Acute Silent Non-Massive (Submassive) Pulmonary Embolism

Geza Bozoky[1*] and Eva Ruby[2]

[1]Hospital of Bács-Kiskun County Municipality
[2]Department of Pulmonology, Kecskemet

***Corresponding Author:** Geza Bozoky, Hospital of Bács-Kiskun County Municipality

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ABSTRACT

The aim of our retrospective study was to define the incidence of silent (occult) acute non-massive (submassive) pul-monary embolism in the period between 1996 and 2017, the characteristic clinical symptoms and the possible methods of setting up the diagnosis. Between 1996 and 2007, we diagnosed acute pulmonary embolism in 965 patients based on the clinical symptoms, increased D-dimer value, ECG-deviations, ventilation-perfusion lung scintigraphy and contrast enhanced CT angiography. Acute massive pulmonary embolism was detected in 98 cases out of 965 patients (10.2%) and acute submassive pulmonary embo-lism in 867 patients (89.8%). Silent acute sub-massive pulmo-nary embolism was recognized in 394 cases (45.4%) out of the 867 patients suffering from acute submassive pulmonary embolism. Nearly 50% of pa-tients suffering from the silent form developed only dyspnoea, while in 50% of the cases thoracic pain, cough and haemoptoe also occurred as a second symptom beside dyspnoea. Deep-vein thrombosis of the lower extremities, mainly in proximal localization, was de-tected in 146 cases (37%) by compressional Doppler ultrasonography of the lower extremities. The diagnosis of acute submassive pulmonary embolism was set up by ventilation-perfusion scintigraphy in fewer cases, while it was detected by contrast en-hanced CT angiography in the majority of the patients.

Keywords: Acute Submassive Pulmonary Embolism; Silent Acute Submassive Pulmonary Embolism; Acute unexplained Dyspnoea; Contrast Enhanced CT Angiography

INTRODUCTION

Although, in most cases the early clinical recognition of acute pulmonary embolism (aPE) does not cause difficulty, the exact, early diagnosis and the onset of targeted, adequate treatment is often in delay (1,2). Proper evaluation of the clinical symptoms of aPE (Table I.) is essential for indicating and performing further diagnostic steps to avoid a fatal outcome and to reduce the probability of developing chronic thrombo-embolic pulmonary hypertension (1, 3,4). The knowledge of predisposing

Table 1: Symptoms and clinical signs in acute pul-monary embolism (1)

SYMPTOMS	CLINICAL SIGNS
Dyspnoea 80%	Tachypnoea (>20/min) 70%
Thoracic pain 52%	Tachycardia (>100/min) 26%
Cough 20%	Deep-vein thrombosis 15%
Haemoptoe 11%	Fever (>38.5°C) 7%
Syncope 19%	Cyanosis 11%

factors (Table II) is highly important from the aspect of recognizing aPE. Among these factors, age, earlier deep-vein thrombosis and/or aPE, different cardiac deviations, COPD, active malignant diseases, diseases of the central nervous system accompanied by limb paresis, lasting immobilization, cardio-pulmonary diseases, hormone therapies and inherited thrombophilias (2, 3, 20) should be highlighted and deserve greater attention. Orthopaedic surgeries and general surgical operations during the first 2-3 weeks in the postoperative period make patients susceptible to developing thromboembolisms, but the tendency to thrombosis lasts for more 2-3 months (1-4, 6).

Acute Pulmonary Embolism With Char-acteristic Symptoms

The knowledge of the clinical symptoms and their proper evaluation is essential in the early recognition of aPE. In Table I the most common clinical symptoms and char-acteristics of aPE defined based on evaluating studies with high case number can be seen (1-3,5). Dyspnoea, tachypnoea and thoracic pain is the leader syndrome in near-ly 90% of patients suffering from aPE (1, 2, 5). Isolated dyspnoea often means centrally localized aPE with haemodynamic consequences (5,8).

Syncope is one of the rare, but highly important symptoms of aPE: it can be evaluated with a serious disease (acute massive pulmonary embolism) and it is mostly accompanied with arterial hypotension as a sign of haemodynamic instabil-ity (2,3,8). The Wells score and the revised Geneva score show the clinical probability of aPE based on the symptoms and the clinical characteristics ("clinical prediction rule"), and mainly these point systems are used in daily clinical practice (2,3,9) (Ta-ble III/a, III/b).

Table II: Risk factors for developing venous thromboembolisms (After Tapson VF) (2)

INHERITED FACTORS	ACQUIRED FACTORS
Antithrombin deficiency	Immobility
Protein S deficiency	Elder age
Protein C deficiency	Malignant disease
Leiden (factor V) mutation	Surgical intervention Traumas
Activated protein C resistance (factor V deficiency)	Cardiac deviations COPD Spinal cord injury
Mutation of prothrombin gene	Pregnancy, postpartum period
Dysfibrinogenaemia	Real polycythaemia
Plasminogen deficiency	Antiphospholipid syndrome Hormone therapies Cytotoxic chemotherapy Central venous catheters

General Criteria of Silent (Occult) Acute Pulmonary Embolism

It is a much more difficult exercise for a clinician to recognize silent (occult) aPE in time (6, 10-13). The significance of silent aPE is shown by the fact that, based on clinical observations, silent appearance of the disease can be expected in 40-60% of patients suffering from aPE (6,13,14). Awareness-raising studies related to silent aPE were firstly published between 1987 and 1989 (10,11). The incidence of silent aPE was defined as 50% by Dorfman, 39.5-49.5% by Huisman, 40-50% by Meignaa and 40% by Moser (10-13). The authors also suggest that not negligible observation that the recognition of aPE in a silent form has a seven-day delay contrary to aPE with characteristic symptoms, and as a result it is also possible to develop chronic thrombo-embolic hypertension (4,6,11,12). It is importatnt to mention that APE is almost symptomless in about 5% of patients with active malignant diseases („clinically unsuspected pulmonary embolism") 13,14.

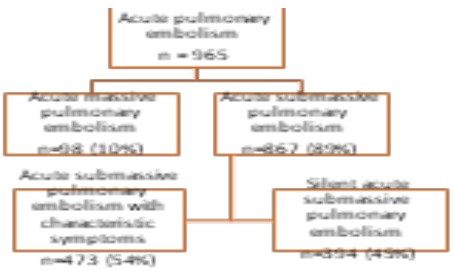
Table IIIa: Clinical probability of acute pulmonary embolism based on symptoms and clinical characteristics (Wells score) (3)

CHARACTERISTICS	POINTS
Predisposing factors:	
Earlier deep-vein thrombosis	+ 1,5
Surgery or immobilization	+ 1,5
Malignant disease	+ 1
Symptoms:	
Haemoptoe	+ 1
Clinical signs:	
Heart rate >100/min	+ 1,5
Signs of deep-vein thrombosis	+ 3
Alternative diagnosis is not probable	+ 3
Evaluation:	
Not probable aPE	0-1
(One level) aPE with medium probability	2-6
Highly Probable aPE	>7
(Two levels) aPE is not probable	0-4
aPE is Probable	>4

Table IIIb: Clinical probability of acute pulmonary embolism based on the symptoms and clinical characteristics (Modified GENEVA score) (3)

CHARACTERISTICS	POINTS
Predisposing factors:	
Age > 65 years	+ 1
Earlier deep-vein thrombosis or pulmonary embolism	+ 3
Surgery or breakage injury during the previous month	+ 2
Active malignant disease	+ 2
Symptoms:	
Pain of extremities	+ 3
Haemoptoe	+ 2
Clinical signs:	
Heart rate 75-94/min	+ 3
Heart rate >95/min	+ 5
Unilateral limb oedema or deep-vein pain in the limb	+ 4
Evaluation:	
Not probable aPE	0-3
aPE with medium probability	4-10
Highly probable aPE	> 11

Table IV: Pattern of acute pulmonary embolism in silent form and with characteristic clinical symptoms in our patients



Clinics of Our Patients with Silent Acute Pulmonary Embolism

The aim of our retrospective study performed between 1996 and 2017 was to research the frequency of the silent (occult) form of the disease in patients suffering from aPE, and the clinical characteristics of this form. It can be seen in Table IV. that aPE occurred in silent form in nearly half (45.4%) of our patients suffering from acute submassive pulmonary embolism. The most common clinical symptoms in patients with silent aPE were dyspnoea (91%) and tachycardia (73%) (Table V). The occurrence of thoracic pain (32%), unproductive cough (25%) and haemoptoe (10%) was significantly less common. The evidence of silent aPE is the fact that only two symp-toms, dyspnoea and tachycardia, could be detected in more than 50% of these patients, and in the other cases thoracic pain, haemoptoe or unproductive cough occurred as a third symptom. Compressive Doppler ultrasound test proved lower limb deep-vein thrombosis in 146 cases (37%) among patients suffering from silent aPE, mainly in proximal localization, which observation is identical to the literary data (5,12,16,21).

Table V: Pattern of clinical symptoms in our patients with silent acute submassive pulmonary embolism

Dyspnoea	n = 359 (91%)
Tachycardia	n = 288 (73%)
Thoracic pain	n = 129 (32%)
Unproductive cough	n = 98 (25%)
Haemoptoe	n = 39 (9.9%)

Pathological thoracic radiological deviation was not found in the majority of patients with silent aPE (n = 273, 69%), while ECG-deviation could be detected in 218 patients (55%) (sinus tachycardia, newly developed right Tawara branch block, atrial fibrillation). D-dimer value of plasma was increased in 94% of patients. In the first three years of the study period the diagnostic value of ventilation-perfusion lung scintig-raphy was dominant in proving the diagnosis of aPE: following the recommendations of PIOPED, the existence of aPE was decided to be proved in cases with two or more "mis-matched" segmental perfusion deficiencies (18). However, after 1999, the performance of contrast enhanced CT angi-ography and its definitely positive result ensured the diagnosis of aPE in all the cases.

Summary

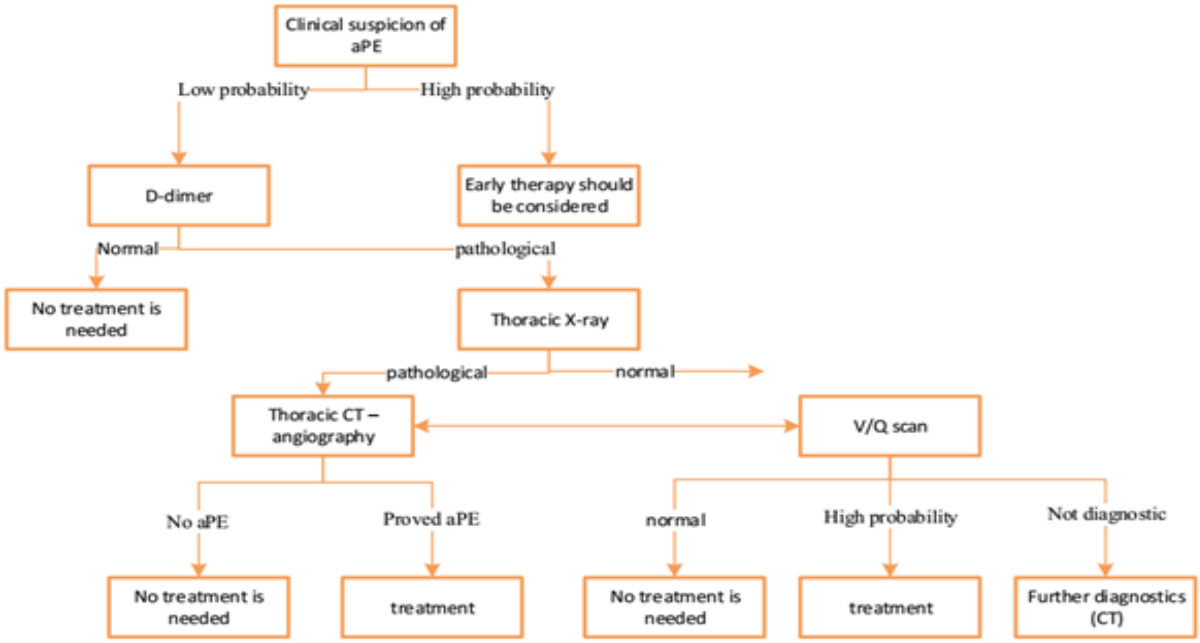
Consideration of predisposing factors, evaluation of the clinical symptoms of deep-vein thrombosis and the targeted di-agnostic interventions which should be per-formed in the typical and silent disease forms are helpful in the early recognition of silent aPE (Table VI.).

The significance of acute unexplained dyspnoea as a leader clinical symptom cannot be emphasized enough in the recognition of silent aPE (1-3, 6). Diagnostic algo-rithm of aPE provides further help in setting up the diagnosis of the disease in time, giving the possibility for the early targeted therapy (1-3, 5, 8, 16) (Table VII).

Table VI: Aspects of early recognition of acute pulmonary embolism

1. Knowledge of predisposing factors
2. Proving deep-vein thrombosis
3. Proper understanding of the symptoms of acute pulmonary embolism, also taking account of silent appearance
4. Performing targeted diagnostic tests to prove acute pulmonary embolism

Table VI: Aspects of early recognition of acute pulmonary embolism



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