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Prognostic value of Mastora obstruction score in acute pulmonary embolism

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Background. To evaluate the clinical significance of Mastora obstruction score in hemodynamically stable patients with acute pulmonary embolism (aPE).

Materials and methods. One-hundred-and-six patients with newly diagnosed aPE, confirmed by computed tomography pulmonary angiography (CTPA), were included in the study and prospectively examined. aPE severity was assessed by using Mastora obstruction score. According to the Mastora index, patients were divided into "non-massive" and "massive" groups. The patients' medical histories and blood laboratory data were collected, and instrumental tests were performed and analyzed.

Results. Eighty-two (77%) of the patients had "non-massive" aPE. Cough (48%), fever (44%), and pleural effusion (48%) occurred significantly more often in the "non-massive" PE group, while syncope (42%) and right ventricular dysfunction (86%) were more frequent in the "massive" PE group. The probability of the right ventricular dysfunction was significantly higher in the presence of increased pulmonary artery pressure (Cramer's $V = 0.410$; $p < 0.0001$) and respiratory failure (Cramer's $V = 0.247$; $p = 0.032$). Increased CRP level was found in the majority of the patients (90%). D-dimer level <500 μg/L (lower than the commonly recommended cut-off level) was found in 5% of cases.

Conclusions. The clinical manifestation depends on the massiveness of aPE. Division of aPE cases into two groups suggests two possible subtypes of aPE: cardiovascular and respiratory. The "non-massive" aPE was associated with respiratory symptoms and an inflammatory response. The "massive" aPE is associated with an increased D-dimer level and leads to cardiovascular disorders. However, the "massive" aPE may be presented by normal D-dimer concentration level.

Keywords: pulmonary embolism, Mastora, chest CTPA, CRP, D-dimer

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INTRODUCTION

Acute pulmonary embolism (aPE) is a life-threatening fatal disease with an annual incidence of 70 cases per 100 000 inhabitants (1, 2). It is the third most frequent cardiovascular disease with an overall annual incidence of 100–200 per 100 000 people (3). Mortality rate of patients not diagnosed and treated in a timely manner for aPE is as high as 30% and the risk of death is higher at 30 and 90 days after the first episode of PE (4–6). In addition, near 40% of cases of aPE and DVT are prone to recur (4, 7). The prognosis for aPE depends on many factors, such as individual response, the size and location of the clot, and comorbidities (8). Traditionally, massiveness of aPE has been defined on the basis of angiographic burden of emboli by use of the radiologic indexes designed for PE severity, such as Qanadli, Miller, or Mastora score (9, 10). The Mastora score is one of the latest radiologic indexes. The aim of the study was to analyze the significance of Mastora obstruction score in hemodynamically stable aPE.

MATERIALS AND METHODS

Study population

The study population consisted of 106 patients (43% males; mean age 68 ± 14 years) with newly diagnosed aPE between January 2010 and April 2013. All patients were examined at Vilnius University Hospital Santaros Klinikos. Inclusion criteria were legal age (≥18 years), an acute, symptomatic, and newly diagnosed PE with objective confirmation by chest CTPA. Exclusion criteria were hemodynamic instability, sepsis (which was diagnosed before aPE), recurrence of PE, surgery or trauma during the acute period (the last four weeks). During the study, the patients' medical histories was collected, blood laboratory (D-dimer and CRP level concentration) and instrumental tests such as transthoracic echocardiography, deep venous ultrasonography of the legs (duplex scanning), and chest CTPA data were performed and evaluated. The demographic features of the study population are shown in Table 1.

According to the Mastora score, patients were allocated to one of two groups: group I ("non-massive" aPE) – when pulmonary vascular obstruction was less than 50%, and group II ("massive" aPE group) – when pulmonary vascular obstruction was ≥ 50% of the pulmonary artery bed (9). A signed informed consent form was obtained from all participants. The study was approved by the local Bioethics Committee.

Table 1. The demographic features of the population

Risk factors	Number of patients	%
Age > 65 years	64	60.4
Smoking history	22	20.8
Deep venous thrombosis	71	67
Congestive heart failure *	33	31.1
Rhythm disturbances *	28	26.4
Malignancy	25	23.6
Obesity	23	21.7
Congestive respiratory failure	19	17.9
Bed rest	16	15.1
Surgery ** (during the last 3 months)	10	9.4
Trauma * (during the last 3 months)	7	6.6
Oral contraceptive therapy/Hormone replacement therapy	3	2.8

* 16 (15.1%) patients suffered from congestive heart failure and arrhythmias.

** 4 (3.8%) patients, who underwent surgery and had trauma (excluding patients who underwent surgery or trauma during last 4 weeks).

Methods

All study tests were performed within one week after aPE confirmation. During the study, chest CTPA was applied to all 106 (100%) patients. Chest CTPA was carried out with 64 computed tomography scans GE VCT (General Electric Healthcare, Milwaukee, WI, USA) using Omnipaque 350 intravenous contrast. aPE scope was evaluated using Mastora method (GE Advantage Workstation software, version 4.4_04.05_EXT_CTT_5.X) for all the study patients. For the purpose of evaluating the CT score of severity of aPE, the scoring system was applied to five mediastinal, six lobar, and 20 segmental arteries. The five mediastinal arteries comprised the pulmonary artery trunk, the right and left main pulmonary arteries, and the right and left interlobar arteries. The six lobar arteries included the right truncus anterior, the left upper lobe pulmonary artery (upper arterial

branch, i.e., the culminal branch), the right middle lobe pulmonary artery, the left upper lobe pulmonary artery (the lower arterial branch, i.e., the lingular artery), and the right and left lower lobe pulmonary arteries. The 20 segmental pulmonary arteries consisted of the three right and left upper lobe (upper division) segmental arteries, the two right middle lobe and left upper lobe (lower division) segmental arteries, and the five right and left lower lobe segmental arteries. The CT severity score was based on the percentage of the obstructed surface of each central and peripheral pulmonary arterial section using a 5-point scale (1: <25%; 2: 25–49%; 3: 50–74%; 4: 75–99%; 5: 100%). The maximum Mastora obstruction score value was 155 (9). All patients underwent deep venous ultrasonography of the legs (GE Logiq 6 ultrasound system) and 76 (71.6%) of them transthoracic echocardiography (GE Vivid 7 ultrasound system). The echocardiographic severity of aPE was defined by the presence of signs of right heart dysfunction, paradoxical movement of the interventricular septum and/or systolic pulmonary hypertension (11). The systolic pressure in the pulmonary artery on cardiac ultrasound was measured in 42 (55.3%) patients. The D-dimer concentration level was evaluated with quantitative immune-turbid-metric (latex agglutination) method. A concentration was considered to be increased when D-dimer >500 mcg/L 1). CRP was explored with a method of latex immune-analysis and the level was consider to be increased when CRP 5 mg/L (1).

Statistical analysis

Statistical analysis was performed with SPSS 20.0. Variables were derived and tested to confirm normal distribution using the Kolmogorov-Smirnov test. Comparisons were made by x test, between the two groups which were evaluated using Student's t-test or the Mann-Whitney U test. All correlation analyses were performed using Pearson's coefficient of rank correlation. Cramer's V test was used to detect the measure of association. Cramer's V is a measure of association between two nominal variables, giving a value between 0 and +1. This measure varies from 0 (corresponding to no association between the variables) to 1 (complete association) and can reach 1 only when the two variables are equal to each other. A value of $p < 0.05$ was accepted as statistically significant.

RESULTS

The Mastora obstruction score varied highly: from 0.65 to 100. Eighty-two (77%) of the patients had "non-massive" aPE, whereas 24 (23%) had "massive" aPE. Cough, fever, and pleural effusion occurred significantly more often in the "non-massive" aPE group. Syncope and right ventricular dysfunction were more frequent in the "massive" aPE group. Some clinical symptoms, such as dyspnea, weakness, and chest pain were statistically observed equally often in both groups (Table 2).

The Mastora obstruction score had a relatively weak association with right ventricular dysfunction (Cramer's $V = 0.335$; $p = 0.022$), syncope (Cramer's $V = 0.369$; $p = 0.002$) and paradoxical movement of the interventricular septum (Cramer's $V = 0.4$; $p = 0.007$). The Mastora index correlated positively with D-dimer level ($r = 0.249$; $p = 0.011$), but negatively with CRP ($r = –0.259$; $p = 0.012$). D-dimer concentration was in a range from 265 μg/L to 25150 μg/L (average 5059 μg/L). Ninety-seven (95%) of all the study patients had D-dimer level higher than 500 μg/L (above commonly recommended cut-off level). D-dimer concentration was significantly higher in the "massive" aPE group compared to the "non-massive" PE group. Importantly, five (5%) of all the patients had D-dimer level below 500 μg/L. One of these patients had thrombosis of the main pulmonary arteries, another one – of the lobar pulmonary arteries, while the remaining three – of the segmental and (or) sub-segmental pulmonary artery branches. Higher D-dimer concentration was associated with obesity (Cramer's $V = 0.345$; $p = 0.033$) and cancer (Cramer's $V = 0.529$; $p < 0.0001$). Increased CRP level was found in 85 patients (80% of all the study patients). CRP level was significantly higher in the "non-massive" PE group comparing with the "massive" aPE group patients. When CRP level was increased (>5 mg/L), 35 patients (41%) had fever. Only one patient had fever while CRP level was in the normal range (<5 mg/L). When CRP >5 mg/L, the opacity/consolidation was found in 27 (31.7%) cases, whereas CRP <5 mg/L – opacity/consolidation was found in one (4%) case. The CRP level is associated with fever (Cramer's $V = 0.439$; $p = 0.001$), hemoptysis (Cramer's $V = 0.35$; $p = 0.021$), CRP and opacities/consolidation on the chest CT scans (Cramer's

Table 2. Demographics and clinical features of aPE. Blood laboratory data and instrumental tests of patients of "non-massive aPE" and "massive aPE" groups

	"Non-massive aPE" group (*n* = 82)	**"Massive aPE" group (*n* = 24)**	***p***
Age (years)	Min 38 Max 93 Mean 67.5 ± 14.6	Min 37 Max 89 Mean 69 ± 14	0.935
D-dimer (μg/L)	Min 265 Max 25150 Mean 4531 ± 4685	Min 1305 Max 20500 Mean 6872 ± 5775	0.638
CRP (mg/L)	Min 1.1 Max 216 Mean 60 ± 52	Min 9.5 Max 99 Mean 36 ± 28	0.130
Dyspnea	72 (88%)	23 (96%)	0.257
Weakness	51 (62%)	16 (67%)	0.690
Chest pain	46 (56%)	10 (42%)	0.121
Cough	39 (48%)	4 (17%)	0.007
Fever	36 (44%)	3 (13%)	0.005
Haemoptysis	16 (20%)	1 (4%)	0.072
Syncope	9 (11%)	10 (42%)	0.001
Right ventricular dysfunction	27 (50%)	19 (86%)	0.003
Pleural effusion	39 (48%)	6 (25%)	0.049
Opacity/consolidation on chest CT	26 (32%)	5 (21%)	0.303

$V = 0.419$; $p = 0.002$). For 31 (29%) of the patients, opacities or consolidation on the chest CTPA scans and for 45 (42%) pleural effusion were found. Pleural effusion was statistically significantly associated with fever (Cramer's $V = 0.295$; $p = 0.002$), but there was no reliable association to CRP. Hemoptysis occurred more frequently when opacities/consolidation were found in CT scans (Cramer's $V = 0.228$; $p = 0.019$). Pleural effusion increased the probability of fever (Cramer's $V = 0.295$; $p = 0.002$), but there was no reliable correlation with CRP. Patients with pleural effusion had a higher probability of rhythm disorders (Cramer's $V = 0.221$; $p = 0.023$) and respiratory failure (Cramer's $V = 0.246$; $p = 0.011$). The systolic pressure in the pulmonary artery on cardiac ultrasound was elevated in 85% cases. The mean pressure of the pulmonary artery was 47 mmHg. Right ventricular dysfunction was detected for 46 (74%) patients, paradoxical motion of interventricular septum for 6 (8%) patients. A higher pulmonary artery systolic pressure was associated with a higher probability of the right ventricular dysfunction (Cramer's $V = 0.410$; $p < 0.0001$) and the paradoxical motion of interventricular septum (Cramer's $V = 0.236$; $p = 0.039$). Patients with rhythm disorders had a higher probability of dyspnea (Cramer's $V = 0.204$; $p = 0.036$). The higher probability of the right ventricular dysfunction was relatively moderately associated with increased pulmonary artery pressure (Cramer's $V = 0.410$; $p < 0.0001$) and presented with respiratory failure (Cramer's $V = 0.247$; $p = 0.032$). During the study period, PE resulted in the death of two (2%) patients. One patient died on the 7th day (Mastora score 6.45), and the second patient on the 20th day (Mastora score 0.65) after the PE diagnosis was confirmed. There was a statistically higher probability of death when there was congestive heart failure (Cramer's $V = 0.206$; $p = 0.034$), DVT (Cramer's $V = 0.198$; $p = 0.042$), opacities/consolidation in CT scans (Cramer's $V = 0.216$; $p = 0.026$), or smoking in a life history (Cramer's $V = 0.271$; $p = 0.005$) and aPE.

DISCUSSION

For decades, clinicians have been taught that aPE – defined by the National Institute of Health as a "sudden blockage in a lung artery" – always matters and to be vigilant because a missed embolism can be fatal. When a patient presents with shortness of breath, pleuritic chest pain, tachycardia, or signs of right heart strain, clinicians are trained to

think "it may be the pulmonary embolism" (12). The fear of missing a diagnosis of this life-threatening disease has led to increased application of invasive diagnostic strategies, with a significant rise in the use of CTPA over the last decade (13). Therefore, increased accessibility of chest CTPA led to the detection of more PE cases (12). After a while, clinicians noticed that clinical manifestation of aPE may have no symptoms and manifest in different ways (14). The aim of the study was to evaluate the role of the Mastora obstruction score in hemodynamically-stable aPE patients. According to the Mastora obstruction score, aPE was classified into two groups ("non-massive – less than 50% obstruction and "massive" – 50% and more obstruction) and compared. The key findings in our study are: (i) clinical and laboratory manifestation of aPE depends on "massiveness" of the thrombosis of the pulmonary arteries; (ii) "massive" aPE more often presented with syncope, right ventricular dysfunction, higher level of D-dimer; (iii) "non-massive" aPE mostly presented with cough, fever, pleural effusion, higher level of CRP; (iv) the normal level of D-dimer did not exclude aPE, even massive one. Our study showed statistically significant differences between clinical features of "massive" aPE compared with the "non-massive" PE group. Some symptoms such as syncope, dyspnea, weakness, chest pain, cough, fever, or hemopthysis can occur in both groups, but some of them are detected more often when emboli have obstructed the pulmonary trunk or its main branches (8). We noticed that syncope, dyspnea, and weakness more often occurred in the "massive" PE group whereas cough, chest pain and fever in the "non-massive" group. Some authors have shown that clinical cardiac consequences become apparent when >30–50% of the pulmonary arterial bed is occluded by emboli (3, 8). The current opinion suggests replacing potentially misleading terms such as "massive", "submassive", and "non-massive" with the estimated level of the risk of aPE-related early death (3). However, our study showed that clinical manifestation depends on the massiveness of the thrombosis. We noticed that there were some clinical and laboratory mismatches between the "massive" and "non-massive" PE groups. According to the scope of the thrombosis, we found different clinical manifestation of PE: "massive" PE is more often associated with an increased D-dimer level and leads to cardiovascular disorders such as syncope and right ventricular dysfunction. Pulmonary artery pressure and pulmonary vascular resistance increase proportionately to the increased flow when a main pulmonary artery or pulmonary trunk is acutely obstructed by emboli and as a consequence we could have violent response – right heart failure (15, 16). Duplyakov et al. found that syncope may be a criterion of a high risk of fatal complications of PE, influenced by emboli (8). Comparing "massive" PE group with the "non-massive" group, patients in the first group had a higher level of D-dimer, but a lower CRP level. These clinical and laboratory findings were suspected. The level of D-dimer concentration shows what is happening during thrombosis: if a higher percentage of pulmonary arteries are obstructed, then fibrinolysis occurs more intensively and, as a result, we have a higher D-dimer level (17, 18). Meanwhile, our study clarified that "non-massive" PE is more associated with respiratory symptoms (such as a cough, hemoptysis, and pleural effusion) and an elevated CRP level. When the pulmonary artery is occluded by emboli, venous blood pressure increases in the preocluded region and may cause hemorrhage in the alveoli and cough with hemoptysis occurs as a response. If embolus occludes only distal or sub-segmental branch of the pulmonary artery, it does not disturb the main blood flow and has no effect on the pressure in the main pulmonary artery, it could still increase systemic inflammatory reaction. Elevated CRP is an inflammatory response to the process of the thrombosis, influenced by released cytokines and neurohumoral factors (16). Folsom showed that elevated CRP is independently associated with an increased risk of VTE (19, 20). It is our accidental study finding: we did not investigate the neurohumoral way during our research and it needs further investigations. All these findings reveal two possible subtypes of PE: respiratory and cardiovascular, respectively. Our study showed that in a small number of cases PE could be diagnosed when D-dimer concentration was below 500 μg/L. This finding was comparable to Wouter's and Goldhaber's data that emboli in subsegmental artery branches may be misdiagnosed when D-dimer concentration level is used as a single test to exclude thrombosis (17, 21). The controversial study by Pierrer and others

showed that plasma D-dimer concentration below 500 μg/L allows the exclusion of PE (22).

Our study has several limitations. Firstly, it had a relatively low number of patients. Nevertheless, it was prospectively investigated and the study population was typical of everyday practice. Secondly, we did not use cardio-specific biomarkers such as TNI and BNP at the beginning of the study because it was not a standard procedure in our clinic several years ago. Since these biomarkers were performed for minority of the study patients, we could not make any further calculations and evaluate them as predictive markers for the mortality rate.

CONCLUSIONS

This study shows that clinical manifestation depends on the massiveness of PE. Moreover, in a small number of cases "massive" aPE may be presented by normal D-dimer concentration level. Dividing aPE cases into two groups (less or more than 50% of the pulmonary artery bed) suggests two possible subtypes of aPE: cardiovascular and respiratory. "Non-massive" aPE was associated with respiratory symptoms and an inflammatory response. "Massive" aPE is associated with increased D-dimer level and leads to cardiovascular disorders, such as syncope and right ventricular dysfunction. Further investigations are needed to clarify this suggestion. However, it should be taken into account that, at present, the Mastora obstruction index itself does not impact the choice for antithrombotic treatment.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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References

1. Hawkins D. Pharmacoeconomics of thrombosis management. Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy. 2004 Jul 1; 24(7P2): 95S–9S.
2. Bates SM, Jaeschke R, Stevens SM, Goodacre S, Wells PS, Stevenson MD, et al. Diagnosis of DVT: antithrombotic therapy and prevention of thrombosis: American College of Chest Physicians evidence-based clinical practice guidelines. CHEST Journal. 2012 Feb 1; 141(2_suppl): e351S–418S.
3. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galie N, et al. 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014 Aug 28: ehu283.
4. Murin S, Romano PS, White RH. Comparison of outcomes after hospitalization for deep venous thrombosis or pulmonary embolism. Thromb Haemost. 2002; 88(3): 407–14.
5. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979–1998: an analysis using multiple-cause mortality data. Arch Intern Med. 2003 Jul 28; 163(14): 1711–7.
6. Nijkeuter M, Tick LW, Kamphuisen PW, Kramer MH, Laterveer L, van Houten AA et al. The natural course of hemodynamically stable pulmonary embolism: clinical outcome and risk factors in a large prospective cohort study. CHEST Journal. 2007 Feb 1; 131(2): 517–23.
7. Kearon C. Natural history of venous thromboembolism. Circulation. 2003 Jun 17; 107(23 suppl 1): I–22.
8. Duplyakov D, Kurakina E, Pavlova T, Khokhlunov S, Surkova E. Value of syncope in patients with high-to-intermediate risk pulmonary artery embolism. Eur Heart J Acute Cardiovasc Care. 2014 Mar 11: 2048872614527837.
9. Mastora I, Remy-Jardin M, Masson P, Galland E, Delannoy V, Bauchart JJ et al. Severity of acute pulmonary embolism: evaluation of a new spiral CT angiographic score in correlation with echocardiographic data. Eur Radiol. 2003 Jan 1; 13(1): 29–35.
10. Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, Goldhaber SZ, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension a scientific

statement from the American Heart Association. Circulation. 2011 Apr 26; 123(16): 1788–830.
11. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography: endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr. 2010 Jul 31; 23(7): 685–713.
12. Wiener RS, Schwartz LM, Woloshin S. When a test is too good: how CT pulmonary angiograms find pulmonary emboli that do not need to be found. BMJ. 2013 Jul 2; 347: f3368.
13. Bokobza J, Aubry A, Nakle N, Vincent-Cassy C, Pateron D, Devilliers C, et al. Pulmonary Embolism Rule-out Criteria vs D-dimer testing in low-risk patients for pulmonary embolism: a retrospective study. Am J Emerg Med. 2014 Jun 30; 32(6): 609–13.
14. Stein PD, Beemath A, Matta F, Weg JG, Yusen RD, Hales CA, et al. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med. 2007 Oct 31; 120(10): 871–9.
15. Piazza G. Beyond Virchow's Triad: does cardiovascular inflammation explain the recurrent nature of venous thromboembolism? Vascular Medicine. 2015 Apr 1; 20(2): 102–4.
16. Elliott CG. Pulmonary physiology during pulmonary embolism. Chest. 1992 Apr 30; 101(4): 163S–71S.
17. Goldhaber SZ, Elliott CG. Acute pulmonary embolism: Part I Epidemiology, pathophysiology, and diagnosis. Circulation. 2003 Dec 2; 108(22): 2726–9.
18. Wakefield TW, Myers DD, Henke PK. Mechanisms of venous thrombosis and resolution. Arterioscler Thromb Vasc Biol. 2008 Mar 1; 28(3): 387–91.
19. Piazza G, Goldhaber SZ. Pulmonary embolism in heart failure. Circulation. 2008 Oct 7; 118(15): 1598–601.
20. Folsom AR, Lutsey PL, Astor BC, Cushman M. C-reactive protein and venous thromboembolism: a prospective investigation in the ARIC cohort. Thromb Haemost. 2009 Oct; 102(4): 615.
21. De Monye W, Sanson BJ, Mac Gillavry MR, Pattynama PM, Büller HR, van den Berg-Huysmans AA, Huisman MV. Embolus location affects the sensitivity of a rapid quantitative D-dimer assay in the diagnosis of pulmonary embolism. Am J Respir Crit Care Med. (2002); 165(3): 345–8.
22. Perrier A, Desmarais S, Goehring C, de Moerloose P, Morabia A, Unger PF, et al. D-dimer testing for suspected pulmonary embolism in outpatients. Am J Respir Crit Care Med. 1997; 156(2): 492–6.

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MASTORA OBSTRUKCIJOS BALO PROGNOSTINĖ REIKŠMĖ ŪMINĖS PLAUČIŲ ARTERIJŲ TROMBINĖS EMBOLIJOS ATVEJU

Santrauka

Tikslas. Įvertinti radiologinio Mastora indekso klinikinę reikšmę ligoniams, sergantiems ūmine plaučių arterijų trombine embolija (ūPATE).

Darbo metodika. Į prospektyvinį tyrimą įtraukti 106 hemodinamiškai stabilūs pacientai, kuriems ūPATE patvirtinta atlikus krūtinės ląstos kompiuterinę tomografiją su angiografija (KT). Pagal apskaičiuotą Mastora obstrukcijos balą pacientai suskirstyti į „masyvios“ ir „nemasyvios“ ūPATE grupes. Vertinti ligos anamnezės, atliktų kraujo ir intrumentinių tyrimų duomenys.

Rezultatai. Aštuoniasdešimt du (77 %) ligoniai pateko į „nemasyvios“ ūPATE grupę. Kosulys (48 %), karščiavimas (44 %), skystis pleuros ertmėje (48 %) statistiškai patikimai dažniau pasireiškė „nemasyvios“ ūPATE ligonių grupėje. Sinkopė (42 %), dešiniojo skilvelio disfunkcija (86 %) – „masyvios“ ūPATE grupėje. Nustatyta, kad esant didesniam slėgiui plaučių arterijoje (Cramer's $V = 0{,}410$; $p < 0{,}0001$) ir kvėpavimo nepakankamumui (Cramer's $V = 0{,}247$; p 0,032), didėja dešiniojo skilvelio disfunkcijos tikimybė. Didžiajai daliai ligonių (90 %) nustatyta didesnė CRB koncentracija kraujyje. <500 ng/l D-dimerų koncentracija fiksuota 5 % atvejų.

Išvados. Klinikinė ūPATE raiška priklauso nuo ligos sunkumo. ūPATE skirstymas į grupes pagal Mastora indeksą padėjo nustatyti 2 ligos potipius: kardiovaskulinį ir respiracinį. „Nemasyvi“ ūPATE labiau susijusi su respiraciniais simptomais ir uždegiminiu atsaku, o „masyvi“ – su didesne D-dimerų koncentracija bei kardiovaskulinės sistemos sutrikimu. Normali D-dimerų koncentracija ne visada paneigia ūPATE.

Raktažodžiai: plaučių arterijų trombinė embolija, Mastora, krūtinės KT, CRB, D-dimerai