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

Baytugan Zafer Nart, Celik Inan Aziz, Bezgin Tahir

Department of Cardiology, Gebze Fatih State Hospital, Kocaeli, Turkey

Primljen/Received 22.05.2022.
Prihvaćen/Accepted 06.07.2022.

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, 263 patients (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.

**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) 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-cTnl [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).

**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, insufficient 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). Tuong 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

Acknowledgments: No

Conflict of Interests: The authors declare no conflicts of interest related to this article.

Funding: None

Licensing
This work is licensed under a Creative Commons Attribution 4.0 International (CC BY 4.0) License

Sažetak

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

Baytugan Zafer Nart, Celik Inan Aziz, Bezgin Tahir

Department of Cardiology, Gebze Fatih State Hospital, Kocaeli, Turkey

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

Rezultati: Od 594 hospitalizovanih pacijenata sa COVID-19 (srednja starost 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 prediktor 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)


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

REFERENCES


*Accepted papers are articles in press that have gone through due peer review process and have been accepted for publication by the Editorial Board of Sanamed. The final text of the article may be changed before the final publication. Accepted papers can already be cited using the year of online publication and the DOI, as follows: the author’s last name and initial of the first name, article title, journal title, online first publication month and year, and the DOI. When the final article is assigned to volumes/issues of the journal, the Article in Press version will be removed and the final version will appear in the associated published volumes/issues of the journal. The date the article was made available online first will be carried over.

**How to cite this article:** Baytugan ZN, Celik IA, Bezgin T. Pulmonary artery diameter on chest CT predicts in-hospital mortality in patients with COVID-19 pneumonia. Sanamed. Online First, August 2022. Doi: 10.5937/sanamed17-38017

**Correspondence to/Autor za korespondenciju**

Nart Zafer Baytugan, MD

**Address:** Department of Cardiology, Gebze Fatih State Hospital, Osman Yilmaz Neighborhood, Istanbul Street, 127, Kocaeli, Turkey

**E-mail:** nartzaf@ hotmail.com

**Telephone number:** +902625022240

**ORCID:** 0000-0003-4732-9367

**TABLE AND FIGURE LEGENDS**

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

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

**Figure-1:** From chest CT the diameter of the main left and right PA was measured at the level of bifurcation on the mediastinal window.
**Figure-2:** Kaplan-Meier analysis revealed that PA diameter >29 mm was a significant predictor of mortality (p<0.001, median survival time was 28 days).

**Figure-3:** 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. [(AUC) was 0.879. (p<0.001)].

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

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

<table>
<thead>
<tr>
<th>Survival</th>
<th>Non-survival</th>
<th>Total</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>45±15</td>
<td>71±13</td>
<td>47±17</td>
</tr>
<tr>
<td>Gender n(%)</td>
<td>44-56</td>
<td>45.5-54.5</td>
<td>41.1-55.9</td>
</tr>
<tr>
<td>DM, n(%)</td>
<td>71 (13.6)</td>
<td>8 (18.2)</td>
<td>79 (13.9)</td>
</tr>
<tr>
<td>HT, n(%)</td>
<td>111 (21.2)</td>
<td>22 (50)</td>
<td>133 (23.5)</td>
</tr>
<tr>
<td>CHF, n(%)</td>
<td>6(1.1)</td>
<td>8 (18.2)</td>
<td>14(2.5)</td>
</tr>
<tr>
<td>CAD, n(%)</td>
<td>15(2.9)</td>
<td>6 (13.6)</td>
<td>21(3.7)</td>
</tr>
<tr>
<td>CPD, n(%)</td>
<td>51(9.8)</td>
<td>15(34.1)</td>
<td>66(11.6)</td>
</tr>
<tr>
<td>Smoking, n(%)</td>
<td>171 (32.7)</td>
<td>14 (31.8)</td>
<td>185 (32.6)</td>
</tr>
<tr>
<td>Saturation O(%)</td>
<td>94±3</td>
<td>97±7</td>
<td>99±4</td>
</tr>
<tr>
<td>Fewer(D)</td>
<td>37.2±0.8</td>
<td>37.5±0.8</td>
<td>37.2±0.8</td>
</tr>
<tr>
<td>Heart rate(mn)</td>
<td>96</td>
<td>98</td>
<td>96</td>
</tr>
<tr>
<td>SBP(mm/hg)</td>
<td>14.99±8.96</td>
<td>110.16±11.05</td>
<td>114.61±9.22</td>
</tr>
<tr>
<td>MPA(mm)</td>
<td>25.74±3.48</td>
<td>32.11±4.45</td>
<td>26.23±3.95</td>
</tr>
<tr>
<td>RPA(mm)</td>
<td>17.81±3.25</td>
<td>24.11±4.18</td>
<td>18.30±3.73</td>
</tr>
<tr>
<td>LPA(mm)</td>
<td>17.65±2.96</td>
<td>23.75±3.77</td>
<td>18.12±3.44</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>100±89</td>
<td>134±106</td>
<td>101±90</td>
</tr>
<tr>
<td>Creatinine(mg/dL)</td>
<td>0.8±0.7</td>
<td>1.2±0.8</td>
<td>0.8±0.7</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>12±10</td>
<td>33±19</td>
<td>13±10</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>23±17</td>
<td>31.5±23</td>
<td>24±18</td>
</tr>
<tr>
<td>ALT(U/L)</td>
<td>22±16</td>
<td>20±13.5</td>
<td>22±16</td>
</tr>
<tr>
<td>Troponin (ng/mL)</td>
<td>0.03±0.13</td>
<td>0.013±0.042</td>
<td>0.001±0.003</td>
</tr>
<tr>
<td>Ferritin (µg/L)</td>
<td>401.0±153.5</td>
<td>109.0±43.5</td>
<td>100.0±41.6</td>
</tr>
<tr>
<td>Hs-CRP (mg/L)</td>
<td>7.2±1.7</td>
<td>93.2±43.8</td>
<td>7.8±1.9</td>
</tr>
<tr>
<td>WBC 10³/µL</td>
<td>6708±2677</td>
<td>11803±6572</td>
<td>7108±3436</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>13.6±1.6</td>
<td>11.5±2.6</td>
<td>13.4±1.8</td>
</tr>
<tr>
<td>Thrombocyte (10³/µL)</td>
<td>233±81</td>
<td>245±129</td>
<td>234±85</td>
</tr>
</tbody>
</table>

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

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


Figure 1 A-B. The diameter of the main, left and right pulmonary arteries from chest CT was measured at the bifurcation level in the mediastinal window.
Figure 2. Kaplan-Meier analysis revealed that PA ≥ 29 mm was significant predictor of mortality. (p<0.001, median survival time was 28 days)
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. [AUC was 0.879. (p<0.001)]
Figure 4. At cox’s regression analysis adjusted with ages, comorbidities, oxygen saturation, fewer, hs-cTnI and inflammatory parameters were predicting in-hospital mortality.

- MPA
- MPA+age
- MPA+age+HT+CAD
- MPA+age+HT+CHF
- MPA+age+HT+CPD
- MPA+HT+saturation+fewer
- MPA+glucose+creatinine+CRP
- MPA+hs-cTnI+fibrinogen+D-Dimer