

An 89-year-old patient with acquired murmur associated with pulmonary embolism

Maciej Godycki-Ćwirko, Agnieszka Bratkowska

Department of Family and Community Medicine, Medical University of Lodz, Poland

Submitted: 25 April 2010

Accepted: 6 July 2010

Arch Med Sci 2011; 7, 5: 902-904

DOI: 10.5114/aoms.2011.25569

Copyright © 2011 Termedia & Banach

Corresponding author:

Agnieszka Bratkowska
Department of Family
and Community Medicine
Medical University of Lodz
20 Kopcinskiego
90-153 Lodz, Poland
Phone: +48 42 679 55 46
Fax: +48 42 678 60 41
E-mail: lusia310@op.pl

Abstract

Pulmonary embolism (PE) is the third most common cause of death in hospitalized patients. Diagnosis is often missed because of a non-homogeneous clinical picture. We present a case of an 89-year-old patient with an acquired murmur associated with pulmonary embolism. When examined by a family physician the patient had no symptoms typical for PE. During hospitalization, dyspnoea was exacerbated; a non-productive cough, chest pain and oliguria were observed. Pulmonary embolism was diagnosed, but because of the renal failure diagnosis was not confirmed by angio-CT.

Key words: pulmonary embolism, heart murmur, symptoms, diagnosis.

Introduction

Pulmonary embolism (PE) is obliteration of a pulmonary artery caused by an embolus. Usually this is a detached fragment of a thrombus although alternatives include air embolus, fat embolus, cancer embolus, foreign body or amniotic fluid. Thrombi usually arise in the veins of the pelvis or lower limb, especially following surgery or confinement to bed, although occasionally they can arise in the upper part of the body. Emboli are most frequently localized in the inferior lobe of the lung.

Pulmonary embolism is the third most common recorded cause of death in hospitalized patients. Autopsy data on patients who died in the hospital revealed its presence in 60% of cases, but the diagnosis was missed in up to 70% of the cases [1]. In the early 1970s, the incidence of PE in the United States was estimated at 1 case per 1000 persons per year [1]. One report from the USA suggested a frequency of 630,000/year with 50,000-100,000 deaths [2]. However, mortality from PE has decreased in the United States. Analysis of almost 43 million death certificates over 20 years found a 1.3% rate of PE, but the age-adjusted rate of death from PE decreased from 191 per million in 1979 to 94 per million in 1998 [3].

A more recent report from the United Kingdom based on 6,550 newly diagnosed patients in the General Practice Research Database gave an incidence of venous thromboembolism of 74.5 per 100,000 people/year [4]. Pulmonary embolism is more common in adults and its incidence increases steadily with age [5, 6].

In this report, we present an 89-year-old patient who had not presented symptoms that are supposed to be typical for PE. Instead a loud heart murmur was observed, which is not a typical symptom in patients with this condition reported in the literature.

Case report

A family physician was asked to see at home an 89-year-old male patient with a medical history of hypertension and ischaemic heart disease.

The patient presented with breathing difficulties and fatigability. Physical examination revealed normal body temperature, regular heart action at 68 beats/min, blood pressure 104/60 mmHg, vesicular pulmonary sounds and oedema of the lower limbs. Cardiac examination revealed a systolodiastolic murmur best heard over the apex. The patient was referred to a hospital with a diagnosis of worsening heart failure. During hospitalization the patient's condition became worse. His dyspnoea was exacerbated and he developed a non-productive cough and chest pain. Oliguria was also observed. Diagnostic investigation revealed increasing levels of troponin and D-dimer. Evidence of renal failure was also found. A 4 mm ST segment depression in V3-V6 was detected by 12-lead ECG. Echocardiography revealed features of pulmonary hypertension, with dilation of the right ventricle, right atrium and pulmonary trunk.

Pulmonary embolism was diagnosed, but because of the renal failure this diagnosis was not confirmed by angio-CT. The poor general condition of the patient disqualified him from invasive treatment in the haemodynamic unit. Over several days the patient's condition deteriorated. Acute left ventricular insufficiency occurred and the renal failure was exacerbated. Intensive treatment was unsuccessful and the patient died several days after admission.

Discussion

Pulmonary embolism is a disease with a non-homogeneous clinical picture. The picture depends on the localization and size of the thrombus as well as the patient's general condition. Many symptoms of PE are found in the literature but most of them are not typical, and this makes diagnosis difficult [7].

In "Prospective Investigation of Pulmonary Embolism Diagnosis II" (PIOPED II), 192 adults with

acute PE, of whom 133 (69%) had no prior cardiopulmonary disease, were compared with 632 patients without PE [8]. The investigation revealed that symptoms such as dyspnoea (at rest not during exertion), cough, circulatory collapse, calf or thigh swelling and pain occurred more often in patients with PE. Orthopnoea, pleuritic pain, wheezing and haemoptysis were found to be not significant. Signs observed in PE were tachypnoea, tachycardia, increased P₂ heart sound, right ventricular lift, jugular venous distention, pulmonary rales (crackles) and decreased breath sounds. There were also signs of deep vein thrombosis (DVT) of the calf or thigh such as oedema, erythema, tenderness or palpable cord. Other signs such as cyanosis, wheeze, rhonchi, or pleural friction rub were not significant.

For the family physician it is important to estimate the risk of PE without using diagnostic tests. Wells *et al.* have created a model for clinical diagnosis of PE, which relies on a careful history and physical examination, and can be useful to a family physician [9, 10]. Wells's model consists of seven signs and symptoms to be scored (Table I).

Patients who scored more than 6 points were classified in the high risk group (probability of PE 37.5%). Patients with less than 2 points belonged to the low risk group (probability of PE 1.3%).

A second decision model, for use in hospital, was developed by Wicki *et al.* [10]. This model does not rely on a judgment of whether an alternative diagnosis is less likely than PE and it uses information from a chest radiograph and arterial blood gas measurements. In this model assessment includes patient's age, pulse rate, PaCO₂, PaO₂, chest radiograph, previous DVT or PE, and recent surgery.

In a systematic review of hospital diagnostic tests for confirmation or exclusion of PE, a number of tests can be found for which the post-test probability is > 85% if positive. In patients with a moderate or high pre-test probability, spiral computed tomography gives a positive likelihood ratio of 24.1; with high pre-test probability, ventilation perfusion lung scan gives a positive

Table I. Wells model for clinical diagnosis of PE

Signs and symptoms	Points
Clinical signs and symptoms of DVT	3
Pulmonary embolism as likely or more likely than an alternative diagnosis	3
Heart rate > 100 beats/min	1.5
Immobilization (bed rest for at least 3 consecutive days) or surgery in the previous 4 weeks	1.5
Previous objectively diagnosed DVT or PE	1.5
Haemoptysis	1.0
Malignancy (treatment that is ongoing, within the past 6 months, or palliative)	1.0

likelihood ratio of 18.3, and leg vein ultrasound gives a positive likelihood ratio of 16.2.

There are also tests for which the post-test probability is < 5% if negative. In patients with a low or moderate pre-test probability, negative spiral computed tomography plus a negative leg vein ultrasound gives a negative likelihood ratio of 0.04; normal or near normal lung scan has a negative likelihood ratio of 0.05 and a D-dimer < 500 µg/l by quantitative enzyme-linked immunosorbent assay has a negative likelihood ratio of 0.08.

To make a diagnosis, one should also estimate risk factors associated with an increased risk of venous thromboembolism. Possible risk factors that should be considered in the presenting patient are varicose veins, overweight, ischaemic heart disease (risk of PE but not DVT) and heart failure (risk of PE but not DVT).

Other possible risk factors noted in the literature are fractures, surgery, cancer, oral corticosteroid use, opposed hormone therapy, oral contraceptives, cerebrovascular disease (risk of PE but not DVT) and inflammatory bowel disease.

In the case reported here, the patient had no symptoms typical for PE when first examined by the family physician. He reported dyspnoea and fatigability. Physical examination revealed low blood pressure and non-significant lower limb oedema. The heart murmur was the main symptom in this case. This clinical picture might suggest exacerbation of cardiac failure. During hospitalization new symptoms such as chest pain and cough occurred and the dyspnoea was exacerbated. The heart murmur observed in this patient was probably the result of right cardiac overload caused by growing pulmonary vascular resistance. We may suspect that in this patient a massive embolus occurred, leading to haemodynamic disorder, without pulmonary infarction, and therefore without cough, pleural pain or haemoptysis. This hypothesis was not confirmed by angio-CT.

In the case of the 89-year-old patient that is reported here, differential diagnosis was particularly difficult because of non-specific presentation. In the elderly we can often find coexisting diseases that can cause similar symptoms and their treatment can change PE progress. Cardiopulmonary conditions may mimic PE and age may influence the diagnostic tests for PE. Fewer than 20% of elderly have the classic triad of dyspnoea, chest pain and haemoptysis. They often present confusing symptoms such as unexplained fever or arrhythmias, shortness of breath, heart failure, wheezing, cardiovascular collapse with hypotension and syncope. The most common symptom in the elderly is tachypnoea.

Pulmonary embolism is often underdiagnosed in the elderly, although its incidence increases

steadily with age. We conclude that this problem is not sufficiently described in the literature. We should be aware that in many cases symptoms supposed to be found in PE do not occur at all. It is possible to make a diagnostic mistake when ruling out PE in a patient without typical symptoms.

Acknowledgments

The authors wish to thank Professor Carl Whitehouse for reviewing the manuscript.

References

1. Kamangar N. Pulmonary embolism. Accessed on emedicine Jan 14, 2010.
2. Dallen JE, Alpert JS. Natural history of pulmonary embolism. *Prog Cardiovasc* 1975; 17: 257-70.
3. Horlander KT, Mannino DM. Embolism mortality in the United States. *Arch Intern Med* 2003; 163: 1711-7.
4. Huerta C, Johansson S, Wallander MA, Garcia Rodriguez LA. Risk factors and short-term mortality of venous thromboembolism diagnosed in the primary care setting in the UK. *Arch Intern Med* 2007; 167: 953-43.
5. Ceccarelli E, Masotti L, Barabesi L, Forconi S, Cappeli R. Pulmonary embolism in very old patients. *Aging Clin Exp Res* 2003; 15: 7-122.
6. Ceccarelli E, Masotti L, Forconi S, Cappeli R. Diagnostic and therapeutic features of pulmonary embolism in elderly patients. *Ital Heart J Suppl* 2003; 9: 745-54.
7. Evans W. The less common forms of pulmonary hypertension. *Br Heart J* 1959; 21: 97-217.
8. Stein PD, Beemath A, Matta F, et al. Clinical characteristics of patients with acute pulmonary embolism. *Am J Med* 2007; 120: 871-9.
9. Ebell MH. Suspected pulmonary embolism: evidence-based diagnostic testing. *Am Fam Physician* 2004; 69: 599-601.
10. Ebell MH. Suspected pulmonary embolism: evidence-based clinical assessment. *Am Fam Physician* 2004; 69: 367-9.