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PULMONARY THROMBOEMBOLISM - DIAGNOSTICS

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ABSTRACT. Pulmonary thromboembolism represents the obstruction in the arterial pulmonary tree by thrombi formed in the deep venous system or in the right cavities of the heart. We present the case of a 77 year old woman who was admitted in the emergency room with progressive dyspnea, fever, cough and sinus tachycardia. Blood test showed positive d-dimers and later the CT scan revealed multiple thrombi in the lung segmentary arteries.

Keywords: psychic factors, cardiovascular illnesses, collagen ilnesses, psychological questionnaires.

INTRODUCTION

Case report

A 77-year-old woman presented with a 3-day history of progressively worsening dyspnea, orthopnea and fatigability. On physical examination, the heart rate was 120 beats per minute, and the blood pressure 120/80 mm Hg. The arterial oxygen saturation was 91% while the patient was breathing ambient air. The neck veins were distended. There was no heart murmur. The lungs were clear, and the extremities appeared normal. Examination of the abdomen showed normal bowel sounds; the liver and spleen were not felt, and no mass, hernia, or tenderness was detected. Examination of the extremities revealed no peripheral edema or cyanosis. Neurologic examination was negative. The urine sediment contained 3-4 red cells,1-2 white cells, and the urine presented 2 crosses of urobilinogen. The white-cell count was 8960, with 67 percent neutrophils, 1,9 percent band forms, 23,5 percent lymphocytes, and 7,71 percent monocytes. The platelet count was 255,000, and the erythrocyte sedimentation rate 66 mm per hour. The hematocrit was 41,4 percent. The urea nitrogen was 36,4 mg per 100 ml, and the creatinine 0,93 mg per 100 ml. The glucose 226 mg per 100 ml with a HbA1c of 7,36%, the conjugated bilirubin 1,7mg per 100 ml, the total bilirubin 3,1 mg per 100 ml. The serum aspartate aminotransferase (ASAT) was 12U (new normal for a man, 10 to 40), the lactic dehydrogenase (LDH) 412 U (new normal, 110 to 210), the creatine kinase (CK) 68 U per liter and the alkaline phosphatase 144 U per liter. The D-dimer level was 5 mg per liter (normal level, less than 0.5). An electrocardiogram showed sinus tachycardia without other significant signs of thromboembolism (fig 1.). An x-ray film of the chest showed a normal heart, the lungs were clear, with mild vascular redistribution.Lower-extremity noninvasive studies gave no evidence of deep venous thrombosis

A computed tomographic (CT) scan showed multiple thrombi in the pulmonary segmentary arteries of the right superior lobe and inferior bilateral lobes (fig.2).

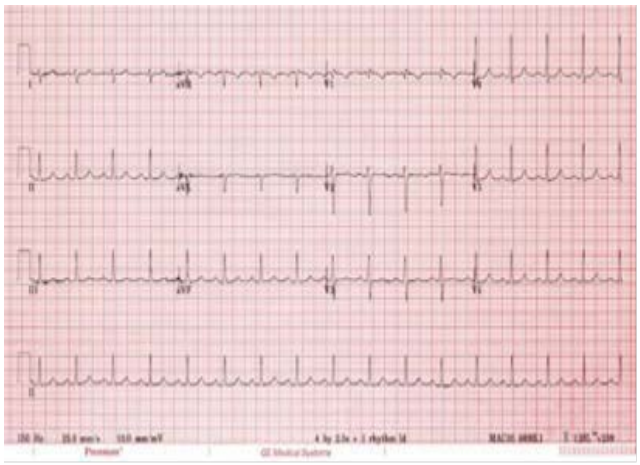

Fig. 1.

The temperature rose as high as 38.1°C during the first two hospital days.

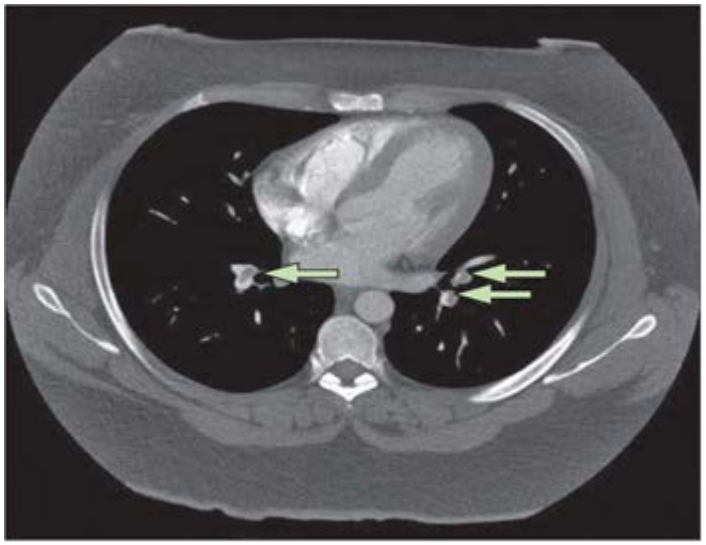

Fig.2. CT scanning of the torax

Medical therapy with low molecular weight heparin (enoxaparinum) was initiated along with oral anticoagulant (acenocumarolum 4mg). Low molecular

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weight heparin was stopped when INR reached a level between 2-3.

DISCUSSIONS

Pulmonary embolism and deep venous thrombosis represent the spectrum of one disease. Thrombi commonly form in deep veins in the calf and then propagate into the proximal veins, including and above the popliteal veins, from which they are more likely to embolize. About 79% of patients who present with pulmonary embolism have evidence of deep venous thrombosis in their legs; if deep venous thrombosis is not detected in such patients, it is likely that the whole thrombus has already detached and embolized (Sandler D.A. Autopsy proven pulmonary embolism in hospital patients: are we detecting enough deep vein thrombosis?).

Conversely, pulmonary embolism occurs in up to 50% of patients with proximal deep venous thrombosis. Because of the dual pulmonary circulation arising from the pulmonary and bronchial arteries, pulmonary infarction is not usually present. In acute pulmonary embolism, anatomical obstruction is undoubtedly the most important cause of compromised physiology, but the release of vasoactive and bronchoactive agents such as serotonin from platelets may lead to deleterious ventilation–perfusion matching (Elliot C.G. Pulmonary physiology during pulmonary embolism).

As right ventricular afterload increases, tension in the right ventricular wall rises and may lead to dilatation, dysfunction, and ischemia of the right ventricle. Death results from right ventricular failure.

Although less common in certain regions, such as Asia, venous thromboembolism is a worldwide problem, particularly in people with known risk factors (Leizorovicz A. Epidemiology of venous thromboembolism in Asian patients undergoing major orthopedic surgery without thromboprophylaxis: the SMART Study). The pacient was admitted in the emergency room with fever, cough and dyspnea, symptoms which arised the suspicion for an acute pneumopathy. But a high blood level of d-dimers (5 mg per liter) and Wells score of 4 indicated de necessity for computer tomography. During this admission the pacient was diagnosticated with type 2 diabetus mellitus and insulin therapy was initiated in association with oral antidiabetic agents.

The diagnostic workup for patients with suspected pulmonary embolism should begin with an assessment of the clinical probability on the basis of validated explicit scores. When the probability is low or intermediate, a negative D-dimer test (level below 0.5 mg per liter) essentially rules out the diagnosis, whereas a positive result indicates the need for further testing, preferably multidetector CT scanning. The patient in the vignette had an intermediate clinical probability and a positive D-dimer test, and a CT scan confirmed the diagnosis of pulmonary embolism. Anticoagulation therapy should thus be initiated promptly. New oral anticoagulants, including the direct thrombin inhibitor dabigatran and the factor Xa inhibitors rivaroxaban and apixaban, are currently being tested as alternatives to acenocumarolum for long-term secondary prophylaxis against venous thromboembolism.

Prognosis

Most patients with acute pulmonary embolism who receive adequate anticoagulant therapy survive. The 3-month overall mortality rate has been reported to be about 15 to 18% (Goldhaber S.Z. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER)).Shock at presentation is associated with an increase in mortality by a factor of three to seven; a majority of the deaths among patients presenting in shock occur within the first hour after presentation (Alpert J.S. Mortality in patients treated for pulmonary embolism.).

Both chronic leg pain and swelling (the post-thrombotic syndrome) and chronic thromboembolic pulmonary hypertension are possible long-term sequelae of acute pulmonary embolism (Schulman S. Post-thrombotic syndrome, recurrence, and death 10 years after the first episode of venous thromboembolism treated with warfarin for 6 weeks or 6 months; Pengo V. Incidence of chronic thromboembolic pulmonary hypertension after pulmonary embolism).

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