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UNCOMMON CASES OF VENOUS THROMBOEMBOLISM

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ABSTRACT

There is a growing incidence of new cases of venous thromboembolismworldwide.Some of these patients do have a predisposition to the development of thrombosis, some of them do however have various risk factors associated with this event, but few of them have unrecognized etiology. This paper aimsto describe different scenarios of venous thromboembolism.

Keywords: venous thromboembolism, etiology,treatment

INTRODUCTION

Venous thromboembolism (deep venous thrombosis or pulmonary thromboembolism) is one of the highest causes of cardiovascular mortality globally because of underdiagnosed diagnosis in clinical practice. Daily, physiciansfacechallenges in the treatment of patients with acute pulmonary embolis. These patients often belong to a very complex heterogeneous population. The improvements in diagnostic methods and the revolution of anticoagulation therapy lead to a decrease of in-hospital mortality, especially in high risk patients.Despite these facts, the pulmonary embolism mortality rate stillremains high. We became familiar with theEuropean renewed Pulmonary embolism Guidelines dealing with diagnosis, risk stratification, treatment and prognosis of PE1. The European consensus forpatients with deep vein thrombosis elucidates the problem of deep venous thrombosis in cardiovascular practice.2

A triad of risk factors that influence thrombosis, includingblood flow stasis, vascular damage, and hypercoagulability was described by Virchow. The occurrence of VTE isbased on acquired and inherited causes of hypercoagulability, however there are scientific data on the association between atherosclerosis and thrombosis.3

Even clinical signs and symptoms of VTE are well known, as there are many uncommon scenarios for this condition. This paper includes fifteen case reports of venous thromboembolism.

CASE REPORT

A 65 year old male patient with ESRD on HD fCase 1. Polycystic ovary syndrome (PCOS) is associated with the elevation of plasminogen activator inhibitor (PAI)-1, in carriers of PAI-1 4G/5G polymorphism, which is a potent inhibitor of fibrinolysis, and may be responsible for the increased predisposition to deep vein thrombosis (DVT). Combined oral contraceptives (COCs) are the most commonly used therapy of choice for menstrual dysfunction in women with PCOS. We present a case of 23year-old female with a history of menstrual dysfunction and PCOS treated with combined oral contraceptives (drospirenone and ethinyl estradiol) for 3 months. She was referred to us from the gynaecologist. The patient was complaining about aswollen and hot left leg below the knee over the course of 3 days, and she hadelevated levels of D dimers (5628ng/ml). Doppler ultrasound showed occlusive thrombus in left common popliteal vein. Electrocardiography and echocardiography were normal without signs of pulmonary embolism. We started immediately with DOAC by protocol. Knowing the corelation between COCs, thrombotic events and thrombophilias, we performed a genetic investigation that showed PAI-1 4G/5G polymorphism.It is known that PAI-1 is a key negative regulator of the fibrinolytic system. The PAI-1 gene 4G/5G polymorphism may modulate the inhibitor's synthesis and thus influence PAI-1 expression and thrombotic risk. After 5 months, a doppler ultrasound showedcomplete resolution of the thrombus. The patient was advised to continue with DOAC as long as the COCs lasted, due to the risk of recurrence.

The 4G/5G PAI polymorphisms are present with a higher frequency in women with PCOS. The use of COCs in these patients are associated with higher risk for DVT. DOAC therapy should be continued as long as the need for use of COCs remains.

Case 2.Factor V Leiden thrombophilia is inherited in an autosomal dominant manner. Homozygosity for the Leiden variant (and a much greater risk for venous thrombosis) is inherited in an autosomal recessive manner.A55year-old male was admitted to the hospital with heart decompensation and symptoms of dyspnea, cyanosis, fast heart rate, tibial edema, and ascites. The patient had a15 yearmedical history of deep venous embolism and pulmonary thromboembolism. Clinical examinations, including an ECG, showed atrial fibrillation with a fast heart rate of 140 bpm and a right bundle branch block. RTG showed an increased cardiothoracic index and pleural effusion. Laboratory testing with increased NT-proBNP 3600pg/ml. Echocardiography showed an enlarged right heart chamber, severe tricuspid regurgitation, dilatated pulmonary artery 38mm, moderate pulmonary regurgitation with severe pulmonary hypertension, HFmrEF, and circumferent pericardial effusion 9mm. CT chest angiography showed a filling defect in the lower right pulmonary artery. After recompensing, the patient was sent for genetic testing for thrombophilia and a lung ventilation-perfusion scan. Genetic testing showed homozygotes for Factor V Leiden (V G1691A), heterozygote MTHFR C677T. A V/Q scan showed a mismatched V/Q Ratio (>0.8). He was anticoagulated with Rivaroxaban 2x15mg for 21 days, and then 20mg indefinitely. He was forwarded to the referent center for pulmonary hypertension.

The outcomes in most patients with FVL are good, but the outcomeswiththis patient and his disorder are serious because of the morbidity associated with VTE development and VTE-associated complications. To decrease the morbidity and mortality associated with VTE, anticoagulation is recommended.

Case 3. Incidenceof cancer associated venous thromboembolism is well known and more frequent than major bleeding in such patients. Sometimes the diagnosis is delayed and start up of treatment is postponed and this could lead toan inferior quality of life or even early mortality. Some specific cancer types are more thrombogenic than others- for example cancer of the pancreas is several timesmore thrombogenic than melanoma. Vena cava superior syndrome (VCS) is sometimes the first and the only manifestation of cancer associated venous thromboembolism. In some cases, the syndrome is not well known and mistaken for an allergenic reaction to treatment. When recognized, VCS shouldbe treated rapidly. Interventional treatments such as PTA and dedicated stent implantationaresome of the options withsuch patients. Sometimes PTA of VCS could be the bridge to furtherconsequent oncology treatment. We represent a case of 46year-oldwoman with a 6 year history of breast cancer, who under-

went haemo- and radio treatment. She was admitted first to the clinic of allergology for oedema of the face and arms. After clinical examination and CT angiography, VCS was diagnosed. Successful PTA with no intraprocedural complications was performed and the patent was referred for further oncology treatment.

Interventional treatment of VCS is an option in elevated risk patients with low rate of complications. Early diagnosis is crucial for successful treatment.

Case 4.Pylephlebitis is a rare abdominal variant of Lemierre's syndrome with extensive portal vein, splenic vein, and mesenteric vein thrombosis. Lemierre's syndrome is a rare but potentially severe thrombotic complication of bacterial infections that usually affects previously healthy adults. Here, we present a case of 68 y.o.woman with pylephlebitis. She was accepted for hospital treatment due to severe pain in the upper abdomen. There was a 7cm tumefaction under the left scapula. Upon ultrasound evaluation, we discovered thrombosis of the portal vein, enlarged spline as well as hematoma below the spline. CT scan evaluation confirmed severe portal and splenic vein thrombosis, hematoma under the spline, which makes a protrusion through left hemidiaphragm under the skin up to the 8th - 10th ribs. Gastroscopy was in context of severe esophageal candidiasis and hemoculture and was positive for peptostreptococcus. A cardio thrombophilia test expressed deterioration in folate metabolism. From anamnestic data we recognized that this patient had been treated with corticosteroids for a long time because of severe spondylitis pain. The patient demonstrated a complete recovery following appropriate long-term intravenous antibiotics and anticoagulation. This case calls attention to the re-emergence of the rare manifestation of this forgotten disease and highlights improved outcomes with prompt recognition and early treatment.

Case 5. Thromboembolic complications during coronavirus COVID-19 infection have been described in multiple case reports and studies after and during the pandemic, and despite the growing understanding of these complications, management and prevention remain individual, pending available recommendations. In this case report, we present the successful management of a patient with high-risk pulmonary embolism (PE). This PE is a complex thromboembolic event in a COVID-19 patient.A patient with COVID-19 developed sudden shortness of breath and hypoxia. Early echocardiography showed enlarged right heart chambers with left ventricular D-shape in parasternal short-axis view. Thrombi were registered in the apex of the right heart chamber. Intravenous crystalloid fluid and unfractionated heparin (UFH) infusion was started with regular aPTT measurements and the patient was closely monitored for hemodynamic instability. Chest computed angiography with contrast was performed in the first hour,confirmingthe diagnosis of a massive PE. UFH was transitioned after 48 hours to oral anticoagulant therapy (Apixaban dose of 10mg b.i.d) with plans to continue anticoagulation long-term having in mind the history of DVT in patients with sub-sequent re-evaluation.

Pulmonary thromboembolism is a commonly reported occurrence in severe COVID-19 infection. In this case report, a fast switch to NOAC and long-term treatment proved to be effective, and it may be considered for high-risk PE in COVID-19 patients.

Case 6. Themorbidity and mortality from pulmonary embolism (PE) are effectively reduced with the early diagnosis and administration of proper anticoagulant therapy. Fibrinolytic therapy is used in clinically unstable patients with massive PE to rapidly dissolve the embolic burden and achieve hemodynamic stability. Careful patient selection is crucial for successful fibrinolysis, all in order to avoid adverse effects, such as major and minor hemorrhage.

A 58-year-old patient was admitted to the Intensive Care Unit in a state of cardiogenic shock, with severe dyspnea and hypotension. PE was suspected, due to a previously performed transthoracic echocardiography that revealed dilated right heart cavities, D-shaped left ventricle, TAPSE 15. From his medical history, we noticed that he had an ischemic stroke 2 months prior, followed by right hemiparesis, treated at the neurology clinic with thrombolytic therapy. Also, he had a thrombophlebitis of the right saphenous magna vein 5 years prior. This was treated with direct oral anticoagulant (DOAC) therapy. On admission he was afebrile, with blood pressure (BP) 80/60mmHg and oxygen saturation (SpO2) 88%. Electrocardiography (ECG) showed a sinus rhythm with a heart rate (HR) of 120 beats/minute and the presence of a S1Q3T3 pattern. Urgent computed tomography pulmonary angiography

(CTPA) confirmed a right pulmonary artery filling defect and atelectasis of the right lung, with bilateral consolidation and unilateral pleural effusion. Laboratory analysis showed elevated values of Troponin 131ng/L, NT-proBNP 5956pg/ml, CRP 289mg/l. The assessed sPESI score was >1, classifying the patient into the high-risk PE group. A central venous catheter was placed and fibrinolytic therapy with Actilyse per protocol was applied. Additionally, dual parenteral antibiotic therapy was given. Hemodynamic stabilization occurred after a few hours, with BP 110/75mmHg, HR 80bpm, SpO2 93%.Bleeding complications were not observed. Venous doppler ultrasound of the lower extremities revealed right femoral and popliteal vein thrombosis with thrombophlebitis of the right saphenous magna vein. After eight days of hospitalization, the patient was discharged in a stable condition with the recommendation for DOAC therapy beyond 3 months.

Patients with a previous ischemic stroke may be at higher risk for venous thromboembolism, due to limb paralysis, prolonged bed rest and increased pro-thrombotic state. Treatment decisions must take into consideration the risk/benefit balance, particularly in patients with relative contraindications forfibrinolysis.

Case 7. Acute dyspnea in patients with known chronic obstructive pulmonary disease (COPD) can be a diagnostic challenge due to the nonspecific nature of atypical presentations. It is suspected that one in four patients with atypical COPD exacerbation may have PE as an underlying or concomitant cause of acute dyspnea. Many cardiopulmonary diseases, including acute pulmonary embolism (PE), are worsenedor masked by the presence of COPD.There are too many risk factors that have been described for PE. However, hereditary factors are important risk factors for the cases especially with recurrent PE.A 48-year-old female patient, non-smoker, morbidly obese (BMI: 38 kg/m2), co-morbidity COPD, presented with increasing dyspnea over 3 weeks and now at rest, chest pain, on acenocumarol therapy. No cough or sputum purulence. She had a history of unprovoked intermediate-risk PE 12 months prior. Respiratory rate 30/min, blood pressure: 115/75 mmHg, heart rate: 110/min; sO2 86% on room air, breath sounds diminished on chest examination, no murmurs on heart auscultation.Laboratory findings: Haemoglobin (Hb) – 122 g/L, total leukocyte count (TLC) 12x10^9/L, PLT 398 x10^9/L, D-Dimers 4,500 mg/L, HS Troponin I 50ng/L. Blood gas analysis: pH: 7.4; PaCO2, 33 mm Hg; PaO2, 53 mm Hg; lactate normal, INR =1.7. Electrocardiogram (ECG) – sinus tachycardia with right axis deviation and right ventricular (RV) strain pattern. 2-D Echocardiography revealed dilated RV – 33mm with right heart pressure overload with systolic pulmonary artery pressure (sPAP) - 80mmHG, with negative 60/60 sign and negative McConnel's sign. Computed tomography pulmonary angiography (CTPA) revealed thromboemboli in 2 segmental arteries involvement but without total lumen obstruction. The patient was switched toNovel oral anticoagulant therapy and was further evaluated. The patient has improved gradually over a period of 1 week with a normal pulse and respiratory rate, maintaining oxygen saturation on room air. ECG improved with normal sinus rhythm. The patient was evaluated at a higher centre for genetic factors and mutations and thefollowing coagulation factors were detected: factor V Leiden mutation, factor VIII mutation, and a prothrombin gene mutation.

Acute dyspnea is a complex symptom. The degree to which there has been pre-existing disease must also be factored into analysis. Therefore, if one encounters pressures of 70mmHg or more on echocardiography in a suspected pulmonary embolus, the scenario of acute on chronic thromboembolic disease or pulmonary embolus superimposed on previously existing pulmonary hypertension must be considered. CTPA is the modality of choice for the diagnosis of acute PE, given its availability as well as excellent sensitivity and specificity. Identification of the genetic risk factors that result in increased tendency to thrombosis has important implications for the patients and their families.A patient with pulmonary emboli of another major illness often have atypical presentations or may not be acutely symptomatic. Future work is directed at understanding the disease pathogenesis through further examinations for Chronic thromboembolic pulmonary hypertension(CTEPH) probability. One must also have in mind that numerous risk factors have been associated with the development of CTEPH, including but not limited to, thrombophilia and chronic inflammatory states.

Case 8. Pregnancyis a well-known cause for hypercoagulability conditions, sometimes resulting in venous thromboembolism in women. It can be located in the deep veins of the lower

extremities, but we have seen many cases complicated by pulmonary embolism as a life threatening condition in young women.

We usedechocardiography as a first diagnostic tool, not only to exclude right heart dysfunction, but also to look for all the structural and functional changes in the heart which can change the pathway totreating patients in pregnancy affected by VTE.

At first glance, the patient was a not so rare case of a pregnant womanin her first trimester. She presented with deep vein thrombosis, affecting the popliteal vein of the left lower extremity. No other symptoms other than swollen extremities were reported. Echocardiography and electrocardiography were normal, and they excluded possible pulmonary embolism as a complication. However, an incidental finding of an aortic aneurysm of the Sinus Valsalva (45mm) in our 33 year-oldpatient was diagnosed. This then changed the strategy, leading to a further follow up. The patient was successfully treated with LMWH, and we dischargedher in an improved health condition after 7 days of hospitalization. We requested a close follow up for the aortic aneurysm at every 1-3 months during pregnancy, with a caesarean section being considered when planning delivery.

Other incidental findings of structural heart changes can change the whole follow up and treatment strategy in pregnant patients with venous thromboembolism. That is why echocardiography plays a key role in pregnant patients affected by any cardiovascular pathology.

Case 9. Antiphospholipidsyndrome (APS) is a condition in which the body produces antiphospholipid antibodies, resulting in arterial and venous thrombosis, most commonly impacting the lower limbs and cerebral arterial circulation. Catastrophic antiphospholipid syndrome (CAPS) is a rare APS subtype characterized by acute thrombotic microangiography that can result in multi-organ failure in a short period of time. Antiphospholipid antibodies cause thrombosis through activating and inhibiting properties. Factors that trigger the production of antiphospholipid antibodies are: genetics, infection, surgery, medications, and malignancy.

A 63 year-old male was transferred to The Special Hospital for Cardiovascular disease in Ohrid with a history of hypertension and a two-day history of worsening shortness of breath and chest tightness. The serum Troponin level was increased from 66 ng/L to 150 ng/L.

Doppler ultrasound showed deep vein thromboses (DVTs) in the right femoral, popliteal, and peroneal veins.EKG with RBBB and complete blood count (CBC) showed a low platelet count of 130x109/L.The echocardiogram showed severe right ventricular (RV) dilatation and reduced RV function. The interventricular septum flattened during diastole.

A computed tomography (CT) with angiogram was ordered and showed sub-massive bilateral pulmonary embolism (PE) from the right and left main pulmonary arteries into the lobar and segmental branches causing the right heart failure.

Rescue thrombolytic therapy with Alteplase was administered. On the fourth day of admission, the platelet count decreased to 90x109/L and there were no signs of hemolysis.The patient was then switched from low-dose heparin to apixaban. Due to the combination of acute thrombosis and thrombocytopenia, APS was suspected. Because the patient was in post thrombolysis status, the drop in platelet count could not be attributed to deep vein thrombosis (DVT) or pulmonary embolism (PE). Tests for antiphospholipid antibodies, anticardiolipin and anti-BGP-1 came back positive. On day five of admission, the patient was switched over to warfarin. The patient was discharged on the seventh day of admission with prednisone and warfarin prescribed. Six weeks later, he was still positive for antiphospholipid antibodies. As a result, this patient was diagnosed with antiphospholipid syndrome.

Considering CAPS is not as frequently seen as typical APS, and yet is also life-threatening in a more elderly male rather than the typical middle-aged female, calls for the need to screen all patients with evidence of intravascular thrombosis for CAPS earlier and reduce mortality from this disease.

Case 10. Venous Thromboembolism (VTE) is a clinical term which includes two closely associated conditions: Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE). There are various risk factors associated with an increase in the incidence of PE. One of them is the intake of hormonal contraceptives, a problem to which doctors sometimes do not pay due attention. We hereby present a case of a woman who developed bilateral PE likely due to combined oral contraceptive pills

use. A 48-year-old female patient was admitted to hospital with complaints of dyspnea and chest discomfort 1 month prior to hospital admission. She had a positive family history for VTE and was a smoker, also declared the use of combined oral contraceptives containing the progestin hormone drospirenone for 6 months. Laboratory tests were taken and showedan elevated D-dimer level. After transthoracic echocardiography, no indirect signs of acute PE were found.Still, the patient was sent for a CT pulmonary angiogram which revealed bilateral acute PE extending into segmental branches with no PAH.Low molecular weight heparin – Enoxaparin at a weight-adjusted dose of 1 mg/kg was administered intravenous for 3 days and the patient was shifted to an oral anticoagulant – Rivaroxaban at a dose of 15 mg twice daily. Bilateral lower limb Doppler was also done which ruled out any DVT. Additionally, thrombophilia genetic tests were completedand showed heterozygous mutation of the methylenetetrahydrofolate reductase (MTHFR) C677T gene, heterozygous mutation of the methionine synthase reductase (MTRR) A66G gene, heterozygous mutation of the β-fibrinogen 455 G>A gene and PAI-4G/4G. After 8 days, the patient was discharged in an improved clinical state on oral anticoagulant rivaroxaban 15 mg twice daily for 16 more days, then 20 mg once daily.

Before combined oral contraceptives are prescribed, careful evaluation of side effects, including VTE, as well as risk factors for VTE, such as cigarette smoking and a positive family history for VTE is recommended. Thrombophilia should be screened in young females under the age of 50 even with the presence of a transient risk factor.

Case 11. Mixed germ cell testicular tumor is a form of malignant tumor that can be treated with surgery. Thrombosis is one of the possible postoperative complications. This case report describes a young male patient who developed popliteal thrombosis in the left leg,fourteen months after surgery for a mixed germ cell testicular tumor (MGCT) and two months after retroperitoneal lymph node dissection surgery (RPNLD).The aim of this report is to describe the clinical presentation, imaging findings and management of popliteal thrombosis following testicular mixed germ cell tumor in a young patient.We present a 26-year-old male patient who underwent surgical removal of a MGCT tumor and RPNLD and subsequently developed popliteal thrombosis, presented with pain and swelling in the left leg. He was hospitalized in the University Clinic of Cardiology in Skopje. The patient underwent echocardiography, Doppler ultrasonography of the lower extremity arteries and laboratory. Echocardiography and laboratory results were normal. TheDoppler ultrasonography of the lower extremity arteries showed an occlusive thrombus in the left popliteal and left superficial femoral vein. The patient was started on a full dose NOAC (Rivaroxaban 15mg BID). Afterthree months, a Doppler ultrasonography of the lower extremity arteries follow up was done. The patient still presented with ongoing thrombosis and NOAC (Rivaroxaban 20mg daily) was further administrated.

Popliteal thrombosis is a serious complication following testicular cancer surgery. Early diagnosis is crucial for the treatment and outcome. Clinicians should have a high index of suspicion for this complication in patients who develop leg pain or swelling after testicular cancer surgery.

Case 12. Venous thromboembolism (VTE) is a common vascular diseaseafter acute myocardial infarction and stroke. The underlying pathogenic mechanisms are only partially known. There are sundry risk factors for VTE, such as hypercoagulability, especially after Covid-19 infection, metabolic syndrome, major surgery, injury, chronic medical conditions, age, and other releated factors. Thus, our intention withthis patient was to identify if there is a genetic predisposition for developing VTE and which of the 12 genes responsible for thrombotic disorders was mutated, or if the disease did not haveany genetic implications.Thepatient, age 42, consulted first about tachycardia and shortness of breath and was treated ambulatory, but after three days, the patient consulted again, but this time with pain and a swollen left leg, suspected asvein thromboembolism and was hospitalized. The patient revealed laboratory high levels of D-dimers (5600 ng/L) and venous Doppler of lower extremities confirmed presence of thrombus in distal third of v. femoralis sin. and along the entire course of v. poplitea sin, and along the entire length of v. tibialis post. sin et v. peronealis sin. After examination, procedures and treatmentoptions, were completed as per protocol, and further genetic examination wasperformed. Genomic DNA was isolated from white cells from peripheral blood sampleusing the real-time PCR method testing the 12 genes. Results from the genetic investigation

showed a mutation in the second allele of the Factor XIII, mutation in the first and the second allele of MTHFR A1298C, first and the second allele of the MTRR A66G and the second allele of the B-fibrinogen. It can be concluded that mutations in the MTHFR gene, MTRR gene, B-fibrinogen gene and factor XIII gene are associated with an increased risk of thrombophilia, thus increasingchances todevelop VTE. Therefore genetic screening for this population is particularly importantin order to prevent future events.

Case 13. Pulmonary thromboembolism (PTE) can be a life-threatening condition without proper diagnosis and treatment. Cancer is a risk factor for thromboembolic disease, and patients with cancer are estimated to be around four times more likely to develop thrombosis than a similar individual without cancer. We present a case of cancer-associated PTE in a patient with prostate cancer.

A 65-year-old patient with dyspnea and sudden chest discomfort for 2 days, was admitted to hospital. From the medical history, the patient had prostate cancer with metastases in the liver and bones, treated with bisphosphonate and hormonal therapy. Initially, blood analyses were made and showed an elevated level of troponin and D-dimer, as well as a transthoracic echocardiography whichrevealed increased dimensions of the right heart cavities, moderate to pronounced TR, significant PAH, pericardium layered behind the back wall 4-5 mm and preserved left ventricle systolic function (EF - 61%). Due to clinical and laboratory findings and the echocardiography report, the patient was suspected of having PE and was immediately hospitalized. During hospitalization, CT pulmonary angiography was performed and central thromboembolic filling defects were monitored at the output level of the lobar branches for the upper, middle and lower lobes on the right including posterobasal segment on the same side as the branches supra and infralingually for posterior lobe to posterobasal segmentwith pleural effusion bilaterally. This confirmed the diagnosis of acute lobar PTE. Bilateral lower limb Dopplersonography was also done, ruling out any DVT. The patient was treated with low molecular weight heparin – Enoxaparin, at a weight-adjusted level of 1 mg/kg for 3 days and then was shifted to NOAK – Rivaroxaban, at a dose of 15 mg twice daily. After 8 days, the patient was discharged in an improved clinical state on Rivaroxaban 15 mg, twice daily, for 13 more days and then 20 mg once daily.PTE is a common and potentially lethal disease in active cancer patients. Therefore, specific strategies for early diagnosis and management of acute PTE in such patients is warranted.

Case 14. Cancer-associated thrombosis (CAT) is the second leading cause of death in cancer patients. The causes of this impaired coagulation are associated with general patient-related risk factors, and others specific to the particular cancer or treatment, contributing to the increased risk of patients developing VTE, when compared with non-cancer patients.In this case report, we described a 67-year-old woman, presented to our emergency department with with fatigue, dyspnea, and pleuritic chest pain. She wascurrently undergoing chemotherapy after surgical treatment of ovarian cancer.Due to assessed pulmonary embolism, likely based on a modified Wells score, a TTE was performed immediately. The TTE showed increased right ventricle size with reduced function, severe tricuspid regurgitation, and signs of pulmonary hypertension. In addition, the diagnosis was confirmed with the CT scan,revealingpulmonary emboli scattered at the bilateral pulmonary arteries. The patient was admitted to the ICU, where treatment with LMWH was started. On the third hospital day, she was started on direct oral anticoagulant therapy with 15 mg of Rivaroxaban, twice daily for 21 days, followed by 20 mg once daily.She was discharged with normalized right ventricle function and regression of the pulmonary hypertension. The patient received a recommendation for 6 months of anticoagulation therapy, as well as cancer activity assessment. Patients at highest risk for venous thromboembolism are those with advanced diseases, receiving systemic chemotherapy and other additional risk factors. Clinical practice is changing in the prevention and management of CAT, and many therapeutic options are becoming available both for prevention and treatment.

Case 15. Pulmonary Thromboembolism (PTE) is one of the most important clinical conditions of Venous Thromboembolism (VTE) and often develops on the background of underlying deep vein thrombosis (DVT). The diagnosis of pulmonary thromboembolism (PTE), with changes shown by electrocardiography (ECG), is a challenge in the clinical practice due to rare pathognomonic findings. Although ECG is a cheap and

rapid diagnostic test for pulmonary embolism (PE), it has some limitations in the differential diagnosis of acute coronary syndrome.

We present a clinical case of 58-year-old woman whowas hospitalized in the University Clinic of Cardiology on account of chest pain,dyspnea, and fatigue. The onset of the symptoms started two days before she was hospitalized. The patient has a historyof arterial hypertension and diabetes mellitus. An electrocardiogram suggested subacuteseptal myocardial infarction (AMI). Transthoracic echocardiography showed elevated pulmonary and right heart pressures and right ventricular hypokinesia Doppler ultrasound exclusivity DVT. Computer tomography pulmonary angiography confirmed the diagnosis of pulmonary embolism.

PTE with ECG changes should be considered in the differential diagnosis of AMI, particularly in young patients with chest pain and ST segment elevation suggestive of acute coronary syndrome.

CONCLUSION

Venous thromboembolism is a serious clinical condition with different clinical facets that need prompt diagnosis and decision for early adequate treatment.

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Резиме

НЕВООБИЧАЕНИ СЛУЧАИ НА ВЕНСКИ ТРОМБЕМБОЛИЗАМ

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Постои растечка инциденца на нови случаи на венскиот тромбемболизам во светот. Некои од овие пациенти навистина имаат предиспозиција за развој на тромбоза, некои од нив имаат различни фактори на ризик поврзани со овој настан, но неколку од нив имаат непрепознатлива етиологија. Овој труд има тенденција да опише различни сценарија на венски тромбемболизам.

Клучни зборови: венски тромбемболизам, етиологија, третман