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MASSIVE PULMONARY EMBOLISM IN OLDER PATIENT: SURVIVAL DESPITE STATISTIC DATA

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Massive pulmonary thromboembolism is presented in this article on example of clinical case. Clinical investigation, prognosis evaluation tools, diagnosis and acute phase treatment along with prevention of recurrent episode of pulmonary embolism presented. Observed and described clinical case of pulmonary embolism in older patient, when patient was mistakenly diagnosed in emergency department as acute coronary syndrome patient.

KEY WORDS: pulmonary thromboembolism, massive, older age, treatment, disease prevention

КЛІНІЧНИЙ ВИПАДОК МАСОВАНОЇ ТЕЛА У ПАЦІЄНТА ПОХИЛОГО ВІКУ: ВИЖИТИ ВСУПЕРЕЧ СТАТИСТИЦІ

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Масивна тромбоемболія легеневої артерії представлена в цій статті на прикладі клінічного випадку. Представлені методи встановлення діагнозу, інструменти для оцінки прогнозу, діагностика та лікування гострої фази поряд з профілактикою рецидивуючих епізодів захворювання. У статті представлений клінічний випадок легеневої емболії у пацієнта похилого віку, коли у відділенні невідкладної допомоги пацієнту був помилково поставлений діагноз гострий коронарний синдром хворого.

КЛЮЧОВІ СЛОВА: тромбоемболізм легеневої артерії, масивний, похилий вік, лікування, профілактика захворювання

КЛИНИЧЕСКИЙ СЛУЧАЙ МАССИВНОЙ ТЭЛА У ПОЖИЛОГО ПАЦИЕНТА: ВЫЖИТЬ ВОПРЕКИ СТАТИСТИКЕ

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Массивная легочная тромбоэмболия представлена в этой статье на примере клинического случая. Описаны постановка диагноза, инструменты оценки прогноза заболевания, диагностика и лечение острой фазы, а также методы профилактики повторного эпизода заболевания. Описан клинический случай тромбоэмболии легочной артерии у пациента пожилого возраста, когда в отделении неотложной помощи был ошибочно диагностирован острый коронарный синдром.

КЛЮЧЕВЫЕ СЛОВА: тромбоэмболизм легочной артерии, массивный, пожилой возраст, лечение, профилактика заболевания

INTRODUCTION

Pulmonary thromboembolism (PE) is an acute blockage of the trunk or branches of the arterial system of the lungs with a formed in the veins of the circulatory system or in the right side of the heart thrombus [1]. In 95 % of cases, PE is a consequence of deep vein thrombosis (DVT), therefore, in modern literature the term «pulmonary embolism» is often replaced by the term «venous thromboembolism» [1–2]. PE is the third most common type of pathology of the

cardiovascular system after ischemic heart disease and stroke. Long-term complication of PE reported in medical literature is chronic thromboembolic pulmonary hypertension with incidence of 0.1–9.1 % within the first two years after a symptomatic PE event [2].

CLINICAL CASE

A 73-year old man was admitted by ambulance in the emergency department (ED) of 25 Kharkiv city clinical multidisciplinary hospital with complains on sudden severe dyspnea in the slightest physical exertion, periodical burning pain in the heart area without clear connection with physical exertion ~ 15 min duration.

ANAMNESIS MORBI

All complains started a day ago, in anamnesis morbi remarkable were myocardial infarction in 2009, transition ischemic attack in January 2016, CAD history and arterial hypertension for many years (bisoprolol and aspirin were taken from time to time).

ANAMNESIS VITAE

Childhood infections, injuries, tuberculosis, sexually transmitted diseases were denied by patient. Hereditary diseases are not identified. Allergic history is not burdened. Smoking – denied, do not abuse alcohol.

OBJECTIVE EXAMINATION

Conciseness – clear, state – severe, body position – active. Patient was orientated in place, time, his personality. Pale skin and mucosae, cyanosis. Thyroid: no pathological changes. Musculoskeletal system – no pathological changes. Breath rate (BR) – 22–24 /min. Lung percussion: no clinically significant changes. Lung auscultation: hard breathing. Borders of the heart: left border – outside of midclavicular left line on 2 cm, others – within normal parameters. Heart auscultation: rhythmic, heart tones – muffled. Pulse – rhythmic, 120 bts/min. Blood pressure (BP) 110/90 mm Hg. Abdomen: normal size, symmetric, unpainful. Liver: liver margin is 5 cm below right rib cage, solid, no pain during palpation in right hypochondria. Spleen: normal. Pasternatsky symptom – negative from both sides. Edemas: right leg was edematous below knee joint comparing with left low extremities, leg slightly painful in edematous area during palpation.

In ED, preliminary diagnosis of unstable angina with community-acquired pneumonia was done because of significant leukocytosis ($19{,}5*10^9$/l) with left-side shift presented in complete blood count (CBC), symptoms and anamnesis morbi data of the patient, who hasn't took medication as it was needed. BR was 20–22 in min, heart rate (HR) around 120 bts in min, BP 110/90, low extremities – not very remarkable changes in left calf area. But clinical probability of pulmonary embolism according to American Academy of Family Physicians (AAFP) (score 13) [3] and the American College of Physicians (ACP) Scores (more than 6) [4] defined possibility of PE in this patient case as high probability (likely). It was possible because of: patient HR was > 95 in min, presence of unilateral lower limb pain with unilateral edema of left low extremity and patient's age was bigger than 65.

LABORATORY AND INSTRUMENTAL TESTS

CBC from 02-sep-2016: leukocytosis (white blood cells (WBC) – $19{,}5*10^9$/l) with left-side shift (bands – 6 %, segments – 76 %) and elevation of Erythrocytes sedimentation rate (ESR) – 37 mm/h.

CBC from 03-sep-2016: leukocytosis (white blood cells (WBC) – $13{,}8*10^9$/l) with left-side shift (bands – some, segments – 85 %) and ESR – 15 mm/h.

CBC from 07-sep-2016: no clinically significant changes except ESR level – 23 mm/h.

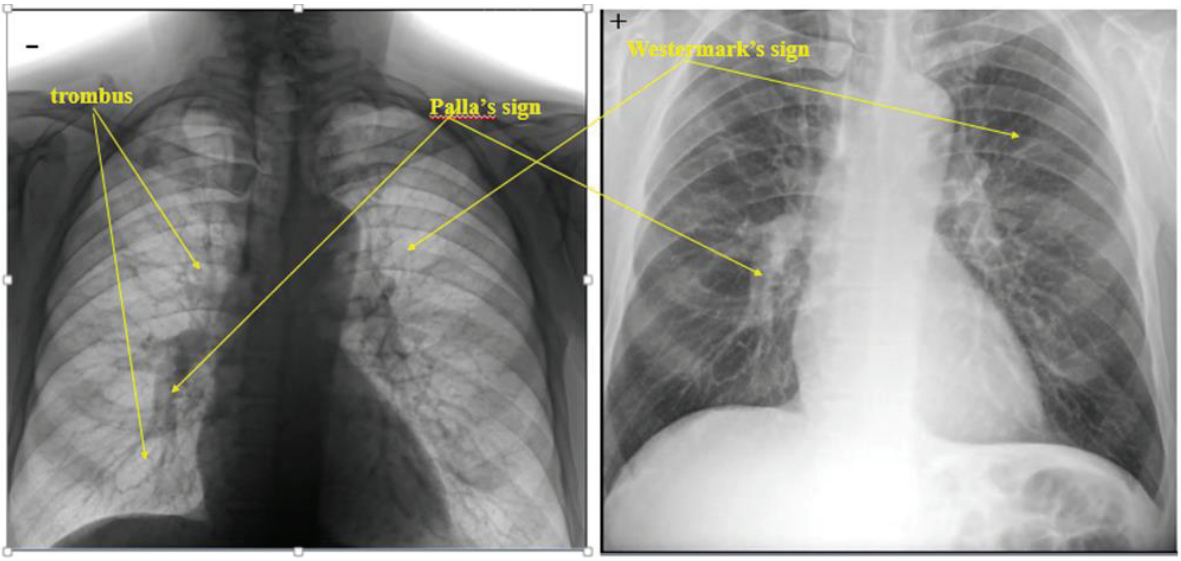
Urinalysis: no clinically significant changes except proteinuria – 0.216 g/l were found.

In biochemistry data significant were: hyperglycemia (fasting glucose levels 8,9 – 8,2 mmol/l), decreased prothrombin time levels (83 – 73,2 %) on the background of medication prescribed (enoxaparin natrium), normal level of Troponin I.

ECG showed classical ECG changes in PE patients as pathological S wave in lead I, Q wave with T-wave inversion in lead III (McGinn-White sign), QR pattern in V1 lead and new right bundle branch block, heart rate – 100 bts/min.

Despite that chest radiograph cannot exclude or confirm diagnosis of PE, but this investigation is useful in further investigations guideless and exclusion or definition of alternative diagnoses. In our patient Chest X-ray data was seen specific for PE Palla's sign

(enlarged right descending pulmonary artery. Diaphragm's cupulas are flattened [5]). Sinuses are poorly differentiated. Westermark sign was seen too (a focus of oligemia (leading to collapse of vessel [5]) seen distal to a pulmonary embolism). Conclusion was: 2-sided pulmonary thromboembolism. But X-ray specialist didn't recognized from the first examination these signs so diagnosis of PE in this patient case wasn't established immediately in emergency department (see pic.1).



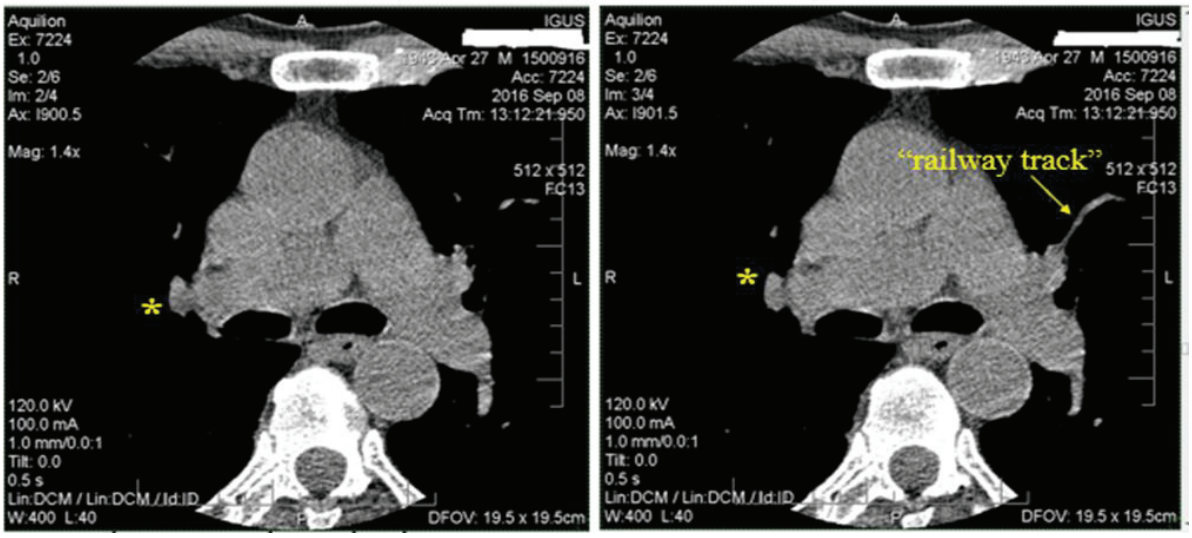
Pic.1 Chest X-ray data

Echocardiography of this patient: EF (ejection fraction) – 60 %. Normal wall movement, myocardium structure with pointed cardiosclerosis changes. Contractility function – not changed. Left Ventricle: FDD – 48 mm (N – 25–35 mm) – enlarged, FSD – 39 mm (N – 23–38 mm), posterior wall thickness – 13 mm (N – 6–13mm). Intraventricular septum size – 13 mm (6–11 mm) – enlarged. Right Ventricle: diameter – 36 mm (N – 9–20 mm) – enlarged, wall thickness – 5,0 mm (N – 2–4 mm) – enlarged, left atrium – not enlarged – 36 mm in diameter (N – till 38 mm), right atrium – enlarged – 50 mm in diameter, interatrial septum – not changed. Valvular apparatus is not changed, except tricuspid valve – regurgitation I degree. In the cavity of the right ventricle clearly seen hyperechogenic formations – clots. Conclusion: Diffuse cardiosclerosis. Aortic atherosclerosis. Hypertrophy of the left ventricle I degree. Dilation of the right heart chambers. Tricuspid regurgitation 1-st degree. Clots in the cavity of the right ventricle. Ultrasound signs of cystitis, chronic prostatitis.

According to the American College of Radiology 2011 guidelines, only multisite CT pulmonary angiography was considered as the gold standard for the detection of PE. [6–7]. For patient with high clinical probability of likely PE, as was seen in our clinical case, multi-detector CT angiography has become an established imaging technique according to the ECS guidelines of acute pulmonary embolism diagnosis and management [1] and the best investigation recommended to prove diagnosis of PE, comparing with not required D-dimer investigation. Guidelines suggest for patients at high risk to skip the D-dimer test and immediately refer patient to CT pulmonary angiography, because even negative D-dimer test result couldn't allow making diagnosis without imaging technics [8]. So plasma D-dimer tests are more useful and effective for patients with intermediate risk of a PE, but also may be not necessary for patients at low and high risk [8].

CT pulmonary angiography findings were: in the main branches of the pulmonary artery clearly seen defects of contrasting thicknesses up to 15 mm on the right and 11 mm on the left, which is spread on all lobular and segmental branches of the pulmonary artery with subtotal or partial occlusion of the lumen (a partial filling defect surrounded by contrast material, producing the «railway track» sign on longitudinal images of the vessel). In both lungs

are visualized subpleural areas of lung parenchyma lightening by the type of «frosted glass». The diameter of the pulmonary artery on both sides is increased (26 mm – pulmonary truncus, 27mm –right pulmonary artery). In the right atrium are visualized defects of contrasting with dimensions of 35×22 mm (thrombus). Conclusion: CT picture of bilateral massive pulmonary embolism (see pic.2)



Pic.2 CT pulmonary angiography

Since the majority of cases PE originates from deep vein thrombosis (VTE), for evaluation of this patient diagnosis and finding of the source of suspected thromboembolism could be useful to refer patient on compression ultrasound of the lower extremities deep veins because patient during objective examination had not very prominent signs and symptoms of deep veins thrombosis as edematous right leg below knee joint and pain in edematous area during palpation.

FINAL DIAGNOSIS

Acute massive pulmonary 2-sided embolism, stable. CAD: stable angina III functional class, post infarction (2009) and diffuse cardiosclerosis. Arterial hypertension III stage, 1st degree, very high risk. CHF 2 A stage with preserved function of LV (EF 59 %), IV D functional class by NYHA. Varicose vein disease of low extremities, right leg phlebitis.

TREATMENT RECEIVED IN HOSPITAL

Zofenopril 7,5 mg 1 time\day at night, nebivolol 2,5 mg 1 time\day morning, warfarin 2,5 mg 1 time\day from 13.09, ceftriaxone 1,0 g 2 times\day IM from 02.09 till 07.09 (preliminary diagnosis was Community-acquired pneumonia), clexan (enoxaparin natrium) 0,4ml (40mg) 2 times a day subcutaneous from 02.09 (preliminary diagnosis in ED was Unstable angina), ivabradin 7,5 mg 2 times\day from 02.09, atorvastatin 40 ml 1 time\day at night from 02.09.16.

RECOMMENDATIONS

According to the American College of Physicians newest guidelines for the evaluation of patients with suspected acute PE (2015), the following recommendations may be applicable for our patient after hospital discharge treatment and prevention of further episodes of PE [8]:

1. Clinical improvement of the patient with PE depends on several main key factors as: at least 3 months duration of anticoagulant treatment received after discharge from hospital, in case of withdrawal of anticoagulant treatment, if anticoagulants are stopped after 6 or 12 months, the risk of recurrence can be expected to be similar to that after 3 months and indefinite treatment reduces the risk for recurrent venous thromboembolism by about 90 % [9–10].
2. In identifying of patients with higher long-term relative risk of PE recurrence useful will be to pay attention at the main risk factors as one or more previous episodes of VTE,

presence of antiphospholipid antibody syndrome or hereditary thrombophilia or residual thrombosis in the proximal veins. Also as additional risk factor was reported the persistence of right ventricular dysfunction at hospital discharge confirmed by echocardiography [1, 8]

In 2016, in the updated American College of Chest Physicians (ACCP) guidelines were recommended prescription for patients with PE of direct factor Xa inhibitors (dabigatran, rivaroxaban etc.) because they are preferable over vitamin K antagonist therapy as first 3 months after PE episode for no cancer patients. But in case of inability for patient to receive direct factor Xa inhibitors or vitamin K antagonist, aspirin is recommended over no aspirin to prevent recurrent PE in patients who are stopping anticoagulant therapy after hospital discharge and do not have a contraindication to aspirin [11], which is more applicable for our patient due to his low adherence to therapy and coast of Xa factor inhibitors in Ukraine for long-term therapy.

CONCLUSIONS

Not every case in medical practice are clearly understandable from the first view, but in the case of diagnostic difficulties, attention should be paid to the possible presence of the main risk factors for thromboembolic complications, the auscultators pattern in the lungs, and the possibility of developing PE (usage of widely unknown prognostic scales makes the task of physician easier and diagnosis evaluation more clear) in each clinical case.

REFERENCES

1. 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism, updated / [S. V. Konstantinides, A. Torbicki, G. Agnelli, et al.] // Eur Heart J. – 2015. – № 36 (39). – P. 2642.
2. Risk factors and basic mechanisms of chronic thromboembolic pulmonary hypertension: a current understanding / I. M. Lang, R. Pesavento, D. Bonderman, J. X. Yuan // Eur Respir J. – 2013. – № 41 (2). – P. 462–468.
3. Wilbur J. Diagnosis of Deep Venous Thrombosis and Pulmonary Embolism Am Fam Physician / J. Wilbur, B. Shian // Am Fam Physician. – 2012. – № 86 (10). – P. 913–919.
4. Current diagnosis of venous thromboembolism in primary care: a clinical practice guideline from the American Academy of Family Physicians and the American College of Physicians / [A. Qaseem, V. Snow, P. Barry, E. R. Hornbake, et al.] // Ann Fam Med. – 2007. – № 5 (1). – P.57–62.
5. Jorge A. Brenes-Salazar Westermark's and Palla's signs in acute and chronic pulmonary embolism: Still valid in the current computed tomography era / A. Jorge // J Emerg Trauma Shock. – 2014. – № 7 (1). – P. 57–58.
6. ACR Appropriateness Criteria® acute chest pain--suspected pulmonary embolism / [M. A. Bettmann, S. G. Baginski, R. D. White, P. K. Woodard, et al.] // J Thorac Imaging. – 2012. – № 27 (2). – P.28–31.
7. Cummings K. W .Multidetector computed tomographic pulmonary angiography: beyond acute pulmonary embolism / K. W. Cummings, S. Bhalla // Radiol Clin North Am. – 2010. – № 8 (1). – P. 51–65.
8. Evaluation of Patients with Suspected Acute Pulmonary Embolism: Best Practice Advice from the Clinical Guidelines Committee of the American College of Physicians / [A. S. Raja, J. O. Greenberg, A. Qaseem, et al]. // Ann Intern Med. – 2015. – № 163 (9). – P.701–11.
9. Aspirin for preventing the recurrence of venous thromboembolism / [C. Becattini, G. Agnelli, A. chenone, et al.] // N Engl J Med. – 2012. – № 66 (21). – P. 1959–1967.
10. Association of persistent right ventricular dysfunction at hospital discharge after acute pulmonary embolism with recurrent thromboembolic events / [S. Grifoni, S. Vanni, S. Magazzini, et al.] // Arch Intern Med. – 2006. – № 66 (19). – P. 2151–2156.
11. Antithrombotic Therapy for VTE Disease: CHEST Guideline and Expert Panel Report / [C. Kearon, E. A. Akl, J. Ornelas, et al.] // Chest. – 2016. – № 149 (2). – P.315–52.