Acute pulmonary embolism registry in the Małopolska region – clinical course

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Abstract

Background: Acute pulmonary embolism (APE) is a life-threatening disease. Mortality in APE still remains very high in spite of progress in diagnostic tools. Mortality rate is about 30% in patients with unrecognised APE. APE is one of the main causes of in-hospital mortality.

Aim: To asses management of patients with APE in the Małopolska region.

Methods: This registry consists of 205 consecutive patients who were hospitalised in 6 cardiology departments between 1 January 2005 and 30 September 2007, with the mean age of 65.1 ± 15.3 years (124 females and 81 males). Mean hospitalisation duration 14.6 days (1-52 days).

Results: During hospitalisation 23 (11.2%) patients died. Complications (death, cardiogenic shock, cardiac arrest, use of catecholamines, respiratory therapy and ventilation) during in-hospital stay were observed in 57 (27.8%) patients. Fifty-three patients were haemodynamically unstable (cardiogenic shock or hypotension). The troponin I or T level was assessed in 147 (71.7%) patients and in 50 (34.0%) was positive. In patients with positive troponin we observed 11 (22.0%) deaths, while in patients with normal troponin T or I level 6 (6.2%) deaths occurred. In patients with normal blood pressure we observed a significant difference in mortality in patients with elevated vs. normal troponin level (14.3 vs. 2.5%, p = 0.02). Thrombolytic therapy was used in 20 (9.8%) patients. In patients treated with thrombolytic therapy 9 (45%) deaths were observed. We divided patients according to the ESC 2008 guidelines risk stratification. The 'non-high risk' group consisted of 152 (74.1%) patients, and mortality was 3.9%. The 'high-risk' group consisted of 53 (26.8%) patients. The 'non-high risk' group was divided into the following subgroups: 1. moderate-high (with 2 risk factors: both RV dysfunction and positive injury markers) mortality – 8.1%; 2. moderate subgroup with one risk factor, mortality – 3.6%; 3. low risk – no risk factors – 0% mortality.

Conclusions: 1. In our registry mortality rate in patients with APE was 11%. 2. In about 30% of patients APE was under mask of acute coronary syndrome or syncope, 34% of patients had elevated troponin level, and 30% of patients had complication during hospitalisation. 3. In patients treated with thrombolytics mortality rate was 45%. 4. Reperfusion strategy (trombolysis or embolectomy) in the high risk group was used in only 41% of patients. 5. Elevated troponin level in normotensive patient was associated with 4-fold times higher risk of death. 6. New risk stratification according to the ESC guidelines 2008 correctly predicts prognosis in everyday clinical practise.

Key words: acute pulmonary embolism, mortality, risk stratification, troponin

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Introduction

Acute pulmonary embolism (APE) is a life-threatening situation. Despite progress in diagnosis and treatment it is still associated with extremely high mortality, reaching 30% in improperly treated patients [1]. It is one of the major causes of in-hospital mortality and the third cause (after myocardial infarction and stroke) of cardiovascular mortality. Acute pulmonary embolism may be responsible for up

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to 50 000-200 000 deaths every year in the United States [2-4]. It has been estimated that the number of diagnosed cases of APE is 1.22-1.83 per 1000 persons per year [1, 3]. According to reports from the United States the number of undiagnosed cases of APE is twice as high as diagnosed ones [1]. Based on the statistical data from the Central Statistical Office estimating the population of Poland at the end of the year 2006 at 38 122 000 there should be approximately 57 000 diagnosed cases of pulmonary embolism and approximately 114 000 undiagnosed ones per year, which adds up to 171 000 cases of APE in Poland [1, 5].

Major efforts of recent years in the field of acute cardiovascular states were focused on treatment and development of new management strategies in acute coronary syndromes (ACS). It led to a marked decrease of in-hospital and long-term mortality and reduction of long-term complications of ACS. Until recently PE had remained an underestimated, background 'silent killer'. It took several actions (including the ZATPOL registry) to point out the problem of underestimation of pulmonary embolism diagnostics and treatment [6].

Our study presents a clinical registry, which seems to reflect the situation in most of the centres. Contrary to randomised clinical trials, registries demonstrate real clinical practice.

Clinical registries are also free from limitations created by the methodology of randomised trials and analyse a common management practice in an inhomogeneous population of consecutive patients. Apart from statistical and educational purposes, registries help in health care planning and allow for an objective assessment of medical care in the region.

Methods

Study population

The registry includes consecutive patients hospitalised between 1 January 2005 and 30 September 2007 in the 5 cardiology departments in the Małopolska region. Data were obtained retrospectively based on a dedicated survey. During the described period there were 205 patients (124 women, 81 men) aged 17 to 87 years (mean age 65.1 ± 15.3 years) admitted to the aforementioned departments with the diagnosis of PE. Mean time of hospitalisation was 14.6 ± 8.7 days (1-52 days). Clinical characteristics of patients are presented in Table I.

Table I. Clinical characteristics of patients

Parameter	
Age [years], mean ± SD	65.1 ± 15.3
Women/men, n (%)	124/81 (60.5/39.5)
Stenocardia, n (%)	87 (42.4)
Syncope, n (%)	71 (34.6)
Obesity, n (%)	64 (31.2)
Immobilisation, n (%)	57 (27.8)
Lower extremity deep vein thrombosis, n (%)	94 (45.6)
Haemoptysis, n (%)	7 (3.4)
Neoplastic disease, n (%)	15 (7.3)
Pre-febrile states under diagnosis, n (%)	26 (12.7)
Duration of hospitalisation [days], mean ± SD	14.6 ± 8.7
Heart failure, n (%)	23 (11.2)

Diagnosis of pulmonary embolism

Pulmonary embolism was diagnosed using the following methods: spiral computed tomography (sCT) (167), scintigraphy (5), detection of thrombi in the right heart chambers on echocardiography (8), presence of right ventricular (RV) overload on echocardiography coexisting with cardiogenic shock/hypotonia on admission (14), presence of embolic material (thrombi) on lower-extremity ultrasound coexisting with RV overload on echocardiography and clinical symptoms (9), autopsy (2).

Echocardiography

Right ventricular overload on echocardiography was defined by the increase of end-diastolic right ventricular diameter > 30 mm and/or coexisting right ventricular free wall hypokinesis; increased right ventricular systolic pressure was diagnosed if peak reverse gradient through the tricuspid valve (TVPG) exceeded 30 mmHg [1, 7].

Biochemical tests

Troponin T or troponin I assays were used to evaluate myocardial injury. Department-dependant diagnostic assays included: troponin T (cut-off – 0.03 µg/l), electrochemiluminescence ECLIA test, Roche Diagnostic; troponin I (cut-off – 0.4 µg/l), electrochemiluminescence, Roche Diagnostic; troponin I (cut-off < 0.1 µg/l), Abbott; troponin I (cut-off < 0.1 µg/l), immunoenzymatic test, BIOMARIEUX.

Risk stratification

Risk stratification of patients with APE was based on current 2008 ESC guidelines [7]. Patients were divided into two groups:

1. high risk – patients with symptoms of cardiogenic shock/hypotonia,
2. non-high risk – other patients without cardiogenic shock/hypotonia.

Two subgroups were distinguished in the non-high risk group:

2.1 intermediate risk (patients with right ventricular dysfunction and/or myocardial injury) comprising two subgroups:

2.1.1. intermediate-high risk (IM 2) (patients with coexisting RV dysfunction and myocardial injury),

2.1.2. intermediate-low risk (IM 1) (patients with either RV

dysfunction or myocardial injury assessed with means of elevated troponin concentration);

2.2. low risk: patients without cardiogenic shock/hypotonia or signs of RV dysfunction and with normal myocardial injury biomarkers.

According to current ESC guidelines, markers of RV dysfunction include: RV dilation, RV overload and hypokinesis on echocardiography, RV dilation on sCT, elevated plasma concentration of BNP or NT-proBNP, increase of RV systolic pressure on catheterisation.

Frequency of the following complications was assessed during in-hospital stay: all-cause mortality, cardiac arrest, the use of catecholamines (cardiogenic shock present on admission or diagnosed during hospitalisation), and the need of mechanical ventilation.

Statistical analysis

Continuous parameters with confirmed normal distribution are presented as means ± standard deviations. Qualitative parameters were compared with means of chi-square test (with Yates correction for small samples). Statistical significance was set for $p < 0.05$ (two-sided). Statistical analysis was performed with Statistica PL version 6.1 (StatSoft Inc.)

Results

In-hospital observation

There were 23 (11.2%) in-hospital deaths. Complicated in-hospital stay was observed in 57 (27.8%) patients (death, cardiogenic shock, cardiac arrest, the need of catecholamines or mechanical ventilation) (Table II). The episode of PE was most frequently accompanied by lower extremity deep vein thrombosis (DVT) (45.6%), chest pain (42.1%) and syncope (36.4%). Coexistence of chest pain and syncope was present in 38 (18.5%) patients, lower extremity DVT and chest pain in 44 (21.5%) patients, syncope and lower extremity DVT in 35 (17.1%) and chest pain, syncope and lower extremity DVT in 19 (9.3%) patients.

Patients with cardiogenic shock

On admission, 38 (18.5%) patients had cardiogenic shock/hypotonia (systolic arterial pressure ≤ 90 mmHg). During in-hospital stay 53 patients had cardiogenic shock or hypotonia (on admission or during hospitalisation).

Troponin level was assessed in 40 patients from this group (75.4%) showing elevation in 24 (60%) patients, of whom 8 died (30% mortality). In the same group normal troponin concentration was found in 16 (40%) patients, of whom 4 died (25% mortality) (NS).

Patients with normal arterial pressure

Normal arterial pressure was observed in 152 (74.2%) patients. In this subgroup troponin T or I level was assessed in 107 (70.4%) patients. Increased troponin concentration was found in 28 (26.2%) patients. There were 4 deaths of patients with troponin elevation (14.3% mortality). Normal troponin concentration was noted in 79 patients (73.8%) with 2 deaths in this group (2.5% mortality). In this group there was a significant difference in mortality between patients with normal and elevated troponin level (2.5 vs. 14.3%, $p = 0.02$).

Table II. Complications during hospital stay

Complications	n (%)
Death	23 (11.2)
Shock on admission	37 (18.1)
Shock occurring during hospitalisation	11 (5.4)
Aborted sudden cardiac death	7 (3.4)
Catecholamine use	48 (23.4)
Mechanical ventilation/respirator	15 (7.3)
Patients with complications during hospitalisation	57 (27.8)

Markers of myocardial injury – troponin

Troponin level was assessed in 147 (71.7%) patients from the whole studied group and was elevated in 50 (34.0%) of them. There were 11 deaths among patients with increased troponin concentration (22.0%) and 6 deaths among patients with normal troponin level (6.2%) ($p = 0.01$).

Four of the patients with normal troponin level who died (57%) were classified as having high risk and two as having intermediate risk – RV overload and increased NT-proBNP concentration > 3000 ng/ml.

The NT-proBNP concentration was assessed only in 7 patients and was elevated > 1200 ng/ml in 6 of them. In this group, 2 (28.6%) patients had in-hospital complications, 1 (14.3%) patient died (hypotonia on admission) and 3 (42.6%) patients had coexisting troponin elevation. According to the current ESC classification, 2 patients had high risk of mortality and 5 had intermediate risk of mortality. All patients with elevated NT-proBNP had RV overload on echocardiography. There were no significant differences in the frequency of complications including syncope, chest pain, RV dysfunction on echocardiography or the use of thrombolysis between patients with normal and elevated troponin level. Patients with increased troponin concentration were more likely to present with cardiogenic shock/hypotonia on admission in comparison to patients with normal troponin level (Table III).

Echocardiography

Transthoracic echocardiography (TEE) was performed in 186 (90.7%) patients during the first 24 h of hospitalisation and showed the presence of RV dilation (RVEDD > 30 mm) in 142 (76%) of them. Thrombi in the pulmonary trunk, right atrium and/or RV on TTE were found in 19 (9.2%) patients.

Treatment

Thrombolysis was used in 20 (9.8%) patients from the whole study group. Thrombolytics were administered in 20 (38%) patients with cardiogenic shock/hypotonia [including 18 patients treated with streptokinase (SK) and 2 patients treated with tissue plasminogen activator (tPA)] and 5 (9.4%) patients were referred for surgical treatment (including 3 with contraindications for thrombolysis). There were 9 deaths among patients treated with thrombolysis – 45% mortality. Other patients with cardiogenic shock/hypotonia received unfractionated heparin (UFH) – 28 (52.8%). Both SK and tPA were administered as 2 hour infusion. Bleeding complications occurred in 1 patient (5%) – gastrointestinal bleeding requiring blood transfusion. Treatment of APE was started with UFH in 128 (62.4%) patients or low-molecular-weight heparin (LMWH) in 77 (37.6%) patients. Starting loading dose of UFH was 80 UI/kg administered as intravenous bolus (4000-7000 UI) and followed by intravenous infusion with APTT therapeutic target (1.5-2.5 prolongation in comparison to baseline, APTT range 46-70 sec.). Fifteen patients (7.3%) were referred for surgical treatment (embolectomy). This group included 1 patient with haemodynamic instability after unsuccessful thrombolysis, 5 (33.3%) patients with haemodynamic instability (cardiogenic shock/marked hypotonia), of whom 3 had contraindications for thrombolysis (recent bleeding – 1 patient, stroke in 2 patients) and another 9 patients referred for embolectomy after sCT demonstrating saddle embolus. There were 5 deaths (33.3%) among patients referred for surgery.

Risk stratification and mortality

Patients were divided into groups according to the current 2008 ESC guidelines.

The high risk group consisted of 53 (26.8%) patients. There were 17 deaths in this group (32% mortality).

Non-high risk group included 152 (74.1%) patients. There were 6 deaths in this group (3.94% mortality). In the aforementioned 3 subgroups of patients with non-high risk there were 3 deaths (8.1% mortality) in the intermediate-high risk subgroup, 3 deaths (3.6% mortality) in the intermediate-low risk subgroup, no deaths (0% mortality) in the low risk subgroup (Table IV).

Discussion

Mortality in the course of APE remains high [1-4]. Current 2008 ESC guidelines propose a very useful and simple risk stratification algorithm for patients with an episode of APE [7]. Experts from ESC estimated that mortality in the high risk group exceeds 15%. Estimated mortality of patients from the non-high risk group is: 3-15% in the intermediate risk subgroup and lower than 1% in the low risk subgroup. Our analysis confirms that the proposed new risk stratification in APE reflects the everyday clinical practice. Mortality in our study reached 32% in the high risk group and 3.9% in the non-high risk group. According to ESC guidelines the non-high risk group consists of an intermediate and low risk subgroup.

In our analysis patients from the intermediate risk group were divided into 2 subgroups. First-degree intermediate risk was defined by the presence of 2 risk factors: RV overload and positive markers of myocardial injury (troponins). Mortality in that subgroup reached 8.1%. Mortality in the second subgroup, named second-degree intermediate risk and defined by the presence of only 1 risk factor – RV overload or positive markers of myocardial injury (troponins) – was 3.6%. In our analysis there was 0% mortality in the low risk subgroup.

Table III. Characteristics of patients according to the presence of myocardial injury markers (troponins)

Parameter	Troponin positive n = 50	Troponin negative n = 97	p
Syncope, n (%)	19 (38)	36 (37)	NS
Stenocardia, n (%)	19 (38)	49 (50)	NS
Shock/hypotonia on admission, n (%)	17 (34)	8 (8.2)	0.002
Thrombolysis, n (%)	6 (12)	7 (7.2)	0.33
Embolectomy, n (%)	8 (16)	3 (3.1)	0.005
RV dysfunction, n (%)	38 (76)	68 (70.1)	NS

Table IV. Risk stratification of patients with APE according to current 2008 ESC guidelines

Risk stratification		Number of patients, n (%)	Death, n (%)
High risk		53 (26.8)	17 (32.0)
Non-high risk	all	152 (74.2)	6 (3.9)
	intermediate-high risk	37 (24.3)	3 (8.1)
	intermediate-low risk	82 (53.9)	3 (3.6)
	low risk	33 (21.7)	0 (0)

Except dyspnoea on admission, patients most often presented with the triad of symptoms: low extremity DVT, chest pain and syncope. The differential diagnosis of APE should include ACS (40% of patients with chest pain in the presented registry) and syncope (35% of patients with syncope on admission or immediately before admission). Troponin level was assessed in 71.7% of patients. Elevated troponin concentration was observed in 34% of patients. In the studies performed between 2003 and 2007 troponin elevation was present in 30-50% of patients with APE. Mehta et al. found troponin I elevation in 47% of patients [8], Kucher et al. in 31% [9], Scridon et al. in 52% [10] and Enea et al. in as many as 77% [11]. Pruszczyk et al. disclosed troponin T elevation in 50% of patients with normal arterial pressure [12]. Becattini et al. performed a meta-analysis which demonstrated that troponin elevation was a short-term mortality predictor in the whole population of patients with APE, but also in patients with normal arterial pressure [13].

In our registry mortality was significantly higher in patients with elevated troponin level. There were 11 deaths in this group (22% mortality) in comparison to 6 deaths (6.2% mortality) in the group of patients with normal troponin level. Patients with troponin elevation were more likely to present with cardiogenic shock/hypotonia on admission, which probably influenced the more frequent use of thrombolytics. Patients with troponin elevation were significantly more often referred for surgical treatment in comparison to patients with normal troponin level.

Reports from recent years have suggested that brain natriuretic peptide (BNP) or NT-proBNP may have a high negative predictive value (approximating 100%) for early mortality from APE, which would support the clinical application of these markers in risk stratification aiming at exclusion of acute, life-threatening PE [14-17]. The assessment of NT-proBNP was performed in only one centre from our study in 7% of patients. Level of NT-proBNP exceeded 1200 ng/ml in 85% of patients. In patients with NT-proBNP elevation in-hospital mortality reached 16.7% and 50% of patients had coexisting troponin elevation. Binder et al. [18] found that isolated NT-proBNP elevation > 1000 ng/ml was not an independent predictor of adverse prognosis in patients with PE. The combination of NT-proBNP and echocardiography is useful in the risk stratification and determination of high, intermediate and low risk groups proposed by the current ESC guidelines.

Right ventricular overload was related to 12-fold increase of the complication risk during hospitalisation, while NT-proBNP elevation > 1000 ng/ml and a lack of RV overload on echocardiography was related to insignificant risk increase during hospitalisation. In the Binders et al. study all patients with RV overload had elevated NT-proBNP concentration [18], while in our registry all patients with NT-proBNP level had signs of RV overload on echocardiography. It has been postulated that in patients with normal troponin and BNP (NT-proBNP) concentration echocardiography is not an obligatory examination. In turn, in the case of elevation of biomarkers, echocardiography should be implemented to confirm RV dysfunction/ /overload [19].

There was no significant difference in mortality in patients with cardiogenic shock/hypotonia and elevated or normal troponin concentration. However, in patients with normal arterial pressure mortality was significantly higher in those with elevated troponin level in comparison with patients with normal troponin level. In our registry symptoms accompanying APE such as chest pain or syncope were found equally often in patients with normal and elevated troponin. Also RV dysfunction on echocardiography was equally present in both groups (in 70% of cases).

Of note, in our registry there was a very high (45%) mortality rate in the group of patients treated with fibrinolysis. It should be underlined that this form of treatment was reserved for the most severe cases. Of 9 patients who died, 7 were admitted in cardiogenic shock with mean systolic arterial pressure of 70 mmHg. Among patients treated with fibrinolysis 15 (75%) belonged to the high risk group according to the 2008 ESC risk stratification. The analysis of the ICOPER study demonstrated that 90-day mortality of patients with massive PE (currently named as a high risk group) treated with fibrinolysis was 46.3% [20]. The ICOPER study also showed that thrombolysis does not reduce 90-day mortality in patients with massive pulmonary embolism [20, 21]. In our registry only 15 out of 53 patients with cardiogenic shock/hypotonia (on admission or with onset during hospitalisation) received thrombolysis and 7 patients (13.2%) were referred for surgery. Pharmacological or surgical reperfusion therapy was implemented in only 41.5% of patients from the high risk group. It should therefore be stressed that more than half of high risk patients did not receive thrombolysis. Similar results were obtained in the ICOPER study, where 39% of patients were treated with thrombolysis or surgery (36% and 3%, respectively) [20].

Detection of APE increased with wide access to multi-slice computed tomography or echocardiography. A lot of new information should come from the ZATPOL registry conducted by Torbicki et al. The ZATPOL registry should show the real status of diagnosis, treatment, prognosis and accessibility to diagnostic tests in Polish cardiological centres [6]. It should be kept in mind that a high percentage of patients with PE are not admitted to cardiology units and remain in the internal, neurology or interventional (surgery, orthopaedic) units without a proper diagnosis.

We would also like to point out the diagnostic challenges in the case of masked by ACS in the form of non-ST-elevation myocardial infarction [22-24] or very rarely as ST-elevation myocardial infarction [25, 26]. Despite very early and modern

invasive diagnosis in those patients, the in-hospital course is often very unfavourable. This refers to patients admitted with clinical (chest pain), electrocardiographic (negative T waves in inferior, anterior and inferior, or anterior leads) or biochemical (increased troponin) signs and symptoms typical for ACS. It is worth noting that a differential diagnosis of syncope (after detailed analysis of predisposing clinical factors) should also include PE.

Our study presents a clinical APE registry from the Małopolska region, which seems to mirror the situation in most centres. We look forward with great interest to the results of the large, national ZATPOL registry which collected information on 2015 patients with suspicion of pulmonary embolism [6].

Conclusions

1. In our study mortality in the group of patients with APE reached 11%.
2. The '30% rule' was observed – APE was masked by ACS or syncope in 30-40% of patients, 34% of patients had troponin elevation and 30% of patients had complications during hospitalisation.
3. Only the high risk group (most severe clinical condition) undergoes thrombolysis, but despite that mortality remains very high and reaches 45%.
4. Only 41% of patients from the high risk group (according to current guidelines) undergo reperfusion therapy (thrombolysis or surgery).
5. Troponin elevation is a useful prognostic marker in patients with normal arterial pressure and is related to 4-fold higher risk of death in this subgroup of patients.
6. The new risk stratification model for patients at risk of death from APE proposed by the 2008 ESC guidelines shows an excellent correlation with everyday clinical practice.

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Mały Małopolski Rejestr Ostrego Zatoru Tętnicy Płucnej – przebieg kliniczny

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Streszczenie

Wstęp: Ostry zator tętnicy płucnej (OZTP) jest stanem bezpośredniego zagrożenia życia. Prezentowana praca ma charakter rejestru chorych z OZTP. Rejestr pozwala na przedstawienie rzeczywistej praktyki klinicznej.

Cel: Analiza postępowania diagnostyczno-leczniczego u chorych z rozpoznaniem zatoru tętnicy płucnej na 5 oddziałach kardiologii w Małopolsce.

Metody: Rejestrem objęto kolejnych pacjentów hospitalizowanych od 1 stycznia 2005 r. do 30 września 2007 r. na 5 oddziałach kardiologicznych. Do badania włączono 205 chorych (124 kobiety, 81 mężczyzn) w wieku 17–87 lat (średni wiek 65,1 ± 15,3 roku) z potwierdzonym OZTP. Średni czas trwania hospitalizacji wynosił 14,6 ± 8,7 dnia (1–52 dni).

Wyniki: W czasie obserwacji szpitalnej zmarło 23 (11,2%) pacjentów. Pobyt powikłany obserwowano łącznie u 57 (27,8%) chorych (zgon, wstrząs kardiogenny, zatrzymanie krążenia, konieczność stosowania amin katecholowych lub sztucznej wentylacji). Łącznie 53 chorych miało wstrząs kardiogenny lub hipotonię. Stężenie troponiny oznaczono u 147 (71,7%) chorych, a jego podwyższenie stwierdzono u 50 (34%) chorych. W grupie osób z podwyższonym stężeniem troponiny doszło do 11 (22%) zgonów, a w grupie z prawidłowym stężeniem troponiny do 6 (6,2%) zgonów (p = 0,01). U chorych stabilnych hemodynamicznie różnica w śmiertelności pomiędzy podgrupą osób z prawidłowym i podwyższonym stężeniem troponiny (2,5 *vs* 14,3%) była istotna statystycznie (p = 0,02). Leczenie trombolityczne stosowano u 20 (9,8%) osób, w tym 18 chorych leczono streptokinazą (STK), a 2 chorych tkankowym aktywatorem plazminogenu (tPA). W grupie leczonej trombolitycznie wystąpiło 9 zgonów – śmiertelność 45%. Do grupy wysokiego ryzyka zgonu (*high risk*) zaliczono 53 (26,8%) chorych, w tej grupie stwierdzono 17 zgonów – śmiertelność 32%. Do grupy niewysokiego ryzyka (*non-high risk*) zaliczono 152 (74,1%) chorych i w tej grupie stwierdzono 6 zgonów – śmiertelność 3,9%. W grupie niewysokiego ryzyka wyróżniono dodatkowo 3 podgrupy: 1. umiarkowanego-zwiększonego ryzyka (IM2) – 3 zgony, śmiertelność 8,1%; 2. umiarkowanego-mniejszego ryzyka (IM1) – 3 zgony, śmiertelność 3,6%; 3. niskiego ryzyka – bez zgonów, śmiertelność 0%.

Wnioski: 1. Śmiertelność w populacji chorych z OZTP wyniosła w niniejszym rejestrze 11%. 2. U 30% chorych OZTP wystąpił pod maską ostrego zespołu wieńcowego lub omdlenia, 34% chorych miało podwyższone stężenie troponiny, 30% miało powikłania w trakcie hospitalizacji. 3. Śmiertelność w grupie chorych leczonych trombolitycznie wyniosła 45%. 4. Leczenie reperfuzyjne (trombolityczne lub chirurgiczne) w grupie wysokiego ryzyka wg aktualnych standardów otrzymało tylko 41% chorych. 5. Podwyższone stężenie troponiny u chorych z prawidłowym ciśnieniem tętniczym wiązało się z 4-krotnie większym ryzykiem zgonu 6. Nowa strategia stratyfikacji ryzyka chorych zagrożonych zgonem w trakcie epizodu OZTP zaproponowana w wytycznych ESC 2008 znakomicie koreluje z codzienną praktyką kliniczną.

Słowa kluczowe: zator tętnicy płucnej, śmiertelność, powikłania, stratyfikacja ryzyka, troponina

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