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Case review

Diagnostic and Therapeutic Approach to a Patient with High-risk Pulmonary Thromboembolism - a Case Report and Commentary in the Context of ESC Guidelines

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Abstract Pulmonary thromboembolism (PTE) is a common and potentially fatal disease for the diagnosis and treatment of which European Society of Cardiology (ESC) guidelines have been published in 2019. They emphasize the importance of risk stratification of early mortality in suspected or confirmed PTE and treatment according to the risk class: use of anticoagulant or systemic fibrinolytic therapy. The aim of this paper is to present a patient with typical electrocardiographic and echocardiographic findings stratified as high - risk PTE who was treated with systemic fibrinolytic therapy. A 70-year-old patient was admitted to the Intensive Care Unit of the Department of internal medicine of the Zajecar Health Center. On admisson he was hypotensive, dyspnoeic, diaphoretic with signs of organ hypoperfusion. Electrocardiographically, the S1Q3T3 sign and right bundle branch block (RBBB) were verified and a high-risk PTE suspected. Bedside echocardiographic exam revealed a large, mobile thrombotic mass that prolapses from the right atrium to the right ventricle. Systemic fibrinolytic therapy with alteplase with concomitant unfractionated heparin infusion was given. The patient hemodynamically stabilized with loss of electrocardiographic changes suggestive of PTE and thrombus resolution in the right heart chambers. The patient was discharged home after 12 days of hospital treatment. Early risk stratification of patients with PTE and development of a local protocol for diagnosis and treatment according to the risk class increases the success rate in recognizing and treating this disease and reduces the risk of death and other adverse clinical events.

Kew words high-risk pulmonary embolism, PTE, systemic fibrinolytic therapy, rTPA, UFH, hemodynamic instability, PESI

Introduction

Pulmonary thromboembolism (PTE) together with deep vein thrombosis (DVT) is the third most common cardiovascular syndrome, after myocardial infarction and stroke[1]. Epidemiological studies have suggested data on the incidence of PTE in the range of 39-115 per 100,000 population per year[2,3]. Clinical diagnosis is challenging despite the existence of developed diagnostic-therapeutic algorithms for PTE. In a retrospective clinical study of hospital-treated patients with a lethal outcome in whom a clinical autopsy was performed over a ten-year observation period in 502 patients (3.8% of the total autopsies), PTE was verified. Of these, in 328 patients it was understood as the main cause of death (fatal PTE) and in the remaining 174 as an associated disease (non-fatal PTE). Interestingly, the clinical diagnosis of PTE was considered antemortal in 48.2% of all cases with significantly more frequent clinical suspicion made in fatal than in nonfatal PTE (61.9 vs 22.4%)[4]. Of particular importance is the massive PTE, which in the latest ESC guidelines for acute pulmonary thromboembolism from 2019 is presented as a class of high-risk PTE in which it is necessary to conduct a rapid diagnostic assessment and reperfusion therapy. In order for PTE to be perceived as high-risk, it is necessary that the patient be hemodynamically unstable: after resuscitated cardiorespiratory arrest ; obstructive shock with systolic pressure below 90 mmHg with adequate volemia and signs of organ hypoperfusion (altered mental status, cold skin, oligo / anuria, increase in serum lactate level); systolic pressure maintained above 90 mmHg on vasopressor support with signs of organ hypoperfusion; persistent hypotension with systolic blood pressure below 90 or a drop of more than 40 mmHg than usual for particular person lasting more than 15 minutes and not caused by hypovolaemia, sepsis or arrhythmia. In such patients, parenteral anticoagulant therapy is given at the onset of clinical suspicion of PTE and an urgent diagnostic workup is performed - transthoracic echocardiography and / or CT pulmonary angiography. If the diagnosis is confirmed, it is considered a high-risk PTE and it is necessary to implement an aggressive therapeutic approach: systemic application of fibrinolytic therapy, mechanical or pharmacomechanical reperfusion with the introduction of a catheter

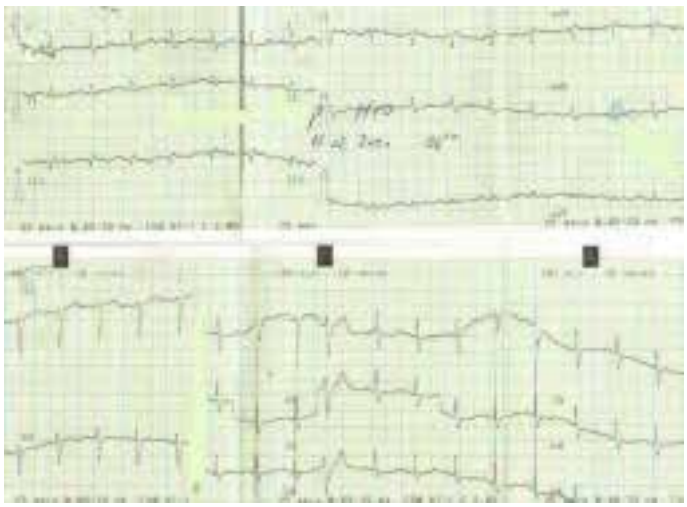

Figure 1

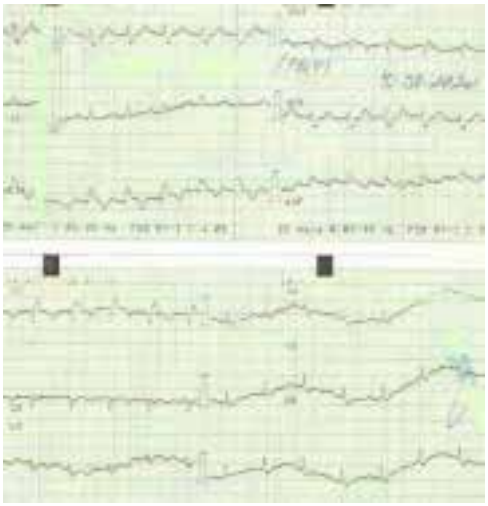

Figure 2

into the pulmonary artery (catheter directed therapy) or surgical embolectomy. The use of systemic fibrinolytic therapy is the most common method of treatment in patients without contraindications for it's use, mainly rTPA (alteplase)[5].

Case presentation

In this paper a male patient aged 70 years with a rare but specific electrocardiographic and echocardiographic presentation of acute high-risk PTE successfully treated with systemic fibrinolytic therapy at the Internal Department of the Health Center Zajecar during February 2020. The paper discusses the diagnostic-therapeutic approach to a specific patient in accordance with the latest published ESC guidelines for acute pulmonary thromboembolism. Special attention is paid to the discussion on the application of anticoagulant and fibrinolytic therapy in the early phase of treatment of the disease and the attempt to develop a local protocol for the treatment of PTE

A patient born in 1950, due to the sudden onset of general weakness, malaise and nausea, called the Emergency Medical Service (EMS). During the examination, it was concluded that the patient is hypotensive, and a discrete ST segment elevation of 0.5 mm in D3, aVF leads is verified on the performed ECG recording. The EMS physician suspected the acute coronary syndrome and referred him to the Internal Department of Health center Zajecar. The patient denied any chest pain since the onset of symptoms. Blood presurre was 80/50 mmHg at presentation and the patient was anuric. Electrocardiography revealed sinus tachycardia with a frequency of 120/min, with ST segment elevation up to 0.5 mm in D3, aVF leads but clear McGini White's - S1Q3T3 sign with a complete RBBB (Figure 1). Arterial blood gas analysis indicated hypocapnia with moderate metabolic acidosis and elevated lactate levels (pO2 78 mmHg; pCO2 22 mhg HCO3-11.3 mmol/l pH 7.22 lactate 8.4 mol/l). PTE was strongly suspected and a bolus of 10,000 I.U. unfractionated heparin (UFH) was immediately administered with continuous infusion at a dose of 1000 I.U./h. An emergency bedside transthoracic echocardiographic exam was performed in the Intensive Care Unit. Along with dilatation of the right heart chambers, it clearly verified the thrombotic mass that prolapses through the tricuspid valve (Figures 3 and 4). With all this in mind, the patient was stratified as a high-risk PTE and fibrinolytic therapy with alteplase was administered according to a two-hour protocol. In the further course, patient was hemodynamically stabilized, BP is 135/80 mmHg, HR 90/min, diuresis is established with symptomatic improvement. In the further course, the patient was treated with unfractionated heparin with the consequent introduction of vitamin K antagonists. Throughout hospitalization, the patient was hemodynamically stable, with regression of the characteristic electrocardiographic findings (Figure 2). Prior to discharge, the patient underwent a control echocardiographic exam which did not detect previously seen thrombotic mass, still withmoderate right heart chamber dilation (Figure 5). He was discharged from hospital after 12 days of treatment. During the outpatient follow-up of one year, there were no other clinically significant events other than hospitalization due to viral pneumonia.

Discussion

Acute coronary syndrome (ACS) is one of the most common differential diagnoses in patients with PTE, especially in the presence of significant ECG changes, especially ST segment elevation in one or more electrocardiographic leads. In the *Kukla et al*[6] study, of 292 patients with acute PTE, 2.7% were initially admitted and treated as ACS. In 71.2% of patients there were ECG changes suggestive of myocardial ischemia (negative T waves, ST segment depression / elevation). In the *Otto et al*[7] study, it was found that out of 123 patients with chest pain and ST segment elevation, as many as 59% had a non-ischemic aetiology. In a study of 171 patients presented to emergency triage services with chest pain and ST segment elevation, 56 had acute myocardial infarction, 50 unstable angina, and 65 (38%) noncoronary final diagnosis[8]. Thus, the ST segment elevation, even with associated chest pain, does not necessarily mean it is an acute myocardial infarction. *Wang et al*[9] provide

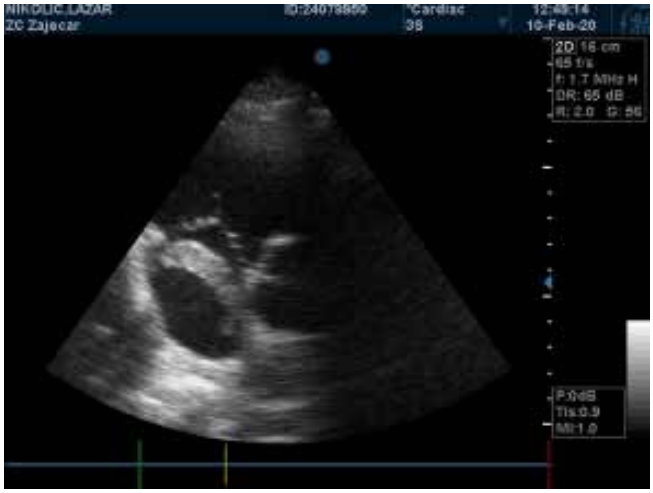

Figure 3

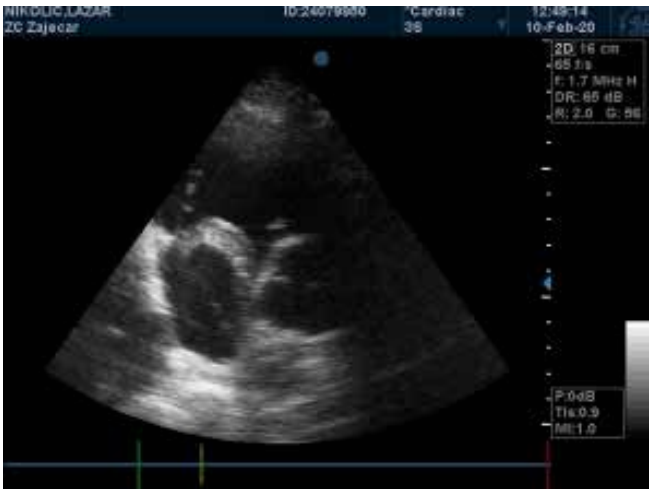

Figure 4

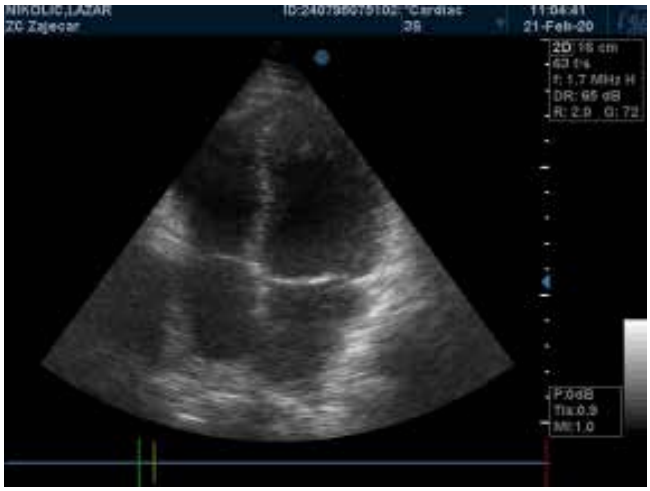

Figure 5

an overview of 12 conditions in which, in addition to myocardial infarction, the ST segment elevation may occur. Patient presented in this paper was reffered to Internal medicine department with suspected ACS. However, the absence of chest pain as well as reciprocal ST segment depression led us to consider alternative diagnoses. Sudden onset of discomfort and hypotension with 3 electrocardiographic findings: S1Q3T3 sign, RBBB and the presence of sinus tachycardia raised suspicion of acute PTE. The meta-analysis showed that the mentioned three findings together with ST segment elevation in aVR lead, negative T waves in V1-V4 and atrial fibrillation form the so-called "RV strain" which is associated with an increased risk of circulatory collapse and death in PTE. Patients with PTE and hemodynamic collapse or death were shown to have a significantly higher Daniel's electrocardiographic score than patients with PTE without these clinical events (5.9 SD +/- 3.9 vs 2.6 SD +/- 1.5)[10]. In our patient, this score was 7. It was previously shown that a score above 8 carries an increased risk of shock, respiratory failure, and death[11].

In our patient, the level of clinical suspicion of PTE was high despite the fact that there were no strong or moderate predisposing risk factors for venous thromboembolism as reported in the works of *Rogers et al*[12] and *Anderson and Spencer*[13]. The patient had only diabetes mellitus as a weak risk factor for PTE (OR <2).

Since high-risk PTE was suspected, a bedside transthoracic echocardiographic examination was performed in the Coronary care unit, in which, in addition to dilatation of the right heart cavities, a thrombus mass was directly visualized prolapsing through the tricuspid valve into the right ventricle. Echocardiographic or CT pulmonary angiography-seen mobile thrombi in the right heart chambers are seen in about 4% of unselected patients with PTE[14] but in one series of 131 patients with massive PTE treated in the Intensive care unit were seen in as many as 18% of patients by early echocardiography[15]. Mobile thrombi in the right heart practically confirm the diagnosis and are a marker of an increased risk of early mortality in PTE, especially in the presence of right ventricular dysfunction[16,17,18]. Considering clinical and echocardiographic findings, CT pulmonary angiography was not performed and the patient was stratified as a high-risk PTE in accordance with the latest ESC guidelines for acute pulmonary thromboembolism[19].

In patients with verified PTE, the level of risk of early mortality can be determined quickly and easily using prognostic risk scores. The most widely used and validated is the Pulmonary Embolism Severity Index (PESI) which combines clinical indicators of PTE severity and patient's comorbidities[20,21]. In our patient, the PESI score was 150, which placed him in the highest, V risk class in which the estimated 30-day mortality is 10-24.5%[22].

Given the confirmed PTE with signs of hemodynamic instability and the highest PESI risk class for early mortality, the patient was clearly stratified as high-risk PTE with minimal time delay to reperfusion therapy - 15 minutes from patient's presentation to the Department of Internal Medicine. In addition to supplemental oxygen therapy, the patient was cautiously volume challenged with crystalloid solutions in order to avoid the risk of reducing the minute volume of the left ventricle[23,24]. The guidelines state that in the presence of PTE with hemodynamic instability, the use of norepinephrine or dobutamine with class IIa recommendation may be considered[5]. Our judgment was that inotropic support was not necessary and was not applied.

It is recommended that the patients receive parenteral anticoagulant therapy at the initial suspicion of PTE, preferably low molecular weight heparin or fondaparinux

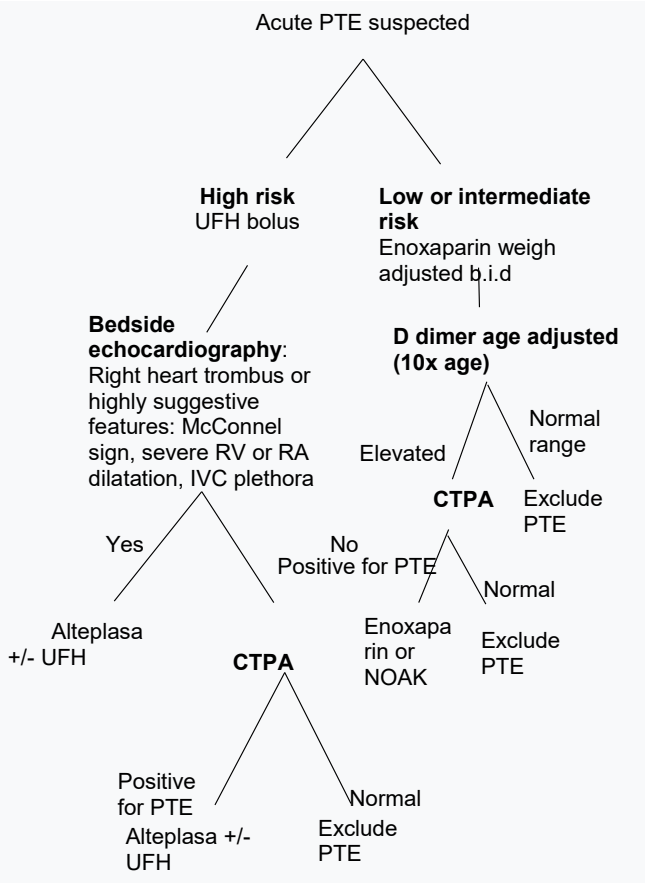

Figure 6

due to a lower risk of clinically significant bleeding and heparin-induced thrombocytopenia than when using unfractionated heparin (UFH) if low or intermediate risk PTE is suspected[5,23,24]. Our patient received a bolus of UFH with the ongoing continuous infusion until echocardiography was obtained, which is in accordance with the ESC guidelines for the treatment of patients with suspected high-risk PTE.[5] The recommended dosage of UFH is 80 I.U./kg immediately with continuous infusion of 18 I.U./kg/h and further dosing according to the activated partial thromboplastin time (aPTT)[27].

According to current ESC guidelines, systemic fibrinolytic therapy is the first choice in the treatment of patients with acute high-risk PTE in the absence of contraindications, while other treatment modalities (catheter directed therapy and surgical embolectomy) are used only in the presence of absolute contraindications or failure of systemic fibrinolysis and are reserved for centers with experience[5]. *Marty et al*[28] conducted a meta-analysis of 15 randomized trials involving 2,057 enrolled patients with PTE, with the first group receiving only parenteral anticoagulant therapy and the second receiving systemic fibrinolytic therapy. They showed that the application of systemic fibrinolytic therapy reduced overall mortality when all studies are included. There was no significant difference in terms of fibrinolytic used (alteplase; tenectaplase; other fibrinolytics, p = 0.86). When 4 studies involving patients with high-risk PTE were excluded from the analysis, the difference in total mortality among the observed groups was not statistically significant. Major bleeding data were available for 12 studies where the use of systemic fibrinolytic therapy carries a significantly higher risk of major bleeding (OR 2.91, p <0.0001). Alteplase administration carries a lower risk of bleeding than tenectaplase administration. However, after exclusion from the analysis of the study *Constantinides et al*[29], which used a more restrictive definition of major bleeding, the analysis of the remaining studies did not find a statistically significant difference between the applied fibrinolytics. In conclusion, it is stated that the use of systemic fibrinolytic therapy reduces PTE-related mortality, cumulative event death and treatment escalation and symptomatic recurrence of pulmonary thromboembolism at a price of an increased risk of fatal, intracranial and major bleeding. Therefore, it is recommended that reperfusion therapy be used only in high-risk PTE and in other categories only in case of hemodynamic deterioration[5].

Since there were no contraindications for the use of systemic fibrinolytic therapy, it was given to our patient, alteplasa 100 mg according to the protocol with simultaneous infusion of UFH, which was then continued after the end of fibrinolytic infusion. There are ambiguities as to whether UFH should be used during systemic fibrinolytic infusion. Alteplase has been approved by the US Food and Drug Administration (FDA) and the European Agency for the Evaluation of Medicinal Products (EMA) for the treatment of acute massive pulmonary embolism with discontinuation of UFH during fibrinolytic infusion and continued use only when aPTT falls below 2 times upper limit of normal[30,31]. The 2019 ESC guidelines for PTE suggest that UFH may be given concomitantly with fibrinolytic infusion in the case of rTPA but must be stopped with streptokinase and urokinase infusion, citing earlier european guidelines from 2014[32]. The 2008 American College of Chest Physicians (ACCP) guidelines state that it is acceptable to either continue or discontinue UFH infusion during the administration of fibrinolytic therapy since the two treatment approaches have never been directly compared[33]. The recommendations of the same society from 2016 do not discuss the simultaneous use of fibrinolytic and anticoagulant therapy[34]. Of the 4 randomized clinical trials in which alteplase was administered in a continuous infusion at a dose of 90-100mg, 3 concomitantly infused UFH at a dose of 1000-1500 I.U./h during fibrinolytic infusion and in one heparin was paused during fibrinolytic infusion[35]. This paper is also an attempt to develop a local treatment protocol for patients with PTE (Figure 6). When PTE is suspected, patients are stratified into those with suspected high-risk and the group with suspected non-high risk PTE. Patients with suspected high-risk PTE are placed in the Coronary Care Unit and receive a bolus of UFH 5000-10000 I.U. depending on body weight with continued infusion of 1000 I.U./h. Blood is sampled for aPTT before heparin is given. Then, bedside transthoracic echocardiography is performed and if it verifies the direct signs of PTE, fibrinolytic therapy with rTPA is given if there are no contraindications. The decision to give UFH during the fibrinolytic infusion is left to the attending physician. In the further course, the dose of UFH is optimized according to the values of aPTT so that it is in the range of 50-70s and oral anticoagulant therapy is introduced. If the echocardiographic

examination is non-conclusive, emergency CTPA is performed. If the test is negative for PTE, alternative clinical diagnoses are considered. If PTE is proven, rTPA is given if there are no contraindications.

In patients with established clinical suspicion of non-high risk PTE, enoxaparin s.c. 1mg / kg body weight on 12h interval is given and D dimer tested. If the age adjusted values are elevated the patient is referred for CTPA. If the D dimer is negative, the diagnosis of PTE is ruled out and an alternative disease is considered.

In rare situations, the existence of absolute contraindications for the use of fibrinolytic therapy decision is made depending on the specific clinical circumstances. Patients who are hemodynamically unstable, in addition to the use of anticoagulant therapy and supportive measures, are generally referred to the Clinic for Emergency Internal Medicine of the Military Medical Academy for the use of catheter directed treatment of PTE.

It is recommended that patients with PTE be treated with some of NOACs (Non-vitamin K antagonist oral anticoagulants) to prevent recurrent episodes of venous thromboembolism[5]. Due to the lack of motivation of the patient to take drugs of this group, oral anticoagulant therapy with vitamin K antagonist (warfarin) was introduced with the achievement of therapeutic values of the International Normalized Ratio (INR).

Conclusion

Stratification of patients with suspected / confirmed PTE into groups according to the risk of early mortality determines the clinician to select an appropriate set of diagnostic and therapeutic procedures. In the high-risk PTE it is necessary to conduct reperfusion therapy after a quick diagnosis, most often using a systemic fibrinolytic.

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Sažetak

Dijagnostičko-terapijski pristup pacijentu sa visokorizičnom tromboembolijom pluća - prikaz slučaja i komentar u kontekstu ESC preporuka

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Tromboembolija pluća (TEP) je često i potencijalno fatalno oboljenje za čiju dijagnostiku i lečenje su 2019 publikovane ESC smernice. U njima se naglašava značaj stratifikacije rizika od rane smrtnosti kod suspektne ili dokazane TEP i lečenje shodno klasi rizika: primena antikoagulantne ili fibrinolitičke terapije. Cilja rada je prikaz bolesnika sa tipičnim elektrokardiografskim i ehokardiografskim nalazima stratifikovanog u grupu visokorizične TEP koji je lečen sistemskom primenom fibrinolitičke terapije. Bolesnik starosti 70 godina primljen je u Jedinicu intenzivne nege Internog odeljenja ZC Zaječar hipotenzivan, dispnoičan, sa znacima hipoperfuzije organa. Elektrokardiografski se verifikuje S1Q3T3 znak uz blok desne grane Hisovog snopa i postavlja sumnja na visokorizičnu TEP. Urađenim ehokardiografskim pregledom uz krevet bolesnika registruje se velika trombna masa koja prolabira iz desne pretkomore u desnu komoru. Ordinira se sistemska fibrinolitička terapija alteplasa-om po 2h protokolu uz nefrakcionirani heparin. Dolazi do hemodinamske stabilizacije bolesnika, gubitka elektrokardiografskih promena sugestivnih za TEP i rezolucije tromba u desnim srčanim šupljinama. Bolesnik se otpušta kući nakon 12 dana hospitalnog lečenja. Rana stratifikacija bolesnika sa TEP po riziku od rane smrtnosti i izrada lokalnog protokola za dijagnostiku i lečenje bolesnika shodno klasi rizika povećava uspešnost u prepoznavanju i lečenju ovog oboljenja i smanjuje rizik od smrti i drugih neželjenih kliničkih događaja kod bolesnika.

Ključne reči: *visokorizična plućna embolija, TEP, sistemska fibrinolitička terapija, rTPA, UFH, hemodinamska nestabilnost, PESI*