

# Lung Ultrasound in COVID-19: Clinical Correlates and Comparison With Chest Computed Tomography

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## Research Article

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# Abstract

**Background:** lung ultrasound (LUS) and chest computed tomography (chest-CT) are largely employed to evaluate coronavirus-disease-19 (COVID-19) pneumonia. We investigated semi-quantitative LUS and CT scoring in hospitalized COVID-19 patients.

**Methods:** LUS and chest-CT were performed within 24 hours upon admission. Both were analyzed according to semi-quantitative scoring systems. Subgroups were identified according to median LUS score.

**Results:** patients within higher LUS-score group were older (79 vs 60 years,  $p < 0.001$ ), had higher C-reactive protein (CRP) (7.2 vs 1.3 mg/dl,  $p < 0.001$ ) and chest-CT score (10 vs 4,  $p = 0.027$ ) as well as lower PaO<sub>2</sub>/FiO<sub>2</sub> (286 vs 356,  $p = 0.029$ ) as compared to patients within lower scores. We found a significant correlation between scores ( $r = 0.390$ ,  $p = 0.023$ ). Both LUS and CT scores correlated directly with patients age ( $r = 0.586$ ,  $p < 0.001$  and  $r = 0.399$ ,  $p = 0.021$  respectively) and CRP ( $r = 0.472$ ,  $p = 0.002$  and  $r = 0.518$ ,  $p = 0.002$  respectively), inversely with PaO<sub>2</sub>/FiO<sub>2</sub> ( $r = -0.485$ ,  $p = 0.003$  and  $r = -0.440$ ,  $p = 0.017$  respectively). LUS-score only showed significant correlation with hs-Troponin T, NT-pro-BNP and creatinine ( $r = 0.433$ ,  $p = 0.019$ ;  $r = 0.411$ ,  $p = 0.027$  and  $r = 0.497$ ,  $p = 0.001$  respectively).

**Conclusions:** semi-quantitative bedside LUS related to the severity of COVID-19 pneumonia similarly to chest-CT. Correlation of LUS-score with markers of cardiac and renal injury suggests that LUS might contribute to a more comprehensive evaluation of this heterogeneous population.

## Introduction

In late 2019 the outbreak in China of a novel type of betacoronavirus 2019-nCov, later renamed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), spread to generate a global pandemic of the so-called Coronavirus disease 2019 (COVID-19) [1]. This is a respiratory tract infection that may lead to severe systemic involvement with interstitial pneumonia and respiratory failure often associated with myocardial injury [2, 3], thrombosis [4], multiorgan failure and death [5]. Mainstay of COVID-19 diagnosis is nasopharyngeal swab and subsequent quantitative real-time reverse transcriptase polymerase chain reaction (qRT-PCR) [5], but a central role has been demonstrated for chest computed tomography (chest-CT) too. Indeed, this can provide differential diagnosis and assess complications [6], while the possibility of detecting COVID-19 pneumonia using a chest-CT-only approach has been explored [7]. Lung ultrasound (LUS) is another technique that can be used for lung evaluation in COVID-19 patients, with an expanding role favored by the possibility of performing it bed-side [8]. Aim of the present study was to investigate semi-quantitative assessment of LUS and CT findings in a cohort of patients hospitalized for COVID-19 pneumonia, providing comparison with clinical and laboratory data.

## Methods

# Patients enrollment

This is a partly-retrospective, observational, single center study on hospitalized patients with COVID-19 pneumonia. Diagnostic work-up and clinical management of COVID-19 patients at our institution have been previously described in detail [9]. For the purpose of the present study, we included in the analysis patients ( $n = 42$ ) who underwent timely LUS within 24 hours upon hospital admission. All patients included in the analysis had established diagnosis of SARS-CoV2 infection (qRT-PCR) with pulmonary involvement (chest CT) and were treated in dedicated “Covid units”. Past clinical history and symptoms were registered by the accepting physician. Within 24 hours upon admission, all patients underwent routine venous and arterial blood gas (ABG) examination as well as 12-lead ECG. On arterial blood gas test, arterial oxygen concentration ( $PaO_2$ ) was normalized to the fractional volume of the inspired oxygen to calculate the  $PaO_2/FiO_2$  ratio.

## *Chest computed tomography (CCT)*

Two multidetector CT scanners (Philips Brilliance 16 and Brilliance 64) were used for all examinations. Scanning parameters were the same as the manufacturer’s standard recommended pre-setting for a thorax routine. Images were acquired with a 1-mm slice thickness and a reconstruction increment of 0,5 mm in all cases using a soft tissue kernel of B20 and a lung kernel of B60. Coronal and sagittal multiplanar reconstructions (MPR) were also done in all cases.

Infection prevention and control measures were guaranteed in all suspected CT cases (sanitation of CT room and patient’s isolation). Suspicion of SARS-CoV2 pneumonia was established by the presence of three CT patterns: ground glass opacity (GGO), crazy paving and consolidation according to previous publications [10, 11]

The semi-quantitative severity score proposed by Pan was used per each of the 5 lobes considering the degree of anatomical involvement [12]. Specifically, we assigned points:

- 0, no involvement;
- 1, < 5% involvement;
- 2, 5–25% involvement;
- 3, 26–50% involvement;
- 4, 51–75% involvement;
- 5, > 75% involvement.

The resulting global CT score was the sum of each individual lobar score and (0 to 25). Collateral features such as fibrosis, subpleural lines, pleural and pericardial effusion, and lymphadenopathy were also depicted. Distribution of lung abnormalities was also classified as predominantly subpleural, centrilobular, random (without predilection for subpleural or central regions), or diffuse (continuous involvement without respect to lung segments). Blinded independent image analysis was performed by

two radiologists (M.D. and F.C., respectively with > 20 and > 10 years of experience in thoracic radiology) with use of the institutional digital database system (Impax Client, Agfa, version 6.6.0.145, Belgium). Any disagreement was resolved by consensus.

## **Lung ultrasound**

Portable ultrasound machines dedicated to exclusive use for patients with COVID-19, both equipped with a 3–6 MHz convex array transducer were used (GE Vividi and Siemens P500). All examinations were performed bedside within 24 hours upon hospital admission. Images were recorded, stored, and analyzed off-line by an experienced operator blinded to patients' clinical data and CT findings.

All subjects underwent bedside US scanning and were systematically studied as suggested by Soldati et al. in 14 areas: 3 posterior, 2 lateral, and 2 anterior on each chest side [13]. A score was assigned to each segment according to ultrasonographic appearances of chest US as follows: 1) presence of horizontal artifact, A-lines pattern, with continuous and regular pleural line (Score 0); 2) indented pleural with vertical artifacts appearing (Score 1); 3) small-to-large consolidated areas with associated white areas (white lung) (Score 2); 4) dense and largely extended white lung with or without larger consolidations (Score 3). Finally, a total score in each patient was calculated by summation of values recorded in all 14 segments. Groups with low- and high- LUS score respectively were identified according to median value and comparison provided.

## **Statistical analysis**

All analysis was performed using SPSS software 25 (SPSS Inc., Chicago, IL). Data are presented as mean  $\pm$  standard deviation, counts (percentages) or median (interquartile range, IQR), as appropriate. Comparisons between groups were performed using Chi-squared test, Fisher's exact test, Student t-test for independent samples or Mann–Whitney U test as appropriate. Analysis of relationships was performed using linear regression analysis and bivariate correlation with corresponding Pearson's or Spearman correlation coefficients as appropriate according to data distribution. Log-transformed values for serum biomarkers (hs-Troponin, NT-pro-BNP, D-dimer, CRP, creatinine) were used to yield approximate normality when performing correlation analysis. All tests were two-tailed, and  $p$  value of < 0.05 was considered statistically significant. All patients provided informed consent for the use of their record for research purposes, the study complied with the content of the Declaration of Helsinki.

## **Results**

### **Baseline characteristics**

Baseline clinical and demographic characteristics in our sample are summarized in Table 1. Mean age was 70 years and 48% were male. Comorbidities were largely prevalent within the study population, where more than half of the patients had known hypertension, approximately one third had pre-existing CVD and 57% were taking an ace-inhibitor or angiotensin II receptor blocker before hospital admission. Median CRP and D-dimer were increased (4.6 and 892 fibrinogen equivalent unit respectively). Blood gas analysis

showed mean PaO<sub>2</sub> of  $73 \pm 16$  mmHg and mean PaO<sub>2</sub>/FI<sub>O2</sub> of  $318 \pm 95$ . Median LUS score was 6 (3, 10), median CT score was 7 (3, 11).

Table 1

Baseline demographic and clinical characteristics of the study population overall and stratified by median LUS score value. LUS (lung ultrasound), CVD (cardiovascular disease) CRP (C-reactive protein), FEU (fibrinogen equivalent unit), CKD (chronic kidney disease), COPD (chronic obstructive pulmonary disease), WBC (white blood cells), NLR (neutrophil to lymphocyte ratio), CT (computed tomography).

Variable	Overall (n = 42)	Low LUS score (n = 21)	High LUS score (n = 21)	P
Age (years)	70 ± 18	60 ± 19	79 ± 11	≤ 0.001
Sex (male)	20 (48%)	11 (55%)	9 (45%)	0.537
Signs and symptoms at presentation				
Cough	20 (48%)	10 (48%)	10 (48%)	0.591
Dyspnea	15 (36%)	5 (25%)	10 (48%)	0.204
Fever	28 (66%)	13 (60%)	15 (71%)	> 0.99
Coexistent Conditions				
Hypertension	29 (%)	11 (55%)	18 (82%)	0.145
Dyslipidemia	8 (19%)	2 (10%)	6 (27%)	0.258
Diabetes	4 (9%)	1 (4%)	3 (15%)	0.613
Previous CVD	14 (33%)	5 (25%)	9 (41%)	0.275
- Atrial Fibrillation	8 (19%)	3 (15%)	5 (23%)	0.709
- Coronary Artery Disease	3 (7%)	3 (15%)	0 (0%)	0.083
- Heart failure	9 (22%)	3 (15%)	6 (28%)	0.476
- Stroke	3 (7%)	1 (4%)	2 (10%)	> 0.99
CKD	3 (7%)	0 (0%)	3 (15%)	0.238
COPD	11 (26%)	7 (35%)	4 (18%)	0.173
Cancer	4 (9%)	3 (15%)	1 (4%)	0.333
Ace-inhibitor therapy	24 (57%)	13 (60%)	11 (55%)	0.743
Laboratory Tests				
Hb (g/dl)	12.4 ± 2.5	12.9 ± 2.6	12 ± 2.4	0.289
WBC (per µL)	7.1 (5, 9.4)	6 (4.3, 9.3)	7.5 (5.3, 10.2)	0.411
Neutrophil (per µL)	4.7 (3.2, 7.2)	4.2 (3.1, 7.4)	5.9 (3.5, 7.3)	0.240
Lymphocyte (per µL)	1.1 (0.9, 1.6)	1.2 (0.99, 1.7)	1 (0.8, 1.5)	0.299

Variable	Overall (n = 42)	Low LUS score (n = 21)	High LUS score (n = 21)	P
NLR	4.2 (2.3, 7.1)	3.4 (2, 6)	5 (2.6, 8)	0.147
Creatinine (mg/dl)	0.8 (0.6, 1.14)	0.72 (0.56, 0.94)	1 (0.64, 1.6)	<b>0.014</b>
CRP (mg/dl)	4.6 (1, 7.7)	1.3 (0.3, 5)	7.2 (4.3, 13)	<b>&lt; 0.001</b>
D-dimer (FEU)	892 (548, 1376)	701 (276, 2168)	971 (745, 1216)	0.647
Hs-Troponin T (pg/ml)	17 (7, 43)	10 (6, 23)	24 (9, 62)	0.123
NT-pro-BNP (pg/ml)	436 (85, 4171)	187 (42, 610)	1068 (199, 1216)	0.057
Blood Gas Analysis				
pH	7.48 ± 0.08	7.5 ± 0.1	7.46 ± 0.05	0.170
pO2 (mmHg)	73 ± 16	79 ± 13	69 ± 16	<b>0.04</b>
pCO2 (mmHg)	35 ± 7	35 ± 6	34 ± 7	0.771
PaO2/FiO2	318 ± 95	356 ± 97	286 ± 83	<b>0.029</b>
Chest CT				
CT score (available in 34/42)	7 (3, 11)	4 (2,8)	10 (7, 13)	<b>0.027</b>
LUS				
LUS score	6 (3, 10)	3 (1, 5)	10 (6, 14)	<b>&lt; 0.001</b>

## Characteristics of population according to LUS findings

On subgroups analysis, patients who had higher LUS score were older (79 vs 60 years,  $p < 0.001$ ) with similar prevalence of CVD (41% vs 25%,  $p = 0.275$ ) and other comorbidities, whereas blood examinations showed higher CRP and creatinine (7.2 vs 1.3,  $p > 0.001$  and 1 vs 0.72,  $p = 0.014$  respectively) and lower PaO2/FiO2 (286 vs 356,  $p = 0.029$ ) as compared with patients with lower LUS score (Fig. 1). On CT examination, significantly higher scores were measured in patients with higher LUS score (10 vs 4,  $p = 0.027$ ); Fig. 2 depicts LUS and CT findings in a representative patient. Rate of ace-inhibitor or angiotensin receptor blocker intake before hospital admission was similar between LUS subgroups.

## Analysis of relationships

Analysis of relationships results are summarized in Table 2 and Fig. 3. We observed a significant correlation between LUS and CT scores ( $r = 0.390$ ,  $p = 0.023$ ). Both LUS and CT scores correlated directly



with patients age ( $r = 0.586$ ,  $p < 0.001$  and  $r = 0.399$ ,  $p = 0.021$  respectively) as well as CRP ( $r = 0.472$ ,  $p = 0.002$  and  $r = 0.518$ ,  $p = 0.002$  respectively), and inversely with PaO<sub>2</sub>/FiO<sub>2</sub> ( $r = -0.485$ ,  $p = 0.003$  and  $r = -0.440$ ,  $p = 0.017$  respectively). On the other hand, only LUS score had significant correlation with hs-Troponin T, Nt-pro-BNP and creatinine ( $r = 0.433$ ,  $p = 0.019$ ;  $r = 0.411$ ,  $p = 0.027$  and  $r = 0.497$ ,  $p = 0.001$  respectively), whereas CT score had none (all  $p > 0.05$ ).

Table 2

Univariable correlation between LUS and CT score with clinical and laboratory parameters. LUS (lung ultrasound), CVD (cardiovascular disease) CRP (C-reactive protein), CKD (chronic kidney disease), COPD (chronic obstructive pulmonary disease), WBC (white blood cells), NLR (neutrophil to lymphocyte ratio), Nt-pro-BNP (), CT (computed tomography).

Variable	LUS score (n = 42)		CT score (n = 34)	
	R	P	R	P
Age	0.586	<b>&lt; 0.001</b>	0.399	<b>0.021</b>
Sex (male)	-0.171	0.280	-0.040	0.822
Previous CVD	0.215	0.171	0.111	0.533
Ace-inhibitor therapy	0.045	0.819	0.146	0.496
WBC	0.153	0.347	0.140	0.436
Neutrophil	0.208	0.198	0.029	0.873
Lymphocyte	-0.175	0.279	0.219	0.221
NLR	0.241	0.135	-0.113	0.531
Creatinine	0.497	<b>0.001</b>	0.302	0.087
CRP (Log10)	0.472	<b>0.002</b>	0.518	<b>0.002</b>
D-dimer (Log10)	0.182	0.418	0.097	0.685
Hs-Troponin T (Log10)	0.433	<b>0.019</b>	0.131	0.541
NT-pro-BNP (Log10)	0.411	<b>0.027</b>	0.163	0.437
PaO <sub>2</sub> /FiO <sub>2</sub>	-0.485	<b>0.003</b>	-0.440	<b>0.017</b>
CT score	0.390	<b>0.023</b>	NA	NA
LUS score	NA	NA	0.390	<b>0.023</b>

## Discussion

We reported LUS and CT findings in a cohort of patients affected by COVID-19. We observed a significant correlation between scores assessed by LUS and CT respectively. Higher scores by either technique

correlated directly with age and CRP, and inversely with PaO<sub>2</sub>/FiO<sub>2</sub>; however, LUS score only showed significant direct correlation with hs-troponin T, NT-pro-BNP and creatinine.

Previous studies in COVID-19 showed that LUS semi-quantitative evaluation provided useful diagnostic [14] and prognostic [15, 16] information, while it correlated well with worsening respiratory insufficiency [14]. In our sample we reported similar results, however, in comparison to others [14, 15] we observed lower values of LUS scores and higher PaO<sub>2</sub>/FiO<sub>2</sub>, possibly partly explained by the non-critical care setting of our population [9]. Of note, COVID-19 is featured by quite heterogeneous clinical behavior, ranging from asymptomatic cases to different degrees of flu-like symptomatology and bilateral pneumonia complicated by respiratory failure [5, 11]. Taken together these data suggest that LUS scoring could be a reliable index of disease severity across all the COVID-19 spectrum.

In the majority of the study population, we were able to provide comparison of LUS with chest CT results evaluated by a scoring system described in literature [12]. As already reported by others [17, 18], we observed significant agreement between techniques, indicating that both could be informative regarding lung and systemic involvement in these patients. Indeed, in our sample both correlated with markers of worsening respiratory insufficiency such as PaO<sub>2</sub>/FiO<sub>2</sub> and inflammation (CRP and D-dimer). Of note, LUS score only showed significant association with markers of cardiac and renal injury. Patients with COVID-19, especially those with older age included in our cohort [9], are characterized by high comorbidity burden including cardiovascular and renal diseases [19–21], which in turn can relate to pulmonary congestion through volume overload.

Previous studies showed that LUS can be more effective than conventional chest X-ray for diagnosis of acute heart failure [22], while it can identify lung congestion in the context of volume overload driven by acute kidney injury [23, 24]. Both conditions at LUS evaluation are characterized by diffuse B-lines. Accordingly, volume overload and lung congestion might at least partly explain why degree of cardiac and renal injury were related to LUS but not CT findings in our cohort. If on one hand this result reiterates the high specificity of a semi-quantitative chest-CT scoring system in evaluating COVID-19 lung pneumonia, on the other it suggests that LUS might potentially provide added value in these highly comorbid patients, in which pneumonia might not be the sole cause of dyspnea and respiratory insufficiency.

Our findings might carry potential clinical implications. COVID-19 is a highly contagious disease [25], for which dedicated multidisciplinary “COVID units” have been created for the safety management of affected patients [26]. Imaging testing constitutes a potential risk for healthcare personnel and its use should be accurately weighted to reduce the number of unnecessary examinations [27]. Our study showed that LUS could be used for pneumonia severity evaluation in COVID-19 providing results comparable to chest-CT in the assessment of lung and systemic inflammatory involvement, with the added advantage of being effectively executable bedside.

## Limitations

Our study should be read in light of several limitations, such as partly retrospective nature, limited sample size and non-critical care setting which can reduce generalizability of our findings. Associations we observed between imaging scoring and systemic COVID-19 involvement must be cautiously interpreted as hypothesis generating only, not allowing to draw patterns of existing cause-effect relationships. Larger prospective studies are needed to fully assess whether LUS could be reliably used as possible alternative to chest CT scan for the diagnosis COVID-19 and in-hospital monitor of disease evolution.

## Conclusions

In patients with COVID-19 pneumonia admitted to non-critical-care wards, semi-quantitative bedside LUS evaluation identified the subgroup of patients with worse respiratory failure and systemic inflammation with results comparable to chest-CT evaluation. Correlation of LUS score with markers of cardiac and renal injury suggests that LUS might contribute to a more comprehensive evaluation, potentially aiding detection of non-pneumonia related causes of respiratory insufficiency within this heterogeneous and highly comorbid population.

## Declarations

**Funding:** none

**Conflicts of interest/Competing interests:** none declared

**Ethics approval:** institutional review board approved the study

**Consent to participate:** the patients provided written informed consent to the used of data for research purpose

**Consent for publication:** the patients consented to the publication of anonymized data

**Availability of data and material:** data will be made available upon request

**Code availability:** not applicable

**Authors' contributions:** performed imaging tests (GP, FC, MD, SM), involved in the care of the patients (all authors), literature search (GP, FC, LA), wrote the draft of the manuscript (GP, FC, LA, MD) provided critical revision and approved the final version of the manuscript (all authors).

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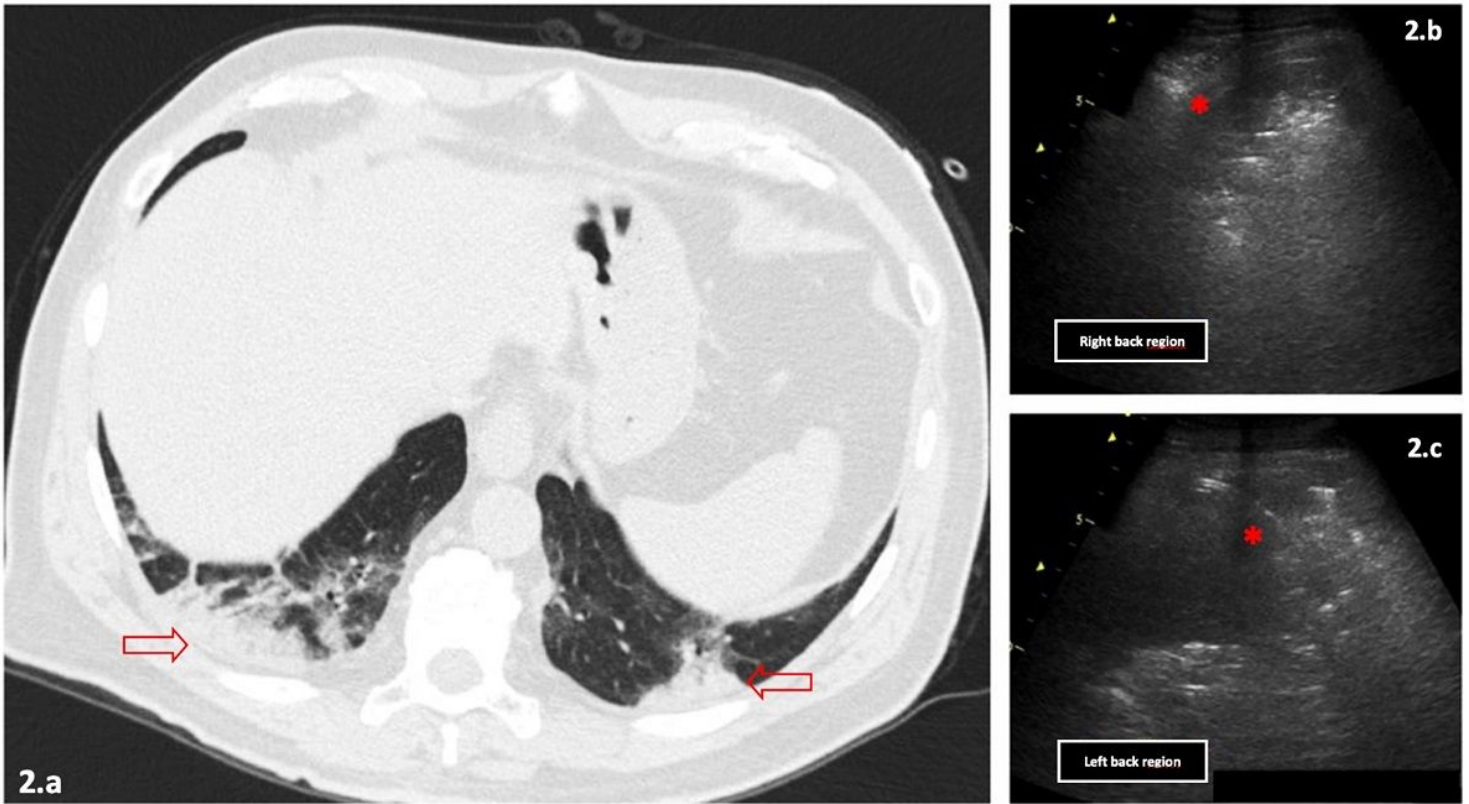
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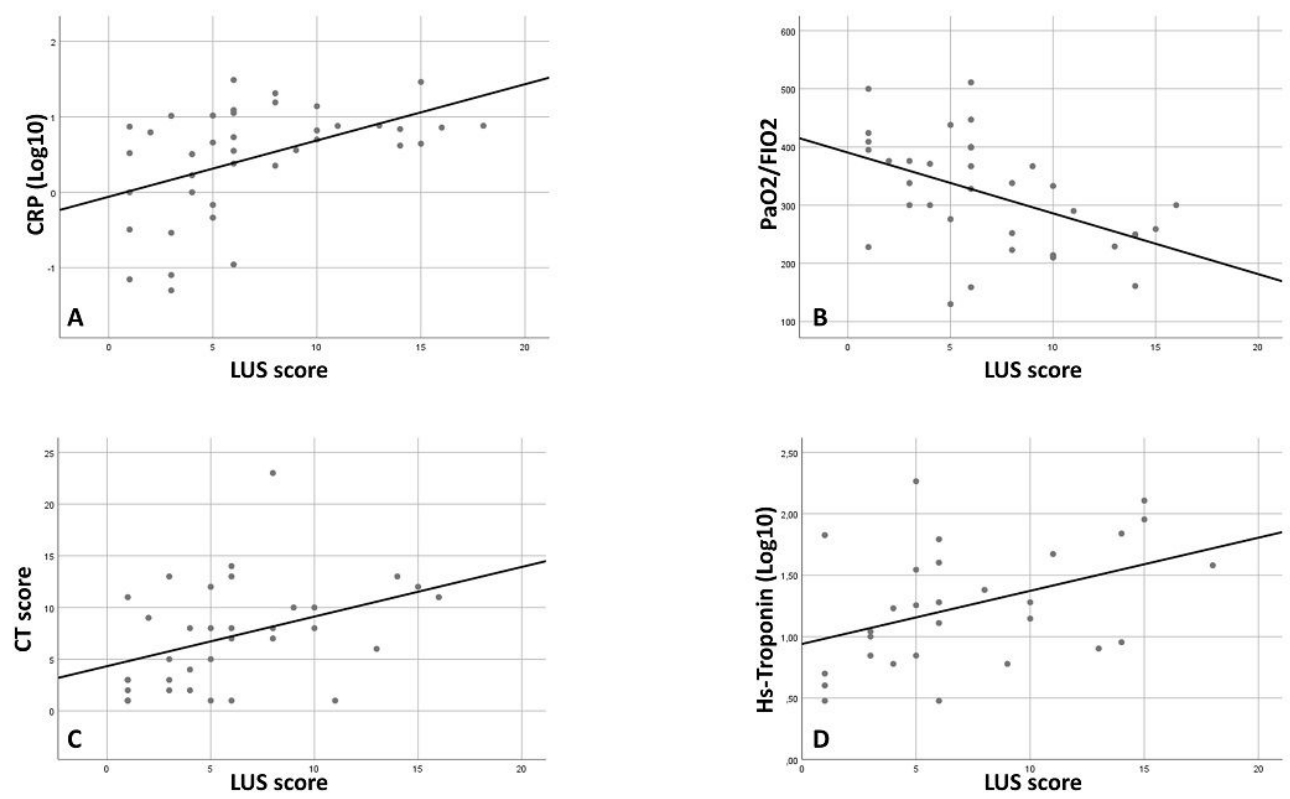
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## Figures



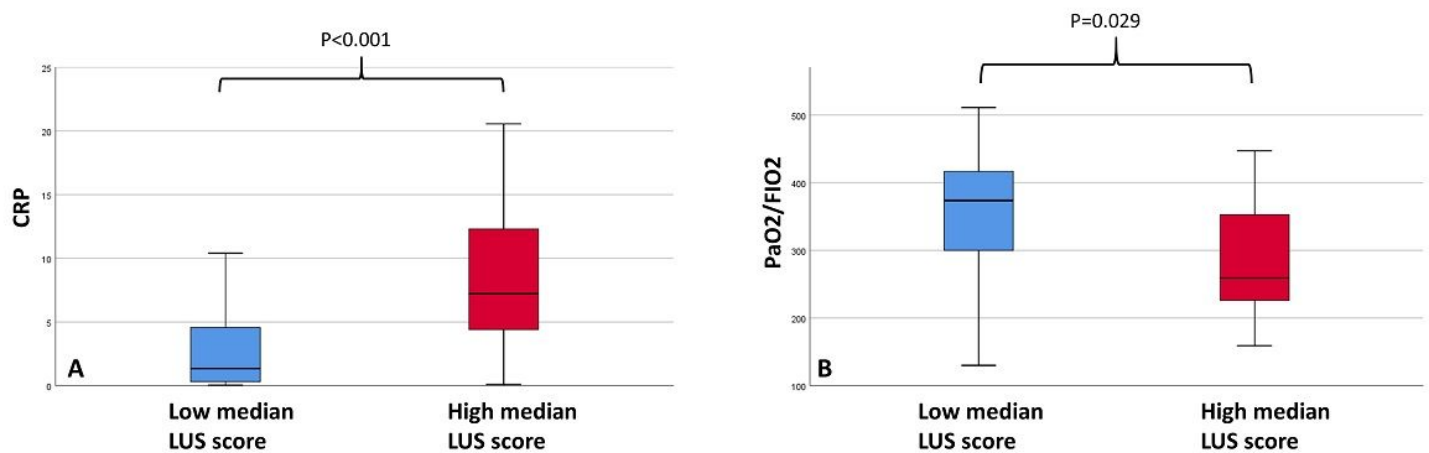
**Figure 1**

Boxplot shows significantly higher CRP (A) and lower PaO<sub>2</sub>/FIO<sub>2</sub> (B) in patient with high median LUS score.



**Figure 2**

Images from a 60-year-old man hospitalized for COVID-19 pneumonia. High resolution chest computed tomography revealed focal consolidation and ground-glass pleural opacities in both inferior lobes (red arrows in panel 2.a). Lung ultrasound confirmed the presence of consolidation areas in both posterior lung lobes (red asterisk in panel 2.b and 2.c).



**Figure 3**

Scatter plots showing correlations between LUS score and CRP (A), PaO<sub>2</sub>/FiO<sub>2</sub> (B), CT score (C) and hs-Troponin (D), all  $p < 0.05$ .