ), compared to the degree of arterial hypoxemia. [9-10,19-20] Since the hemodynamic effects of PEEP are enhanced at relatively higher  $C_{RS}$ , [20-21] the hemodynamic impact of mechanical ventilation on kidney perfusion could be more pronounced in patients with SARS-CoV-2 ARDS than in patients with ARDS due to other etiologies. Our finding of a linear relationship between RRI and PEEP only in patients with SARS-CoV-2 ARDS (figure 2) seems to support this hypothesis, although we should point out that our study was not specifically designed to document the impact of PEEP on renal blood flow.

Our results confirm the role of RRI and RVF in predicting occurrence of AKI. The RRI evaluation has gained importance in last years to monitor renal perfusion and to predict the occurrence of AKI, [11-13,22] as well as its severity. [23] Indeed, the genesis of kidney injury includes intrarenal vasoconstriction and endothelial damage of the micro-vessels which lead to impaired macro- and microvascular flow, both causes of increased RRI. [22,23] On the other hand, the RVF pattern is able to identify the occurrence of glomerular congestion, which is an additional cause of renal blood flow impairment [11]. High RRI values have been associated with the need for vasopressor support, hypotension and lactate levels [12], whereas non-continuous RVF have been described in the context of increased pulmonary wedge pressure [11]. We have also showed that high RRI values are common in patients undergoing lung-protective mechanical ventilation, regardless of the need of vasopressor or the presence of hyperlactacidemia.

Our study has some limitations. First, the small sample size could limit the interpretation of our results and does not account for covariate adjustment. Second, our study was not designed to separate between the different mechanism that could explain the impairment of renal blood flow. Nonetheless, the correlation between PEEP levels and RRI seems to suggest a possible role of mechanical ventilation in the RRI impairment.

## Conclusions

In conclusion, we showed that during lung-protective mechanical ventilation, the renal blood flow is more impaired in patients with SARS-CoV-2 ARDS than in patients with ARDS due to other etiologies. Our findings suggest to carefully assess renal blood flow and even subtle signs of AKI in patients with SARS-CoV-2 ARDS requiring mechanical ventilation. Further studies are needed to assess the specific role of mechanical ventilation in the pathogenesis of the SARS-CoV-2 ARDS kidney perfusion impairment and, eventually, if different approaches to PEEP setting could better preserve renal perfusion.

### What is known:

- AKI is common in patients with SARS-CoV-2 ARDS
- Lung protective ventilation may impair renal blood flow
- Kidney doppler ultrasound allows the evaluation of renal resistivity index

### What is new:

- Renal blood flow is more compromised in SARS-CoV-2 ARDS than in “traditional” ARDS.
- PEEP-induced impairment in renal blood flow might be higher in SARS-CoV-2 ARDS
- Ultrasound evaluation of renal blood flow is able to predict AKI in mechanically ventilated patients

## Declarations

### Competing Interests

The authors have no competing interests.

## Ethics Approval

The study was approved by our ethic committee (approval number 339/2020).

All patients gave written consent to the study; for patients unable to give, the consent was taken by next of kin and delayed consent was given by patient if he became able to give.

## Funding

This study was financially supported by the University of Ferrara, Ferrara, Italy

## Author contributions

FA, VCA and SS was involved in the conception and the design of the study, analyzed the data and wrote the paper. FA and ME collected the data. FA performed the statistical work. VCA and SS were involved in the conception and the design of the study. MD and GS and LG contributed to analysis of the data and critical revision of the manuscript for important intellectual content. All authors reviewed the manuscript and agreed with the final version.

## Abbreviations

ARDS: acute respiratory distress syndrome; SARS-CoV-2: acute respiratory syndrome coronavirus 2; MV: mechanical ventilation; RRI: renal resistivity index; RVF: renal venous flow pattern; KDIGO: Kidney Disease: Improving Global Outcomes; RRT: renal replacement therapy; ROC: Receiver operator characteristic;

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## Tables

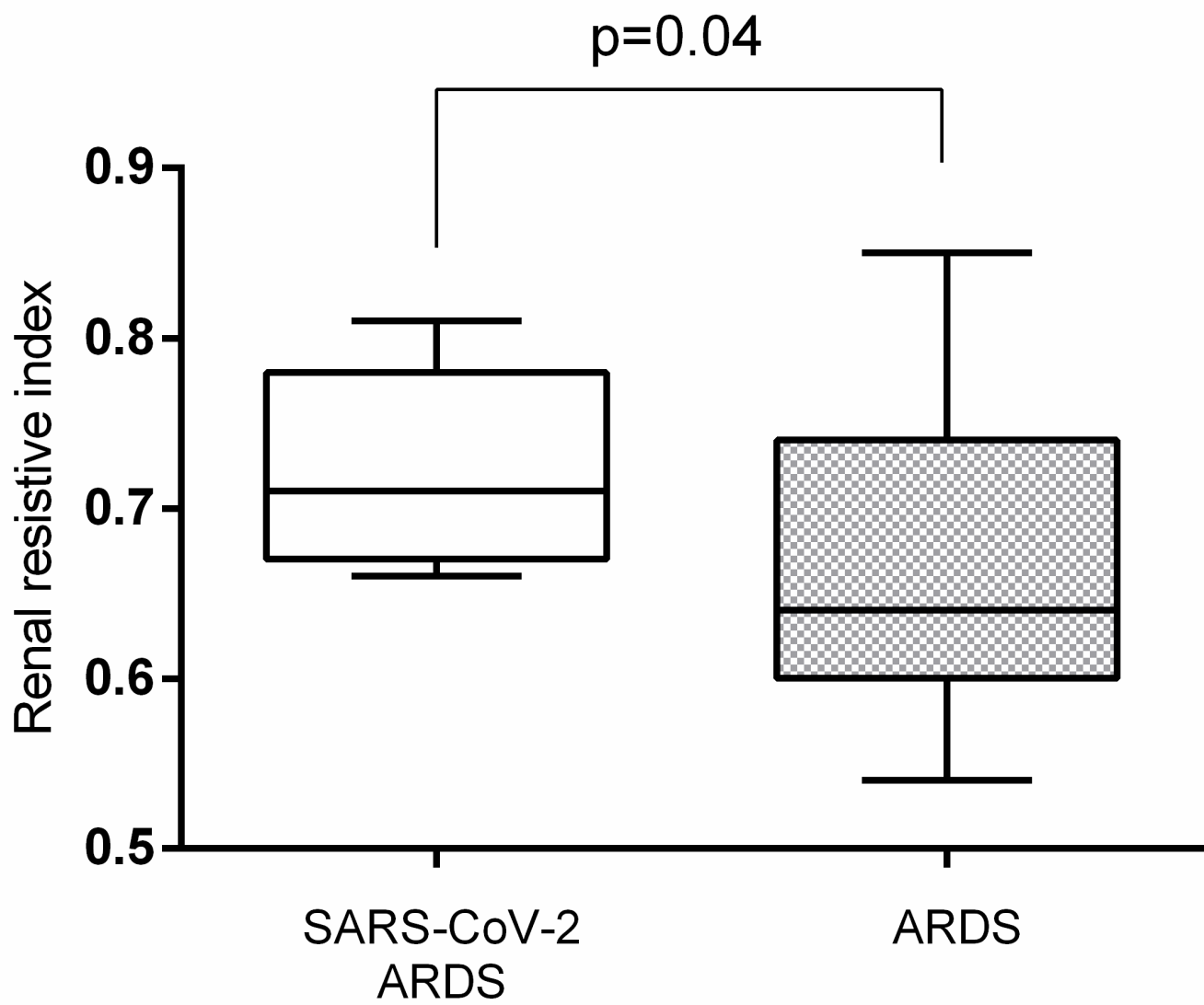
Variables	All patients n=30	SARS-CoV-2 ARDS n=15	ARDS n=15	P value
Age	64 [60 – 72]	62 [55 -69]	67 [62 – 74]	0.89
Sex (F/M)	5/25	2/13	3/12	0.99
SOFA score at ICU admission	8 [5 -10]	6 [4 – 10]	9 [8 – 10]	0.05
Need for vasopressor, n (%)	12 (40)	5 (33)	7 (46)	0.71
<i>Comorbidities</i>				
Hypertension, n (%)	17 (57)	8 (53)	9 (60)	0.71
CKD, n (%)	7 (23)	3 (20)	4 (27)	0.99
BMI>35 (kg/m <sup>2</sup> ), n (%)	3 (10)	1 (7)	2 (13)	0.99
Diabetes, n (%)	6 (20)	3 (20)	3 (20)	0.99
<i>Reasons for ICU admission</i>				
Septic shock	4	4 (27)	/	0.09
ARDS	26	11 (73)	15	0.09
<i>Laboratory data at ICU admission</i>				
Hemoglobin (g/dL)	11.0 [9.8 – 13.6]	10.9 [9.6 – 12.4]	12.8 [9.9 – 13.6]	0.49
Platelets (10 <sup>9</sup> /L)	219 [179 – 302]	263 [204 – 305]	208 [143 – 301]	0.19
Serum creatinine (mg/L)	1.0 [0.7 – 2.5]	0.8 [0.7 – 1.2]	2.5 [0.7 – 3.6]	0.11

**Table 1.** Clinical and demographical characteristics of patients at ICU admission. CKD: Chronic kidney disease; BMI: Body Mass Index; PEEP: Positive end-expiratory pressure; PaCO<sub>2</sub>: arterial carbon dioxide tension

Variables	SARS-CoV-2 ARDS n=15	ARDS n=15	P value
<b><i>Renal blood flow evaluation</i></b>			
Renal resistivity index	0.71 [0.67 – 0.78]	0.64 [0.60 – 0.74]	0.04
Renal venous flow pattern			
· Continuous	6 (40)	10 (66)	0.27
· Biphasic	4 (27)	3 (20)	0.99
· Monophasic	5 (33)	2 (14)	0.40
<b><i>Respiratory mechanics</i></b>			
Tidal volume (mL)	440 [400 – 480]	395 [350 – 480]	0.27
Respiratory rate	18 [18 – 22]	18 [16 – 20]	0.14
Minute ventilation (L/m)	7.9 [7.2 – 9.0]	7.4 [6.0 – 8.6]	0.10
Plateau pressure (cmH <sub>2</sub> O)	22 [22 – 24]	21 [18 – 24]	0.11
PEEP (cmH <sub>2</sub> O)	14 [12 – 14]	10 [10 – 12]	0.004
Driving pressure (cmH <sub>2</sub> O)	10 [8 – 10]	9 [8 – 12]	0.90
Static compliance (ml/cmH <sub>2</sub> O)	45 [41 – 52]	40 [37 – 47]	0.03
<b><i>Hemodynamic parameters</i></b>			
Mean arterial pressure (mmHg)	75 [70 – 80]	82 [67 – 91]	0.21
Heart rate	90 [86 – 104]	89 [79 – 95]	0.84
<b><i>Blood gas analysis</i></b>			
PaO <sub>2</sub> /FiO <sub>2</sub> (mmHg)	118 [94 – 151]	193 [148 – 209]	0.001
PaCO <sub>2</sub> (mmHg)	57 [48 – 64]	46 [36 – 52]	0.02
Lactate (mmol/L)	1.2 [1.2 – 1.6]	2.2 [1.1 – 3.2]	0.13

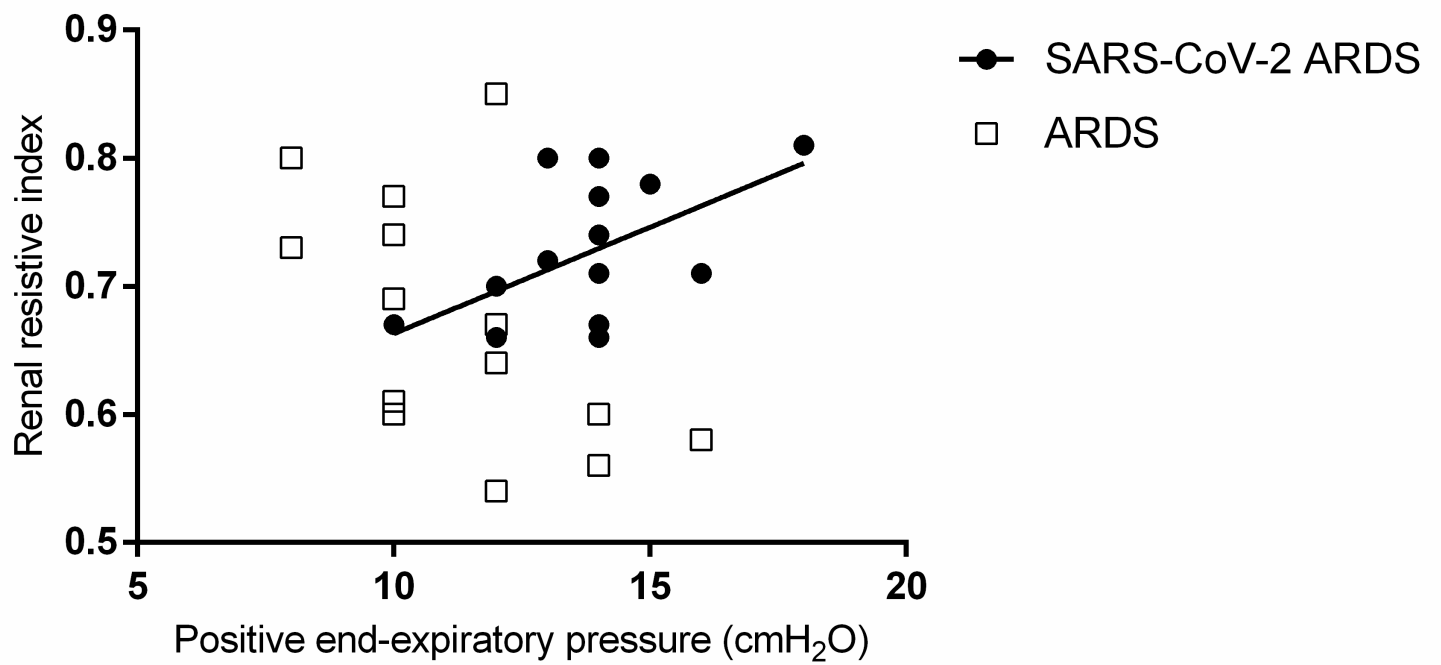
**Table 2.** Renal blood flow evaluation and respiratory mechanics in patients with C-ARDS and ARDS. SARS-CoV-2 ARDS: Coronavirus-induced ARDS; PEEP: Positive end-expiratory pressure; PaCO<sub>2</sub>: arterial carbon dioxide tension

## Figures



**Figure 1**

Comparison of renal resistivity index in patients with SARS-CoV-2 ARDS or ARDS.



**Figure 2**

Correlation between PEEP level and renal resistivity index in patients with SARS-CoV-2 ARDS or ARDS.

## Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

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