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When pulmonary embolism is not an obvious diagnosis - pathophysiology, risk factors, diagnosis, treatment and a review of the most unusual cases

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Abstract:

Introduction: This review paper aims to emphasize how pulmonary embolism is a seriously life-threatening disease entity, point out risk factors, symptoms, diagnostic trails, treatment and prove that a diagnosis, specifically aimed at embolism is often not obvious in given clinical cases.

Materials and methods: A review of chosen literature in the PubMed database was conducted, using the following keywords: „ pulmonary embolism”, „vein thrombosis’’, „thrombosis risk factors”, „anticoagulation”.

Summary: Pulmonary embolism is a dangerous disease that can lead to serious complications and death, if not properly diagnosed and treated. Proper diagnosis is crucial, as symptoms can be uncharacteristic and embolism can occur in a variety of configurations. Prompt implementation of treatment, including the use of anticoagulants, as well as appropriate medical care, can significantly improve patients' prognosis.

Conclusions: Lesser-known risk factors for pulmonary embolism include sepsis, urinary retention, hematologic, rheumatic and endocrine diseases, organ transplants, treatment with bone cement or tranexamic acid. It is important to conduct diagnostic testing for pulmonary embolism when characteristic symptoms occur and introduce appropriate therapy as well as thromboprophylaxis depending on the patient's risk and clinical condition.

Key words: pulmonary embolism, vein thrombosis, thrombosis risk factors, anticoagulation

Introduction

Pulmonary embolism (PE) is a medical condition in which one or more of the pulmonary arteries become blocked by a thrombus (clot), leading to restricted blood flow through the lungs. Pulmonary embolism can result from displacement of the thrombus from other parts of the body (most often from the deep veins of the legs, in the case of deep vein thrombosis - DVT), although in rare cases it can be caused by other factors, such as fat, air, or amniotic fluid. [1] This condition disrupts normal physiology and can result in severe hemodynamic instability, particularly when the clot burden is substantial. The primary determinant of pulmonary embolism outcomes is the effect on the right ventricle and its ability to adapt. The right ventricle, a thin-walled chamber, normally operates against a low-pressure, low-resistance pulmonary circulation. Pulmonary vascular resistance is influenced by various factors, including oxygen-sensing mechanisms. In the presence of a pulmonary embolism, right ventricle afterload can increase acutely due to both mechanical obstruction and hypoxic vasoconstriction. [2] The leading cause of mortality in acute pulmonary embolism is right ventricular dysfunction and subsequent failure. [3]

Symptoms

Symptoms of pulmonary embolism can vary depending on the size and location of the embolism, as well as the patient's condition. In some cases, symptoms are mild and difficult to identify, while in others they can be sudden and life-threatening. The most common symptoms include: shortness of breath, chest pain, which can be sharp, burning, or resemble the pain associated with a heart attack, cough, sometimes with expectoration of blood (hemoptysis), dizziness, fainting, accelerated heart rate (tachycardia), low oxygen saturation, swelling of the lower extremities, especially if accompanied by deep vein thrombosis, increased sweating, pain on breathing. [4] In the case of a large embolism, symptoms of heart

failure and shock can occur, which is a life-threatening condition. These symptoms are the result of inadequate oxygenation of the body and can lead to multi-organ failure. Key predictors of short-term mortality in patients with acute pulmonary embolism include overall clinical status, right ventricular dysfunction and myocardial injury, as indicated by elevated troponin levels. [5] It is estimated that pulmonary embolism affects over 600,000 patients annually and is implicated in 50,000 to 200,000 deaths each year. [6] A 2012 study reported an in-hospital mortality rate of 31.8% in hemodynamically unstable patients with pulmonary embolism, compared to 3.4% in those who were hemodynamically stable. [7]

Diagnosis

Diagnosis of pulmonary embolism is difficult because its symptoms may resemble other conditions, such as heart attack, heart failure or pneumonia. The diagnostic process consists of several steps: history and clinical evaluation. The doctor takes a detailed history, noting risk factors, history of thrombosis, recent surgeries, hospitalizations, long-term immobilization and symptoms. So-called risk scales, such as the Wells scale for assessing the likelihood of pulmonary embolism, are also often used. Physical examination and laboratory tests including: D-dimers - determination of the level of D-dimers in the blood, which are products of thrombus breakdown. High levels of D-dimers can suggest the presence of a blood clot, but this test is not specific, as elevated levels can occur in other inflammatory conditions or after surgery. Blood gasometry - can show hypoxemia (low oxygen levels) and hypocapnia (low carbon dioxide levels). Imaging is also necessary: Computed Tomography (CT) of the chest with contrast is the most commonly used test in the diagnosis of pulmonary embolism. It allows visualization of blockages in the pulmonary arteries. Lung scintigraphy - a test that involves the injection of a radioactive substance that visualizes areas of the lungs with inadequate blood supply. Echocardiography - an ultrasound of the heart that can help assess the possible right ventricular burden of congestion. Pulmonary angiography - this is a more invasive diagnostic method, used mainly when other methods have not yielded conclusive results, but is being used less and less due to the availability of CT scans. [4] Risk stratification plays a vital role in assessing pulmonary embolism severity and mortality, making it an essential aspect of hospital-based clinical management. [8,9] The evaluation of suspected pulmonary embolism includes assessing the pre-test probability, categorizing it as low, intermediate, or high, based on the Wells and Geneva scoring systems. [10,11] Accurate and rapid evaluation of pulmonary embolism-specific risk factors, along with the prompt

initiation of appropriate therapy, is critical to minimizing morbidity and mortality associated with the condition. [12,13]

Risk factors

The risk of pulmonary embolism increases for patients who have predisposing factors for thrombosis, especially in the context of deep vein thrombosis. Pulmonary embolism is more common in the elderly, as well as in hospitalized patients, especially after surgery, during periods of prolonged immobilization, or in cases of severe trauma. Among the most common risk factors for pulmonary embolism are: DVT - clots in the veins of the legs or other parts of the body that can break off and travel to the lungs, cancer diseases, especially those with a higher risk of clots (e.g., pancreatic cancer, ovarian cancer), blood clotting disorders (e.g., hereditary hemorrhagic diathesis), use of hormonal contraception or hormone therapy, pregnancy and puerperium, heart rhythm disorders such as atrial fibrillation, obesity, smoking, age. [14]

Treatment

Treatment of pulmonary embolism depends on the severity of the patient's condition and the size of the embolism. Anticoagulant therapy (e.g. heparin, oral anticoagulants) remains the cornerstone of acute pulmonary embolism treatment and should be administered to all suspected cases while awaiting diagnostic confirmation, provided there is no active bleeding. [15] Fibrinolytic (clot-dissolving) therapy may be used in cases of severe pulmonary embolism, especially when the patient is in a life-threatening condition. In very severe cases where the embolism is large, surgical intervention or the use of mechanical devices to remove clots may be necessary. In cases of severe hypoxemia, interventions such as oxygen therapy and, in extreme cases, mechanical respiratory support (mechanical ventilation) may be also necessary. [16]

Unusual cases

Cancer

In our study we pointed out that cancer diseases are one of the main risk factors of pulmonary embolism. A retrospective cohort study including autopsy reports of 9,571 cancer patients

showed that over 12% of them had pulmonary embolism, suggesting that PE is an important complication of the disease in cancer patients. [17]

Hematology issues

Not only cancer patients are at risk, but also those who have hematological disorders. The occurrence of a thromboembolic event is not an unlikely event in a patient with hemophilia, which was proven by a 33-year-old patient with factor V and factor VIII deficiency who developed pulmonary embolism during pregnancy. [18] Hereditary thrombophilia caused by gene mutations is also the cause of thrombosis, consequently, pulmonary embolism. The case of a 56-year-old man with a medical history of a homozygous mutation of the prothrombin gene (G20210A), who experienced unprovoked thrombosis of the right lower limb covering both the proximal and distal veins, resulted in massive pulmonary embolism. [19] PE is a rare complication in patients with AIHA (autoimmune hemolytic anemia), but medicine likes exceptions to the rule; 59-year-old female presented to the emergency department complaining of severe chest pain and shortness of breath, experienced syncope, and soon developed cardiac arrest. After the diagnostics, it turned out that the patient had pulmonary embolism secondary to autoimmune hemolytic disorder. [20]

Electroconvulsive therapy

Occasionally, pulmonary embolism may be induced iatrogenically, for example by electroconvulsive therapy. A few hours after electroconvulsive therapy the patient complained of shortness of breath, then his condition worsened, he developed a painful cough, constant tightness in the chest. An extensive somatic examination was performed - his O2 saturation was 82% and blood tests showed a pathological increase in troponin and D-dimer. Computed tomography angiography (CTA) confirmed bilateral pulmonary embolism of medium or high risk, the source of the clot was not identified, ultrasonography of the lower limbs did not reveal vein thrombosis. Limited data are available on the association between pulmonary embolism and electroconvulsive therapy. Further research on this topic may help develop a stratification and scoring system for patients who are at higher risk and who could benefit from antiplatelet or anticoagulant therapy before initiating therapy. [21]

Transplantology

A case was reported involving a living liver donor who underwent a left liver lobectomy and developed a severe pulmonary embolism on postoperative day 2, despite having no identifiable risk factors for venous thromboembolism or pulmonary embolism. [22] The patient was treated with tissue plasminogen activator and heparin infusions and was discharged one week later. According to the literature, the incidence of venous thromboembolism in living liver donors ranges from 1.8% to 2%, but numerous studies have documented successful liver transplants without any thrombotic complications. [23-25]

Pulmonary embolism may also occur in organ transplant recipients. On the second day following liver transplantation, a 55-year-old patient experienced an acute and severe episode of sudden-onset dyspnea. Computed tomography angiography revealed an elongated embolus extending into both main pulmonary arteries, causing near-total occlusion. [26] Thrombolytic therapy with rtPA was initiated, leading to significant circulatory stabilization within the first 60 minutes of administration. Despite the anticipated high risk of bleeding, rtPA thrombolysis can serve as a life-saving intervention for pulmonary embolism in post-liver transplant patients.

There was also a case of a 35-year-old male kidney transplant recipient who experienced a cerebral paradoxical embolism linked to a spontaneous venous thromboembolism. [27] Paradoxical embolism (PDE) occurs when a thrombus passes through an intracardiac defect and enters the systemic circulation. [28,29] The symptoms vary depending on the location of the embolization, potentially affecting the brain, heart, gastrointestinal tract, or extremities. [28,30] The patient had a history of recurrent deep vein thrombosis, with emboli affecting both the lungs and paradoxically the brain via a patent foramen ovale. Bubble echocardiography played a crucial role in the diagnosis, helping to prevent contrast-induced nephropathy. [27]

Orthopedic surgeries

A 46-year-old woman, who had undergone L4 vertebroplasty three years earlier, presented with sudden-onset shortness of breath and pleuritic chest pain. ECG revealed right ventricular strain, while blood tests showed elevated D-dimer and troponin levels. [31] A CT pulmonary angiogram detected a 5 cm cement pulmonary embolus lodged in the right main pulmonary artery, surrounded by a thrombus. Bone cement pulmonary embolism is a serious

complication of vertebral surgery, with an estimated incidence of approximately 0.9%. [32] Polymethylmethacrylate (PMMA) rapidly polymerizes after injection but can leak into the thoracic venous system through the basivertebral veins and vertebral venous plexus. [33] From there, it may travel to distant locations, including the pulmonary vasculature. Symptoms typically appear weeks to months after the procedure. [34] It has been suggested that performing a chest radiograph after vertebroplasty may aid in detecting cement embolism; however, this is not a standard practice. [35] In cases of symptomatic cement pulmonary embolism, case reports indicate that therapeutic anticoagulation may be a recommended treatment. [36]

Another case involves a 73-year-old patient who underwent percutaneous vertebroplasty (PVP) for a thoracolumbar vertebral compression fracture. [37] Following the procedure, the patient developed chest tightness and dyspnea. Further evaluation revealed multiple high-density foreign bodies in the blood vessels and heart, accompanied by multi-organ dysfunction. The embolism was attributed to bone cement leakage. The patient's condition improved after surgical intervention and anticoagulant therapy. Key risk factors of bone cement pulmonary embolism include the fracture location, the number of affected segments (more frequently observed in thoracic vertebrae [38]), the nature of the fracture or bone destruction (more prevalent in tumor-related fractures [39]), the volume of bone cement used, its mixing ratio, viscosity, polymerization state [40] , and the surgical technique employed. [41]

Tranexamic acid (TXA)

A 46-year-old Asian woman, who was generally healthy with no notable medical history aside from a one-year history of menorrhagia treated with tranexamic acid, developed a pulmonary embolism. [42] She initially presented to the emergency department with a two-week history of intermittent, pleuritic, central chest pain. Due to the absence of significant thromboembolic risk factors and largely unremarkable diagnostic findings, she was discharged with a diagnosis of musculoskeletal pain on two separate hospital visits. On her third visit to the emergency ambulatory clinic with persistent pleuritic chest pain, a pulmonary computed tomographic angiography confirmed bilateral subsegmental pulmonary embolism. Tranexamic acid is widely considered a safe, well-tolerated and effective antifibrinolytic agent used to manage excessive bleeding in various medical and surgical conditions. [43] However, in this case, TXA was suspected to be the primary contributing factor to the pulmonary embolism, as no

other significant risk factors were identified. The absence of significant risk factors for PE and negative D-dimer probably explained why the patient was reassured and discharged to home during her first and second visits to the ED. It is important to know that TXA can alter D-dimer test results, causing false-negative results. [44, 45]

Right basilic vein thrombosis

One of the not so obvious cases is a 40-year-old woman who came to the emergency room with pain in her right upper limb after sleeping on that side overnight. Before falling asleep she had no symptoms. The only deviation in patient examination was tachycardia. Her risk factors included smoking and oral contraception. After all, she was diagnosed with thrombosis of the antecubital vein, which caused pulmonary embolism. [46]

Cushing's syndrome

Sometimes pulmonary embolism may be a non-specific symptom of chronic diseases such as Cushing's syndrome. The case concerns a 37-year-old woman who suffered from hypertension, obesity and gestational diabetes. The occurrence of pulmonary embolism after delivery led to an appropriate diagnosis - PE. [47] Another case; A 25-year-old woman with no significant past medical history and using oral contraceptives presented to the emergency department due to shortness of breath. [48] Laboratory tests showed an increased concentration of D-dimer. Computed tomography of the pulmonary arteries showed peripheral pulmonary embolism and an incidental tumor of the left adrenal gland. In-depth diagnostics revealed Cushing's syndrome. These cases illustrate that a thromboembolic event may be the first, life-threatening symptom of Cushing's syndrome. Hypercortisolemia should be included in the differential diagnosis of a thrombotic event, despite the coexistence of a minor transient factor, such as the use of oral contraception. Although hypercoagulability in Cushing's syndrome is attracting increasing attention, there are still no guidelines for a standardized anticoagulation regimen in these patients.

Urinary retention

In many cases, medicine can surprise us by demonstrating its extraordinary ability to identify rare, difficult-to-diagnose diseases and to use innovative diagnostic and therapeutic methods. Complicated interactions between various diseases, the complexity of the body's response to treatment and the unpredictability of the course of many diseases can lead to unexpected

results that require flexibility, advanced knowledge and creativity in the approach to diagnosis. This was the case of a 77-year-old patient who developed deep vein thrombosis and PE after resolution of urinary tract obstruction. [49] In another case of a 68-year-old man with multiple pulmonary emboli as the first sign of compression of the inferior vena cava by a bloated 5-liter bladder. [50] Another example is a 75-year-old Japanese man who presented to the hospital with lower extremity weakness and bilateral leg swelling. Contrast-enhanced CT scan showed thrombus in both intra-pelvic veins and the right pulmonary artery secondary to a distended bladder and retained urine. [51]

Septic shock

Pulmonary embolism can result from the embolization of thrombus containing microorganisms into the pulmonary vasculature, leading to infarctions and microabscess formation. [52] A case was reported involving a 45-year-old man with poorly controlled diabetes who developed septic pulmonary emboli. [53] The patient initially presented with fever and painful abscess in the area of the left buttock . An incision and drainage procedure was performed and the collected pus was sent for analysis. Gram staining revealed gram-positive cocci in clusters, and subsequent culture identified methicillin-resistant Staphylococcus aureus (MRSA). Two days later, the patient experienced a new-onset cough and shortness of breath. CT scan confirmed multiple cavitating nodules with a feeding vessel sign and bilateral pleural effusion. Based on the clinical presentation and imaging findings, the final diagnosis was septic pulmonary embolism (SPE) secondary to a gluteal abscess. Septic pulmonary embolism is commonly associated with intravenous drug use, tricuspid valve infective endocarditis, septic thrombophlebitis, the presence of indwelling intravascular catheters and infections of the skin and soft tissue. [54] A systematic review of SPE from various causes found that blood cultures most frequently identified methicillin-sensitive Staphylococcus aureus (MSSA) in 48 out of 168 cases (28.6%) and methicillin-resistant Staphylococcus aureus (MRSA) in 27 out of 168 cases (16.1%). SPE was linked to a poor prognosis, with a mortality rate of 10.1%. [54]

Another case involved an 85-year-old man who presented with a two-day history of fever and worsening malaise, without any respiratory symptoms. [55] Laboratory tests revealed leukocytosis and elevated C-reactive protein levels. A chest radiograph showed multiple small infiltrates in both lungs, prompting further evaluation with a CT scan, which revealed multiple bilateral pulmonary nodules predominantly in the subpleural regions - findings suggestive of

SPE. After hospital admission, Parvimonas micra was identified in the patient's blood culture. Further investigations led to the identification of apical periodontitis and an infratemporal fossa abscess as the primary sources of infection contributing to SPE.

Hormonal therapy of menopause

Systemic hormone therapy for menopause is considered to be the most effective method of treating prolapse symptoms, but this carries certain risks, including deep vein thrombosis. Study based on registries of a population of women in Sweden concluded that the risk of pulmonary embolism was significantly increased in women using oral but not transdermal hormone therapy, with the highest risk in women using oral estrogen in combination with medroxyprogesterone acetate for the first time. The risk was significantly lower in women with repeat treatment. [56]

Antiphospholipid syndrome

A 25-year-old woman experienced sudden-onset dyspnea following an elective cholecystectomy. [57] She had a history of lower limb DVT one year prior, for which she completed six months of anticoagulation. On examination, she exhibited right leg edema. Laboratory findings showed elevated troponin, pro-B-type natriuretic peptide, and D-dimer levels. Computed tomography pulmonary angiography (CTPA) confirmed a large, occlusive PE, while an echocardiogram revealed right ventricular dysfunction. The patient underwent successful thrombolysis with alteplase. She had an uneventful recovery and was discharged on a vitamin K antagonist. Given the recurrence of unprovoked thrombotic events, an underlying thrombophilia was suspected. Subsequent hypercoagulability studies confirmed primary antiphospholipid syndrome (APS) and hyperhomocysteinemia. APS is a systemic autoimmune disorder with an incidence of 5 per 100,000, characterized by vascular thrombosis, obstetric complications and the presence of antiphospholipid antibodies (aPL). [58] It can manifest as primary APS or be associated with other autoimmune diseases, most commonly systemic lupus erythematosus (SLE). [58,59] Hyperhomocysteinemia is linked to an increased risk of venous thromboembolism (VTE) and can further exacerbate the thrombotic predisposition in APS. Studies indicate that hyperhomocysteinemia coexists in approximately 30.8% of patients with primary APS. [60] Overall, individuals with APS face

up to a tenfold increased risk of an initial VTE compared to the general population, with recurrence rates ranging from two - to sixfold higher. [61] However, the occurrence of high-risk PE due to the combined effects of primary APS and hyperhomocysteinemia remains uncommon.

Pregnancy

Pregnancy is associated with an increased risk of embolism, this is due to a number of mechanisms e.g. changes in the balance between coagulation and anticoagulation factors, an increase in blood viscosity. The growing uterus puts pressure on blood vessels (especially veins), blood flow in the lower extremities may be impeded, leading to blood stasis and promoting the formation of thrombi. During pregnancy, especially in the later stages, a woman may be less active, which can lead to a disruption of proper circulation, especially in the legs. This, in turn, can increase the risk of thrombosis and possibly pulmonary embolism. The increased risk of venous thromboembolism during pregnancy peaks shortly after delivery and may persist for up to 12 weeks after delivery. [62]

Catheter in the jugular vein

Due to severe septic shock from a 57-year-old male patient, a central catheter was inserted into the right jugular vein to facilitate rapid fluid resuscitation and administer vasoactive drugs. During hospitalisation the hemodynamics was stable, low molecular weight heparin calcium was added for anticoagulation to prevent venous thromboembolism. On day 15, during the process of removing the venous catheter, the patient suddenly lost consciousness and suffered cardiac arrest. A bedside ultrasound showed a thrombus in the right jugular vein. The patient was given intravenous urokinase.

Twenty minutes after the administration of thrombolysis, the patient's autonomic heart rhythm was restored, but hypotension and coma continued, followed by multiple organ failure. The patient died 50 hours later. [63]

There is no evidence that routine ultrasound examination for thrombosis is required before catheter removal. However, according to clinical experience, ultrasound before catheter removal can detect asymptomatic thrombosis at an early stage and anticoagulant treatment can be given in time to reduce the risk of pulmonary embolism. Despite appropriate

antithrombotic prophylaxis, the patient developed this complication. For patients at particularly high risk for CRT, consideration can be given to using higher doses of anticoagulant as prophylaxis, although there are virtually no data to support this approach

Gender differences

The prospective cohort study included patients with confirmed PE over a 10-year period showed that men were more likely to have cancer and unprovoked PE, while women were more likely to present risk factors for venous thromboembolism, such as older age, surgical procedure/injury/immobilization and hormone therapy. Adverse effects of treatment at 30 days did not differ between male and female PE patients. [64]

Summary and conclusions

Pulmonary embolism is a life-threatening condition that occurs when a thrombus (embolism) blocks one of the main blood vessels in the lungs, impeding blood flow and reducing oxygenation to the body. The embolism can originate in the deep veins of the legs (deep vein thrombosis) or elsewhere in the body. Symptoms of PE are often nonspecific and can include shortness of breath, chest pain, coughing (sometimes with blood), accelerated breathing and heart rate, and fainting or unconsciousness. Diagnosis of PE is often difficult because the symptoms can resemble other conditions, such as heart disease (heart attack) or lung disease (pneumonia). Therefore, suspected PE may be missed or misdiagnosed. In addition, some cases of PE may be atypical, making diagnosis even more complicated.
Non-obvious diagnosis of pulmonary embolism can include situations where the symptoms of PE are not classic or are confused with other conditions. Examples include: pulmonary embolism with atypical symptoms - in some cases, PE may manifest with only mild chest pain, pain in the right upper limb or a feeling of fatigue, which can easily be ignored or mistakenly attributed to other conditions. Pulmonary embolism in patients with comorbidities. People with chronic diseases such as urological diseases, diabetes, hematological diseases or endocrine imbalance may experience symptoms of PE that are more difficult to recognize because they may be mistaken for exacerbations of these diseases. Pulmonary embolism in younger patients - in younger people, especially those without apparent risk factors, PE may be overlooked and symptoms may be misinterpreted as the result of stress, fatigue or viral

infections. Pulmonary embolism in people who have undergone surgery - especially orthopedic surgery or transplantation, the symptoms of PE may be overlooked because they are considered a common side effect of surgery. Given the difficulties in diagnosing PE, proper risk assessment, the use of advanced diagnostic techniques such as computed tomography, echocardiography or blood gas analysis and a thorough patient history are crucial and necessary to include the appropriate treatment.

Disclosure

Author's contribution

Conceptualization: Julia Kozakiewicz

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Formal analysis:

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