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Original article

PULMONARY ARTERY DIAMETER ON CHEST CT PREDICTS IN-HOSPITAL MORTALITY IN PATIENTS WITH COVID-19 PNEUMONIA

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Abstract: **Background:** Enlargement of the pulmonary artery (PA) could be helpful in risk stratification by the chest CT on the admission of COVID-19 patients.

Methods: This study aimed to associate PA diameter and overall mortality in COVID-19 pneumonia. We designed a retrospective study between January 2021 and May 2021 in tertiary-level hospitals in Gebze, Turkey. Subjects were evaluated in two groups according to their survivor status (survivors and non-survivors). Then biochemical, demographic, and clinical values were compared via the groups to define the predictive value of PA diameter on chest CT images.

Results: In the enrolled 594 COVID-19 in-hospital patients (median age was 45 (34-58) years, 263patients (44.3%) were female. 44 patients (7.4%) died during hospitalization. Multivariate Cox-proportion regression model yielded main PA ≥ 29 mm on admission showed that as independent predictors of death (long rank <0.001, median survival time 28 days). Cumulative survival rates were MPAD ≥ 29 mm 45% and < 29 mm 90% yielded (p < 0.001).

Conclusions: PA dilatation is strongly linked with in-hospital mortality in hospitalized patients with COVID-19 infection. Thus increased PA diameter on chest CT at admission may guide rapid and early diagnosis of high-risk patients.

Keywords*:* COVID-19, Computed tomography, pulmonary artery, mortality, pneumonia.

INTRODUCTION

The coronavirus 2019 (COVID-19) infection has become a global health problem that affects large populations in a short time over the world (1, 2). Its clinical presentation ranges from asymptomatic patients to acute respiratory failure, multiple system dysfunction, and death. It also impairs the vascular endothelial structure and function (3). Severe complications more frequently occur in advanced age, smoking, and comorbidities like hypertension (HT), diabetes mellitus (DM), cardiovascular disease, cardiac arrhythmia, dementia, cancer, chronic kidney, cerebrovascular, and respiratory disease (4, 5, 6). Chest computed tomography (CT) may have a crucial role in diagnosing and prognosis of this infection (7, 8). CT is widely used, especially in the emergency department, to make a risk assessment, and evaluate lung involvement and differential diagnosis. PA enlargement is a predictor of hemodynamic instability such as; right ventricular failure, pulmonary hypertension (PH), and embolism (9, 10, 11). Although PA dilatation reflects vascular injury, abnormal coagulation, hypoxia, and inflammation, the optimal cut-off value of PA diameter in COVID-19 patients is unknown. We hypothesized that the enlargement of PA could be helpful in risk stratification on the admission to hospital in the COVID-19 patient population. Therefore, we aimed to relationship PA diameters and in-hospital mortality of COVID-19 pneumonia.

MATERIAL and METHODS

Patients population

This study planned a retrospective and observational between January 2021 and May 2021. Five hundred ninety-four COVID-19 patients, diagnosed by real-time reverse transcriptase-polymerase chain reaction (RT-PCR) tests and non-cardiac gated thoracic CT scans, were enrolled in the study. Baseline laboratory findings were obtained from the hospital's electronic database system. Complete blood counts and biochemical parameters including blood glucose, creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT), high sensitive CRP (hs-CRP), ferritin, fibrinogen, D- Dimer, and high sensitive cardiac troponin I (hs-cTnI) were evaluated on admission. For patients under 18 years old, CT images

cannot be evaluated, pneumonia other than COVID-19 infection, non-hospitalized patients, and history of PH and thromboembolism were excluded. The study conforms to the principles in the Declaration of Helsinki and the local ethics committee's approval.

CT imaging

Thoracic CT imaging was performed using a 64-slice CT scanner (Aquilion 64, Toshiba Medical Systems, Japan) with 3-mm reconstructed slice thickness. CT images were obtained in the supine position, end of inspiration, and hands raised by the side. Tube current and voltages were 300 mA, and 120 kV, respectively, and gantry rotation time was 0.4s. All images were unenhanced and non-gated. The main PA diameter (MPAD), left PA diameter (LPAD), and right PA diameter (RPAD) were measured at the level of PA bifurcation from CT images by two cardiologists who were bound to the study (Figure 1).

Statistical analysis

Data were analyzed via the SPSS 22.0 version (SPSS Inc, Chicago, Illinois). The mean and standard deviation were used to describe continuous variables with normal distribution. Median, minimum, and maximum values were used to describe without normal distribution. Categorical variables were described with frequency and percentage. Continuous variables between two dependent groups were compared using Paired t-test and Wilcoxon t-test according to their distribution. Student t-test and Mann-Whitney U test were used to compare continuous variables with normal and without normal distribution respectively. Receiver operating characteristic (ROC) curve analyses were used for the optimal cut-off point of MPAD, LPAD, and RPAD. The area under the ROC curve (AUC) was reported with a % 95 confidence interval (CI). Pearson Chi-Square and Fisher's Exact tests were used in group comparison. Multivariable cox regression analysis was employed to assess the relationship between CT parameters (MPAD, LPAD, RPAD) and death as the outcome, summarized by hazard ratios (HR) and associated 95% confidence intervals. Survival analyses were calculated by the Kaplan-Meier method, and differences in the parameters were evaluated using a log-rank test. P-value was set at 0.05 in all statistical analyses.

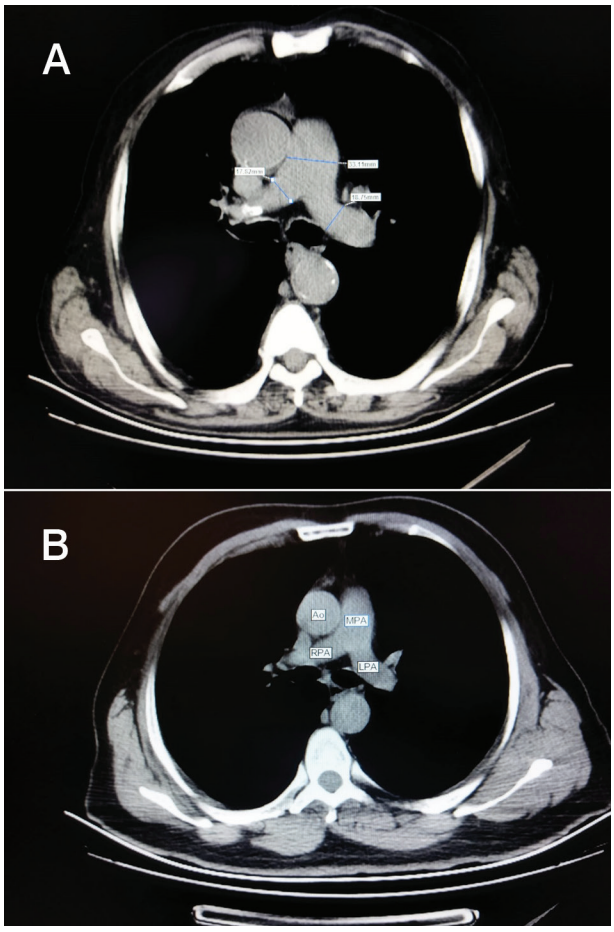


Figure 1. A-B: *From chest CT the diameter of the main, left and right pulmonary artery was measured at the level of bifurcation on the mediastinal window*

RESULTS

A total of 594 SARS-CoV-2 patients were hospitalized and divided into two groups according to their survival status [survivor (n = 550) and non-survivor (n = 44)]. Baseline characteristics, and clinical and laboratory parameters of the study population are demonstrated in Table 1. The median age was 45 (34-58), and 263 patients (44.3%) were female. One hundred eighty-five patients (31.1%) were smokers, 79 patients (13.3%) had DM, 133 patients (22.4%) had HT, 14 patients (2.3%) had congestive heart failure (CHF), and 66 patients (11.1%) had chronic obstructive pulmonary disease.

Non-survivors were older [median age 72 (63–80) vs 44 (33–55), $p < 0.001$] and had a higher prevalence of HT (50% vs 21.2%, $p < 0.001$), CHF (18.2% vs 1.1%, $p < 0.001$), coronary artery disease (CAD) [13.6% vs 2.9%, $p < 0.001$] and chronic obstructive pulmonary disease (34.1% vs 9.8%, $p < 0.001$). DM was similar frequency in the groups [18.2% vs 13.6%, $p = 0.397$]. Compared to survivors, non-survivors had higher fever [37.5 (38.3 - 36.8) vs 37.2 (36.4 - 38.0)C, $p = 0.019$], and heart rate [98 (91 - 106) vs 94 (89 - 102), $p = 0.04$], lower systolic blood pressure [110 ± 11 mm/Hg vs 114 ± 8 mm/Hg, $p = 0.002$], and lower oxygen saturation on admission [90 (83 - 97) vs 94 (91 - 97), $p < 0.001$]. On laboratory examination, non-survivors had higher fasting blood glucose [134 (106 - 235) vs 100 (87 - 115)

Table 1. *Baseline patient characteristics and clinical features of cohort*

Survival	Non-survival	Total	P value
Age (years) 45 ± 15	71 ± 13	47 ± 17	< 0.001
Gender n (%) (male/female) 44-56	45.5-54.5	41.1-55,9	0.85
DM, n (%) 71 (13.6)	8 (18.2)	79 (13.9)	0.397
HT, n (%) 111 (21.2)	22 (50)	133 (23.5)	< 0.001
CHF, n (%) 6 (1.1)	8 (18.2)	14 (2.5)	< 0.001
CAD, n (%) 15 (2.9)	6 (13.6)	21 (3.7)	< 0.001
CPD, n (%) 51 (9.8)	15 (34.1)	66 (11.6)	< 0.001
Smoking, n (%) 171 (32.7)	14 (31.8)	185 (32.6)	0.905
Saturation O (%) 94 ± 3	90 ± 7	93 ± 4	< 0.001
Fewer (D) 37.2 ± 0.8	37.5 ± 0.8	37.2 ± 0.8	0.019
Heart rate (mn) 96	98	96	0.053
SBP (mm/hg) 114.99 ± 8.96	110.16 ± 11.05	114.61 ± 9.22	0.002
MPA (mm) 25.74 ± 3.48	32.11 ± 4.45	26.23 ± 3.95	< 0.001
RPA (mm) 17.81 ± 3.25	24.11 ± 4.18	18.30 ± 3.73	< 0.001
LPA (mm) 17.65 ± 2.96	23.75 ± 3.77	18.12 ± 3.44	< 0.001
Glucose (mg/dL) 100 ± 89	134 ± 106	101 ± 90	< 0.001
Creatinine (mg/dL) 0.8 ± 0.7	1.2 ± 0.8	0.8 ± 0.7	< 0.001
BUN (mg/dL) 12 ± 10	33 ± 19	13 ± 10	< 0.001
AST (U/L) 23 ± 17	31.5 ± 23	24 ± 18	< 0.001
ALT (U/L) 22 ± 16	20 ± 13.5	22 ± 16	0.352
Troponin (ng/mL) 0.001 ± 0.003	0.03 ± 0.13	0.013 ± 0.042	< 0.001
Ferritin (µg/L) 100.1 ± 41.6	401.0 ± 153.5	109.0 ± 43.5	< 0.001
Hs-CRP (mg/L) 7.2 ± 1.7	93.2 ± 43.8	7.8 ± 1.9	< 0.001
WBC 103/µL 6708 ± 2677	11803 ± 6572	7108 ± 3436	< 0.001
Hemoglobin (g/dL) 13.6 ± 1.6	11.5 ± 2.6	13.4 ± 1.8	< 0.001
Thrombocyte (103/µL) 233 ± 81	245 ± 129	234 ± 85	0.344

CAD: coronary artery disease, CHF: chronic heart failure, CPD: chronic pulmonary disease, DM: diabetes mellitus, HT: hypertension, SBP: systolic blood pressure, MPA: Main pulmonary artery, RPA: Right pulmonary artery, LPA: left pulmonary artery, D: degree, mn: minute

mg/dL, p < 0.001], creatinine [1.2 (0.8 - 2.2) vs 0.8 (0.7 - 0.9) mg/dL, p < 0.001], AST [31.5 (23 - 46.5) vs 22 (17 - 30) U/L, p < 0.001], D-Dimer [1.2 (0.52 - 3.1) vs 0.37 (0.27 - 0.68) ng/ml], hs-CRP [93.2 (43.8 - 192) vs 7.4 (2 - 22.6) mg/L, p < 0.001], ferritin [401 (153.5 - 585) vs 98 (41 -220.1) ng/mL, p < 0.001], white blood cell count (WBC) [11.8 ± 6.5 vs 6.6 ± 2.6 x103/ml, p < 0.001], fibrinogen [447 (389 - 525) vs 382 (321 vs 446) mg/dl] and hs-cTnI [30 (9 - 132) vs 1 (0.1 - 3) pg/mL, p < 0.001] levels. However, hemoglobin levels [11.5 ± 2.6 vs 13.6 ± 1.6 g/dL, p < 0.001] were lower in non-survivors, and ALT levels were similar in both groups (20 (13.5 - 37.5) vs 22 (16 - 36) U/L, p = 0.352). MPAD [32.11 ± 4.45 vs 25.74 ± 3.48, p < 0.001] LAPD [23.75 ± 3.77 vs 17.65 ± 2.96, p < 0.001], and RPAD [24.11 ± 4.18 vs 17.81 ± 3.25, p < 0.001] were significantly higher in non-survivor group compared to survivor group. The mean hospital stay was 5 (3-7) days and hospitalization period was longer in non-survivor group [8 (4-12) days vs 5 (3-7) days (p < 0.001)].Cumulative survival rates were MPAD > 29 mm 45% and < 29 mm 90% respectively (p < 0.001) (Figure 2). Receiver operator characteristic curve of main, left and right PA diameter for predicting deaths. MPA ≥ 29 mm, with 79.55% sensitivity and 87.19% specificity. Area under the rock curve (AUC) was 0.879 (p < 0.001) (Figure 3) At cox's regression analysis adjusted with ages, comorbidities, oxygen saturation, fewer, hs-cTnI and inflammatory parameters were predicting in-hospital mortality (Figure 4, Table 2).

Table 2. *At cox's regression analysis adjusted with ages, comorbidities, oxygen saturation, fewer, hs-cTnI and inflammatory parameters were predicting in-hospital mortality*

Variable	HR [95% CL]	P value
MPA	1.252 [1.180-1.327]	< 0.001
MPA + age	1.168 [1.085-1.258]	< 0.001
MPA + age + HT + CAD	1.158 [1.072-1.250]	< 0.001
MPA + age + HT + CHF	1.156 [1.074-1.244]	< 0.001
MPA + age + HT + CPD	1.168 [1.081-1.262]	< 0.001
MPA + HT + sat + fewer	1.244 [1.161-1.332]	< 0.001
MPA + glucose + crea + CRP	1.217 [1.134-1.306]	< 0.001
MPA + hs-cTnI + fibrinogen + D-dimer	1.305 [1.033-1.650]	0.026

MPA: Main pulmonary artery, HT: hypertension, sat: saturation, CAD: coronary artery disease, CHF: chronic heart failure, CPD: chronic pulmonary disease, crea: creatinine.

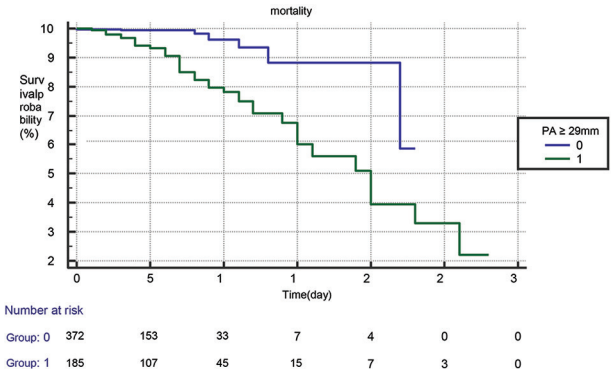
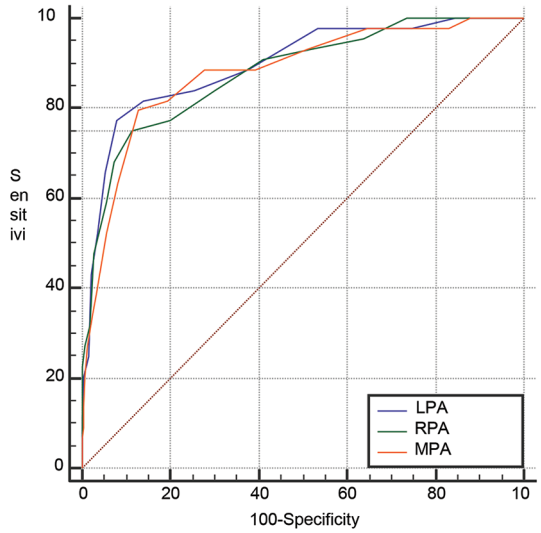
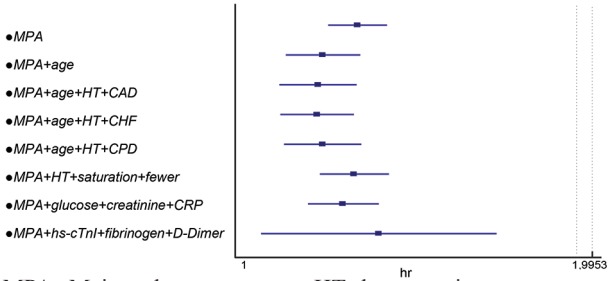


Figure 2. *Kaplan-Meier survival curves for PA trunk diameter showed that PA diameter ≥ 29 mm was significant predictor of mortality (long-rank p < 0.001, median survival time was 28 days)*



MPA: Main pulmonary artery, RPA: Right pulmonary artery, LPA: Left pulmonary artery

Figure 3. *Receiver operator characteristic curve of main, left and right pulmonary artery diameter for predicting deaths. MPA ≥ 29 mm, with 79.55% sensitivity and 87.19% specificity. Area under the rock curve (AUC) was 0.879, (p < 0.001)*



MPA: Main pulmonary artery, HT: hypertension, sat: saturation, CAD: coronary artery disease, CHF: chronic heart failure, CPD: chronic pulmonary disease, crea: creatinine.

Figure 4. *At cox's regression analysis adjusted with ages, comorbidities, oxygen saturation, fewer, hs-cTnI and inflammatory parameters were predicting in-hospital mortality*

DISCUSSION

The role of chest CT imaging in the COVID-19 infection is apparent as to determine the prevalence and severity of the disease, early screening, and making different diagnoses. In a study by Fang et al., the sensitivity of chest CT with COVID-19 was 98% (12). Multifocal bilateral distribution of ground-glass opacities, consolidations, air bronchogram, crazy-paving pattern, pulmonary vascular enlargement, linear opacification, and airway and pleural changes are the typical CT evidence of COVID-19 (7, 8).

We showed that MPAD ≥ 29 mm was an independent predictor of the severity of the COVID-19 infection. The enlargement of PA was considered to be a specific determinant of mortality and in-hospital duration and was found to be a negative correlation with the oxygen saturation at the time of admission. PA enlargement detected by CT imaging is a finding that helps predict worse outcomes (13). Although PA enlargement is associated with poor prognosis in acute pulmonary edema, embolism, and heart failure, insuf-

ficient data on its prognostic significance and optimal cut-off PA diameter in COVID-19 infection. A normally mean PA diameter calculated in a healthy population was 26.1±2.4 mm in men and 22.9±1.9 mm in women (14). This value was 25.74 ± 3.48 mm in the entire study group. A study conducted by Esposito et al., which included 1461 patients, determined that an MPAD ≥ 31 mm in COVID-19 patients was an independent predictor of mortality (15). The study by Zhu et al. demonstrated that MPAD ≥ 29 mm is a significant predictor of mortality (10). Truong et al. demonstrated that the predictive value of MPAD of 31 mm or greater in diagnosis PH and associated with 2-3 fold increased mortality risk compared to normal (11). In parallel, we found similar findings in our study cohort with an MPAD ≥ 29 mm, and this patient has more inflammation, heart injuries, and co-morbid disease. MPAD predicted worse outcomes in various regression models adjusted with age, comorbidities, clinical status, and inflammatory parameters.

COVID-19 negatively affects the endothelial system by activating multiple inflammatory, pro-thrombotic, and thrombotic cascades**.** Erdoğan et al. have suggested that disrupted endothelial system, increased inflammatory process, myocarditis, and active coagulopathy are linked with the severity of infection (16). Increased inflammatory status is accompanied by the severity of the infections, and high mortality (13, 16). It also caused deterioration in lung functions and a related increase in PA pressure. In addition, many patients had elevated inflammatory parameters, liver enzymes, CPK, and prothrombin time (13). Furthermore, Cai et al. demonstrated the increase in liver enzymes from severe pneumonia might be related to increased pulmonary pressure (17). In our cohort, similar to these results, AST and inflammatory levels; hs-CRP, ferritin, troponin, BUN, WBC, D-Dimer, and creatinine levels were significantly associated with PA diameter. Although thrombocytopenia is a common finding in COVID-19 patients in previous studies, no correlation was found between platelet count and PA diameter in our study (18, 19).

Although the etiology of PH is not known exactly, as possible causes; pulmonary small vessel thrombosis, vasculopathy, hypoxemia, and vasoconstriction were reported as the leading cause of PH in COVID-19 disease. PH can rapidly worsen right heart function and impair oxygenation, thus the length of hospital stay is prolonged, and the risk of the patient's multi-organ failure, bacterial infections, sepsis, hypercoagulation, and thrombosis**.** We found that severe CT findings of pneumonia and relation with hypoxemia were correlated with higher MPAD.

COVID-19 maybe affects the cardiovascular system. The underlying mechanism of cardiac damage is not clearly understood. Possible reasons are; increased cardiac stress due to acute respiratory distress and progressive hypoxemia, direct myocardial toxic effect of the COVID-19 increased inflammatory status, or their combination. In addition, SARS-CoV-2 infects host cells with angiotensin-converting enzyme 2 receptors and can cause myocardial damage. It has been shown that cardiovascular complications and heart failure may be responsible for 40% of deaths in COVID-19 patients (20).

There is a need for criteria to predict the prognosis of the disease in these patients. Thus increased MPAD may guide rapid and early diagnosis and treatment of high-risk patients.

In our study, pulmonary disease, CAD, CHF, and HT at the time of admission adversely affected the prognosis in COVID-19 patients. On the contrary, the presence of DM did not affect the prognosis in our patient population.

Limitation of the study

Although our study emphasized the association of PA diameter with mortality, there are several limitations. We did not have information about the clinical status of the patients before admission to the hospital. There was also no follow-up data. Follow-up and repeated measurements of the PA diameter will provide further information. Also, our cohort included only hospitalized patients, so it cannot be used for all patients. The frequency of pulmonary embolism that could lead to PA enlargement was unknown. Furthermore lack of data on electrocardiography and echocardiography imaging.

CONCLUSIONS

Chest CT imaging in the diagnosis of COVID-19 is obvious, simple, and of great value in early screening. A rapid diagnosis of high-risk COVID-19 patients is crucial, especially in order to dissolve the patient density in the emergency department. An enlargement of PA on chest CT may be an indicator of hemodynamic instability and worse outcomes. It should be considered that these patients may be at high risk, and should be evaluated carefully.

Abbreviation

ALT — Alanine aminotransferase
AST — Aspartate aminotransferase
CAD — Coronary artery disease
CT — Computed tomography
CHF — Congestive heart failure
DM — Diabetes mellitus
hs-CRP — high-sensitive CRP

HT — Hypertension
LPAD — Left pulmonary artery diameter
MPAD — Main pulmonary artery diameter
PA — Pulmonary artery
PH — Pulmonary hypertension
RT-PCR — Real-time reverse transcriptase-polymerase chain reaction test
RPAD — Right PA diameter
WBC — White blood cell count

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Sažetak

DIJAMETAR PLUĆNE ARTERIJE NA CT-u GRUDNOG KOŠA KAO PREDIKTOR MORTALITETA HOSPITALIZOVANIH PACIJENATA SA COVID-19 PNEUMONIJOM

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Uvod: Proširenje plućne arterije (PA) može biti od pomoći u stratifikaciji rizika pomoću CT grudnog koša pri prijemu pacijenata sa COVID-19.

Metode: Ova studija je imala za cilj da poveže prečnik PA i ukupni mortalitet kod pacijenata sa COVID-19 pneumonijom. Osmislili smo retrospektivnu studiju između januara 2021. i maja 2021. u bolnicama na tercijarnom nivou u Gebzeu, Turska. Ispitanici su evaluirani u dve grupe prema statusu preživelih (preživeli i nepreživeli). Upoređivane su biohemijske, demografske i kliničke karakteristike između ovih grupa kako bi se definisala prediktivna vrednost prečnika PA na CT grudnog koša.

Rezultati: Od 594 hospitalizovanih pacijenata sa COVID-19 (srednja starost je bila 45 (34-58) godina, 263 pacijenta (44,3%) su bile žene. 44 pacijenta (7,4%) su umrla tokom hospitalizacije. Na osnovu multivarijantnog regresijskoga modela Cox proporcije, PA ≥ 29 mm pri prijemu pokazao se kao nezavisni prediktori smrti (long rank < 0,001, srednje vreme preživljavanja 28 dana). Kumulativne stope preživljavanja bile su MPAD ≥ 29 mm 45% i < 29 mm 90% (p < 0,001).

Zaključak: Dilatacija PA je usko povezana sa bolničkim mortalitetom kod hospitalizovanih pacijenata sa infekcijom COVID-19. Povećani prečnik PA na CT-u grudnog koša pri prijemu može omogućiti brzu i ranu detekciju visokorizičnih pacijenata.

Ključne reči: COVID-19, kompjuterizovana tomografija, plućna arterija, mortalitet, pneumonija.

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