Original article

Kardiologia Polska
2011; 69, 3: 235–240
ISSN 0022–9032

How often pulmonary embolism mimics acute coronary syndrome?

Piotr Kukla[1], Robert Długopolski[2], Ewa Krupa[3], Romana Furtak[4], Ewa Mirek-Bryniarska[5], Roman Szełemej[6], Marek Jastrzębski[7], Jacek Nowak[8], Łukasz Kulak[8], Jerzy Hybel[9], Krzysztof Wrabec[6], Kalina Kawecka-Jaszcz[7], Leszek Bryniarski[7]

[1]General and Cardiology Ward, H. Klimontowicz Memorial Hospital, Gorlice, Poland
[2]Cardiology Ward, Hospital, Nowy Targ, Poland
[3]Cardiology Ward, E. Szczeklik Memorial Hospital, Tarnow, Poland
[4]General and Cardiology Ward, John Paul II Memorial Hospital, Rzeszow, Poland
[5]Cardiology Ward, J. Dietl Memorial Hospital, Kraków, Poland
[6]Cardiology Ward, A. Sokołowski Memorial Specialist Hospital, Walbrzych, Poland
[7]1st Department of Cardiology and Hypertension, University Hospital, Krakow, Poland
[8]Cardiology Ward, City Hospital, Chrzanow, Poland
[9]General and Cardiology Ward, Nowy Sacz, Poland

Abstract

Background: The clinical picture of acute pulmonary embolism (APE) is often uncharacteristic and may mimic acute coronary syndrome (ACS) or lung diseases, leading to misdiagnosis. In 50% of patients, APE is accompanied by chest pain and in 30–50% of the patients markers of myocardial injury are elevated.

Aim: To perform a retrospective assessment of how often clinical manifestations and investigations (ECG findings and elevated markers of myocardial injury) in patients with APE may be suggestive of ACS.

Methods: We included 292 consecutive patients (109 men and 183 women) from 17 to 89 years of age (mean age 65.4 ± ± 15.5 years) with APE diagnosed according the ESC guidelines.

Results: Among the 292 patients included in the study 33 patients died during hospitalisation (mortality rate 11.3%) and 73 (25.0%) patients developed complications. A total of 75 (25.7%) patients were classified as high risk according to the ESC risk stratification, 163 (55.8%) as intermediate risk and 54 (18.5%) as low risk. Chest pain on and/or before admission was reported by 128 (43.8%) patients, including 73 (57.0%) patients with chest pain of coronary origin, 52 (40.6%) patients with chest pain of pleural origin and 3 patients with pain of undeterminable origin based on the available documentation. A total of 56 (19.2%) patients had a history of ischaemic heart disease and 5 (1.7%) had a history of myocardial infarction. A total of 8 (2.7%) patients were admitted with the initial diagnosis of ACS. The high-risk group consisted of 15 (20.6%) patients with a typical retrosternal chest pain and 60 (27.3%) patients without the typical anginal pain. Elevated troponin was observed in 103 (35.3%) patients. The ECG changes suggestive of myocardial ischaemia (inverted T waves, ST-segment depression or elevation) were observed in 208 (71.2%) patients. The following findings were significantly more common in high-risk *versus* non-high-risk patients: ST-segment depression in V4–V6 (42.6% *vs* 23.9%, p = 0.02), ST-segment elevation in V1 (46.7% *vs* 20.0%, p = 0.0002) and aVR (70.7% *vs* 40.1%, p = 0.0007).

Conclusions: One third of patients with APE may present with all the manifestations (pain, elevated troponin and ECG changes) suggestive of ACS. The ECG changes suggestive of myocardial ischaemia are observed in 70% of the patients with ST-segment depression in V4–V6 and ST-segment elevation in V1 and aVR being significantly more common in high-risk *vs* non-high-risk patients.

Key words: acute pulmonary embolism, acute coronary syndrome, ECG

Kardiol Pol 2011; 69, 3: 235–240

Address for correspondence:
Piotr Kukla, MD, PhD, General and Cardiology Ward, H. Klimontowicz Memorial Hospital, ul. Węgierska 21, 38–300 Gorlice, Poland, tel: +48 18 35 53 415, e-mail: kukla_piotr@poczta.onet.pl
Received: 27.07.2010 **Accepted:** 06.12.2010

INTRODUCTION

Acute pulmonary embolism (APE) continues to be one of the leading causes of cardiovascular deaths. The mortality rate in APE is high, up to 15% in the high-risk group [1]. Initiation of appropriate treatment is of paramount importance, as it reduces mortality to 2–8% [2]. This, however, depends on the correct diagnosis. The clinical picture of APE is often uncharacteristic and may mimic acute coronary syndrome (ACS), syncope, lung diseases or including infections. Observational studies have shown that in nearly 50% of the patients APE is accompanied by chest pain and in 30–50% of the patients markers of myocardial injury are elevated [3, 4]. The ECG in APE patients very often reveals changes typical of myocardial ischaemia [5]. Autopsy studies have shown that APE is correctly diagnosed in a mere 20% of the patients [6, 7]. Clinical manifestations, elevated troponin and ECG abnormalities may all suggest ACS, leading to misdiagnosis, mistreatment and poor outcomes in this group of patients.

The aim of our study was to retrospectively determine how often clinical manifestations and investigations (ECG and elevated levels of myocardial injury markers) in patients with APE may be suggestive of ACS.

METHODS

Study population

We included in our retrospective study a total of 292 consecutive patients (109 men and 183 women) from 17 to 89 years of age (mean age 65.4 ± 15.5 years) hospitalised at 9 cardiology wards between 2005 and 2007, discharged with the final diagnosis of APE. The mean duration of hospitalisation was 15 days (range 1–46 days). Table 1 summarises the clinical characteristics of the patients.

Table 1. Demographic and clinical characteristics of patients with acute pulmonary embolism

Age [years]	65.4 ± 15.5
Male-to-female ratio	109/183 (37.3%/62.7%)
Chest pain:	128 (43.8%)
of coronary origin	73 (57.0%)
of pleural origin	52 (40.6%)
of indeterminate origin	3 (2.4%)
Syncope	87 (29.8%)
Obesity	92 (31.5%)
Immobilisation	85 (29.1%)
Lower limb thrombophlebitis	136 (46.6%)
Haemoptysis	10 (3.4%)
Cancer	22 (7.5%)
HRT/hormonal contraception	8 (2.7%)
NYHA class III/IV CHF	36 (12.3%)
Chronic obstructive pulmonary disease	24 (8.2%)
Low-grade fever for diagnosis	36 (12.2%)
Duration of hospitalisation [days]	15,08

CHF — chronic heart failure; HRT — hormone replacement therapy; NYHA — New York Heart Association

Table 2. Investigations performed to detect acute pulmonary embolism

Computed tomography	252 (86.3%)
Echocardiography:	24 (8.2%)
Right ventricular dysfunction	16
Embolic material/thrombi	8
Venous Doppler ultrasound	9 (3.0%)
Scintigraphy	5 (1.7%)
Autopsy	2 (0.7%)

Diagnosis of pulmonary embolism

The diagnosis of PE was made on the basis of the diagnostic evaluations provided in Table 2, in accordance with the current European Society of Cardiology (ESC) guidelines [1]. Multislice spiral computed tomography was the most commonly used diagnostic modality in 7 out of the 9 study sites, while 2 sites most commonly utilised single-slice spiral computed tomography. In 16 patients with cardiogenic shock and/or significant hypotension (the high-risk group according to the ESC risk stratification) the diagnosis of APE was made on the basis of echocardiographic signs of right ventricular dysfunction.

ECG analysis

We evaluated the first available 12-lead ECG obtained because of the signs and symptoms that prompted hospitalisation (ECG recorded at paper speeds of 25 mm/s or 50 mm/s, calibrated at 10 mm/mV). The ECG were reviewed for inverted T waves in III and aVF; inverted T waves in V2–V4; ST-segment depression in V4–V6; ST-segment elevation in aVR, III, V1 and V2–V4. The ST-segment depression or elevation was defined as an upward or downward excursion of the J point by at least 1 mm, respectively. The ST-segment depression was considered significant if it was present in at least two of the leads V4–V6. A T wave was considered inverted if its amplitude was at least 1 mm. Patients with left bundle branch block or a pacemaker were not included in the study.

Markers of myocardial injury

Troponin T or troponin I were determined, depending on the study site. The following (manufacturer) kits were used for troponin determinations (the cut-off values for positive troponin are given in brackets): troponin T (cut-off value 0.03 μg/L) measured by an electro-chemiluminescence

immunoassay (ECLIA; Roche Diagnostics), troponin I (cut-off value 0.4 μg/L) measured by ECLIA; Roche Diagnostics, troponin I (cut-off value < 0.1 μg/L; Abbott), troponin I (cut-off value < 0.1 μg/L) by enzyme immunoassay (bioMérieux).

Statistical analysis

Continuous variables with a normal distribution are presented as mean ± SD. Qualitative variables were compared using the χ^2 test (with Yates correction in cases of small sizes). A p value < 0.05 (two-sided) was considered significant. The statistical calculations were performed using Statistica PL v6.1 (StatSoft, Inc.).

RESULTS

Out of the 292 patients included in the study, 33 patients died in hospital (mortality rate 11.3%), including 26 (8.9%) deaths directly attributable to PE. In-hospital complications were observed in 73 (25.0%) patients. A total of 75 (25.7%) patients were classified as high risk according to the ESC risk stratification, 163 (55.8%) as intermediate risk and 54 (18.5%) as low risk. All-cause mortality in the high-, intermediate- and low-risk groups was 32.0%, 5.5% and 0.0%, respectively, while mortality directly attributable to PE was 29.3%, 3.0% and 0.0%, respectively.

Chest pain

Chest pain on and/or before admission was reported by 128 (43.8%) patients. Based on the available documentation we found that chest pain was most commonly of coronary origin, less frequently of pleural origin and least frequently of undeterminable origin (Table 1).

In the entire study population, 56 (19.2%) patients had a history of coronary artery disease (CAD) and 5 (1.7%) had a history of myocardial infarction. A total of 8 (2.7%) patients were admitted with the initial diagnosis of ACS and received dual antiplatelet therapy and low-molecular-weight heparin, 2 patients underwent urgent coronary angiography and 1 patient died during the 30-day follow-up. Ten (13.7%) patients with a typical chest pain of coronary origin and 11 (20.0%) patients with chest pain of non-coronary origin had a history of CAD. Thirty-six (21.9%) patients without chest pain had a history of CAD. The high-risk group included 15 (20.6%) patients with a typical retrosternal pain and 60 (27.3%) patients without a typical chest pain of coronary origin.

In 85 (66.4%) patients with chest pain ischaemic changes in ECG were observed. The most common ischaemic changes in ECG observed in patients with chest pain included: inverted T waves in III and aVF (56 [43.8%] patients) and inverted T waves in V2–V4 (48 [37.5%] patients). Elevated troponin was found in 44 (34.8%) patients with chest pain.

Table 3. The incidence of ECG changes suggestive of myocardial ischaemia in patients with acute pulmonary embolism

T wave inversion in III and aVF	142 (48.6%)
T wave inversion in V2–V4	122 (41.8%)
ST-segment depression in V4–V6	77 (26.4%)
ST-segment elevation in V1	74 (25.3%)
ST-segment elevation in III	39 (13.3%)

ECG changes suggestive of myocardial ischaemia

The ECG changes suggestive of myocardial ischaemia, namely inverted T waves in III, aVF and V2–V4, ST-segment depression or elevation in III, V1, V2–V4, were observed in 208 (71.2%) patients. The incidence of ECG changes typical for ischaemia is summarised in Table 3.

The most common ECG abnormalities were inverted T waves in III and aVF, inverted T waves in V2–V4 and ST-segment depression in V4–V6. Simultaneous presence of inverted T waves in III, aVF and V2–V4 was observed in 80 (27.4%) patients (Table 3). The right bundle branch block was observed in 35 (11.9%) patients.

In the high-risk group of patients the following were significantly more common than in the non-high risk group: ST-segment depression in V4–V6, ST-segment elevation in V1 and aVR (Table 4).

Markers of myocardial injury: troponin

Troponin was measured in 224 (76.7%) patients and was elevated in 103 (45.9%). In the subgroup of patients in whom troponin was measured on admission, all the three symptoms suggestive of ACS, namely chest pain, ischaemic changes in ECG and elevated troponin, were observed in 38 (16.9%) patients.

Table 4. Ischaemic ECG changes by risk subgroup

ECG change	High-risk	Non-high-risk	P
T wave inversion in V2–V4	43 (57.3%)	101 (38.9%)	NS
ST-segment depression in V4–V6	32 (42.6%)	62 (23.9%)	0.02
ST-segment elevation in III	16 (21.3%)	29 (11.1%)	NS
ST-segment elevation in V1	35 (46.7%)	52 (20.0%)	0.0002
ST-segment elevation in aVR	53 (70.7%)	104 (40.1%)	0.0007

In 44 (19.6%) patients, both chest pain and elevated troponin level were present. In 90 (40.2%) patients both elevated troponin and ischaemic ECG changes were present. Thirteen (12.6%) patients with elevated troponin level did not have any ischaemic changes on ECG. In the 103 patients with elevated troponin, the most common ischaemic ECG changes were inverted T waves in III and aVF (63 [61.1%] patients) and inverted T waves in V2–V4 (58 [56.3%] patients).

DISCUSSION

The course of APE is characterised by a plethora of symptoms and may be extremely insidious. The principal clinical manifestations of APE include dyspnoea (80% of the patients) and chest pain (64%) [8, 9]. Electrocardiogram is one of the first examinations that are performed in patients hospitalised for chest pain or dyspnoea. Łabyk et al. [10] found that the diagnostic criteria of ACS (at least 2 of the following: chest pain, signs of myocardial ischaemia on ECG and elevated troponin) were observed in 36% of the patients. They found elevated troponin level in 68% of the patients with chest pain and inverted T waves on ECG [10]. It should be noted that Łabyk et al. [10] analysed ischaemic ECG changes without taking ST-segment elevation into account. Kostrubiec et al. [11] showed that inverted T waves and ST-segment depression are significantly more common in patients with APE and elevated troponin concentration than in patients with normal troponin levels (97% *vs* 75%).

Electrocardiogram in patients with APE very often mimics myocardial infarction of the inferior wall with persistent ST-segment elevation (the so-called inferior wall pseudoinfarction). In addition to ST-segment elevation in III, there is also ST-segment elevation in V1. This results from the fact that lead V1 (V2) reflects processes occurring in the anterior part of the right ventricle, while the lead III overlies the inferior part of the right ventricle [12]. In our study, 13% of patients had ST-segment elevation in III.

The ST-segment elevation in V1–V4 can also be observed in APE [13–15]. We detected ST-segment elevation in V1 in 24% of our patients. Kucher et al. [16] found ST-segment elevation in V1 in 20% of the patients.

Ferrari et al. [17] observed inverted T waves in precordial leads in 68% of the patients with APE, while Geibel et al. [18] found that 45% of the patients had inverted T waves in V2–V3 and 35% in V4–V6. Punukollu et al. [19] observed inverted T waves in V1–V3 in 43% of the patients. We found inverted T waves in V1–V3 in 41% of our patients.

Geibel et al. [18] found ST-segment depression in I, II and V4–V6 in 39% of the patients, while we observed ST-segment depression in V4–V6 in 26% of our patients, similarly to Kaczyńska et al. (24%) [20]. These changes were significantly more common in the subgroup of patients with elevated troponin T *vs* patients without elevated enzymatic markers of myocardial injury (41.4% *vs* 0.0%, $p = 0.004$) [20]. The ST-segment depression was also significantly more commonly associated with death and in-hospital complications [20].

Our aim was to draw the physician's attention to cases of APE that mimic ACS, most commonly in the form of non-ST-elevation myocardial infarction (NSTEMI) [21, 22] and very rarely in the form of ST-elevation myocardial infarction [23, 24]. Such cases of APE are often associated with poor outcomes despite correct early diagnosis. Examples include patients with clinical symptoms (chest pain), ECG signs (inverted T waves in leads overlying the inferior wall, the anterior wall or the inferior and anterior wall) and biochemical manifestations (elevated troponin), typical of ACS. In our study, as many as 71% of the patients had ischaemic ECG abnormalities on admission (inverted T waves in leads overlying the inferior wall, inverted T waves in V2–V4, ST-segment depression, ST-segment elevation) suggestive of ACS. Nearly 44% of the patients on admission were experiencing chest pain or had a history of chest pain with chest pain of coronary origin being identified in 57% of them. Troponin was elevated in 35% of the patients. This combination of signs and symptoms results in a very frequent diagnosis of ACS, most commonly NSTEMI, and in referring patients for invasive coronary evaluation. Initial treatment with low-molecular-weight or unfractionated heparin improves their clinical condition. Coronary angiography shows normal coronary vessels or insignificant atherosclerotic lesions, and such patients, given the initial and subsequently confirmed diagnosis of ACS, fail to receive antithrombotic therapy (as they should if they had a diagnosis of PE), and instead they are put on antiplatelet treatment (as is appropriate in ACS patients) [25]. This misdiagnosis and mistreatment may, in the worst-case scenario, lead to sudden death, which is why every patient with an initial diagnosis of ACS and normal (or near-normal) angiographic picture of the coronary vessels should undergo a thorough diagnostic evaluation for APE.

CONCLUSIONS

1. One third of patients with APE may present with all the manifestations (pain, elevated troponin and ECG changes) suggestive of ACS.
2. The ECG changes suggestive of myocardial ischaemia are observed in 70% of the patients.
3. ST-segment depression in V4–V6 and ST-segment elevation in V1 and aVR are significantly more common in high-risk *vs* non-high-risk patients.

Conflict of interest: *non declared*

References

1. Torbicki A, Perrier A, Konstantinides S et al. Guidelines and management of acute pulmonary embolism of the European Society of Cardiology. Eur Heart J, 2008; 29: 2276–2315.
2. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet, 1999; 353: 1386––1389.
3. Konstantinides S, Geibel A, Olschewski M et al. Importance of cardiac troponins I and T in risk stratification of patients with acute pulmonary embolism. Circulation, 2002; 106: 1263–1268.
4. Giannitis E, Muller-Bardorff M, Kurowski V et al. Independent prognostic value of cardiac troponin T in patients with confirmed pulmonary embolism. Circulation, 2000; 102: 211––217.
5. Kukla P, Jastrzębski M, Bryniarski L. Elektrokardiogram w podejmowaniu decyzji na ostrym dyżurze: ostry zator tętnicy płucnej czy ostry zespół wieńcowy? Pol Przegl Kardiol, 2009; 11: 127––132.
6. Gillum RF. Pulmonary embolism and thrombophlebitis in the United States, 1970–1985. Am Heart J, 1987; 114: 1262–1264.
7. Karwinski B, Svendesn E. Comparison of clinical and postmortem diagnosis of pulmonary embolism. J Clin Pathol, 1989; 42: 135–139.
8. Palla A, Petruzzelli S, Donnamaria V et al. The role of suspicion in the diagnosis of pulmonary embolism. Chest, 1995; 107: 21S–24S.
9. Miniati M, Prediletto R, Formichi B et al. Accuracy of clinical assessment in the diagnosis of pulmonary embolism. Am J Respir Crit Care Med, 1999; 159: 864–871.
10. Łabyk A, Kalbarczyk A, Piaszczyk A et al. Zatorowość płucna — trudny problem diagnostyczny. Pol Arch Med Wew, 2007; 117: 68–72.
11. Kostrubiec M, Jankowski K, Pedowska-Włoszek J et al. Signs of myocardial ischemia on electrocardiogram correlate with elevated plasma cardiac troponin and right ventricular systolic dysfunction in acute pulmonary embolism. Cardiol J, 2010; 17: 157–162.
12. Kosuge M, Kimura K, Ishikawa T et al. Electrocardiographic differentiation between acute pulmonary embolism and acute coronary syndromes on the basis of negative T waves. Am J Cardiol, 2007; 99: 817–821.
13. Wilson GT, Schaller FA. Pulmonary embolism mimicking anteroseptal acute myocardial infarction. J Am Osteopath Assoc, 2008; 108: 344–349.
14. Yeh KH, Chang HC. Massive pulmonary embolism with anterolateral ST-segment elevation : electrocardiogram limitations and the role of echocardiogram. Am J Emerg Med, 2008; 26: 632e1–3.
15. Jastrzębski M. Czy obraz elektrokardiograficzny w zatorze tętnicy płucnej wynika z niedokrwienia prawej komory? Kardiol Pol, 2008; 66: 899–901.
16. Kucher N, Walpoth N, Wustmann K et al. QR in V1: an ECG sign associated with right ventricular strain and adverse clinical outcome in pulmonary embolism. Eur Heart J, 2003; 24: 1113–1119.
17. Ferrari E, Imbert A, Chevalier T et al. The ECG in pulmonary embolism. Predictive value of negative T waves in precordial leads: 80 case reports. Chest, 1997; 111: 537–543.
18. Geibel A, Zehender M, Kasper W et al. Prognostic value of the ECG on admission in patients with acute pulmonary embolism. Eur Respir J, 2005; 25: 843–848.
19. Punukollu G, Gowda RM, Vasavada BC et al. Role of electrocardiography in identifying right ventricular dysfunction in acute pulmonary embolism. Am J Cardiol, 2005; 96: 450––452.
20. Kaczyńska A, Bochowicz A, Kostrubiec M et al. Electrocardiography and prediction of myocardial damage in patients with acute pulmonary embolism. Pol Arch Med Wew, 2004; 112: 1039–1046.
21. Lewczuk J, Guziewicz M, Piszko P et al. Zatorowość płucna fałszywie rozpoznawana i leczona jako choroba niedokrwienna serca. Konsekwencje kliniczne. Kardiol Pol, 2000; 52: 467––471.
22. Kosuge M, Kimura K, Ishikawa T et al. Prognostic significance of inverted T waves in patients with acute pulmonary embolism. Circ J, 2006; 70: 750–755.
23. Ludwin B, Lewczuk J, Piszko B. Prawidłowy wynik koronarografii u pacjenta z rozpoznaniem ostrego zespołu wieńcowego z uniesieniem odcinka ST zakwalifikowanego do przezskórnej interwencji wieńcowej. Czy można było wcześniej rozpoznać ostrą zatorowość płucną? Kardiol Pol, 2006; 64: 68–71.
24. Latacz P, Rostoff P, Wyderka R et al. Masywny zator tętnicy płucnej imitujący ostry zespół wieńcowy z uniesieniem odcinka ST skutecznie leczony terapią hybrydową u chorej po złożonym urazie ortopedycznym otrzymującej nadroparynę. Kardiol Pol, 2007; 65: 1235–1242.
25. Bassand JP, Adrissino D, Budaj A et al. ESC guidelines for the management of non-ST segment elevation acute coronary syndromes. Eur Heart J, 2007; 28: 1598–1660.

Jak często zator tętnicy płucnej może imitować ostry zespół wieńcowy?

Piotr Kukla[1], Robert Długopolski[2], Ewa Krupa[3], Romana Furtak[4], Ewa Mirek-Bryniarska[5], Roman Szełemej[6], Marek Jastrzębski[7], Jacek Nowak[8], Łukasz Kulak[8], Jerzy Hybel[9], Krzysztof Wrabec[6], Kalina Kawecka-Jaszcz[7], Leszek Bryniarski[7]

[1]Oddział Internistyczno-Kardiologiczny, Szpital im. H. Klimontowicza, Gorlice
[2]Oddział Kardiologii, Szpital, Nowy Targ
[3]Oddział Kardiologii, Szpital im. E. Szczeklika, Tarnów
[4]Oddział Kardiologii i Chorób Wewnętrznych, Szpital im. Jana Pawła II, Rzeszów
[5]Oddział Kardiologii, Szpital im. J. Dietla, Kraków
[6]Oddział Kardiologii, Specjalistyczny Szpital im. A. Sokołowskiego, Wałbrzych
[7]I Klinika Kardiologii i Nadciśnienia Tętniczego, Szpital Uniwersytecki, Kraków
[8]Oddział Kardiologii, Szpital Miejski, Chrzanów
[9]Oddział Internistyczno-Kardiologiczny, Nowy Sącz

Streszczenie

Wstęp: Obraz kliniczny ostrego zatoru tętnicy płucnej (OZTP) jest często niecharakterystyczny i może występować pod maską ostrego zespołu wieńcowego (OZW) czy chorób płuc, co prowadzi do pomyłek diagnostycznych. U 50% chorych OZTP przebiega z bólem w klatce piersiowej, a u 30–50% osób obserwuje się podwyższenie markerów uszkodzenia miokardium.

Cel: Celem pracy było retrospektywne określenie, jak często objawy kliniczne i badania dodatkowe (elektrokardiogram i podwyższone stężenie markerów martwicy mięśnia sercowego) u chorych z OZTP mogą sugerować rozpoznanie OZW.

Metody: Do badania włączono 292 kolejnych chorych (183 kobiety, 109 mężczyzn) w wieku 17–89 lat (średni wiek 65,4 ± ± 15,5 roku) z rozpoznaniem OZTP.

Wyniki: W grupie 292 chorych w trakcie hospitalizacji wystąpiły 33 zgony (śmiertelność 11,3%), a u 73 (25%) pacjentów zaobserwowano powikłania. Do grupy wysokiego ryzyka wg stratyfikacji ryzyka ESC zaliczono 75 (25,7%) chorych, do grupy umiarkowanego ryzyka — 163 (55,8%) osób, a do grupy niskiego ryzyka — 54 (18,5%) pacjentów. Ból w klatce piersiowej przy przyjęciu i/lub przed przyjęciem do szpitala podawało 128 (43,8%) chorych, w tym u 73 (57,0%) osób ból miał charakter wieńcowy, u 52 (40,6%) — opłucnowy, u 3 — nieokreślony na podstawie dostępnej dokumentacji. W całej badanej grupie 56 (19,2%) osób miało wywiad choroby niedokrwiennej serca, a 5 (1,7%) chorych przebyło zawał serca. Ze wstępnym rozpoznaniem OZW przyjęto 8 (2,7%) chorych. Do grupy wysokiego ryzyka należało 15 (20,6%) osób z typowym bólem zamostkowym oraz 60 (27,3%) pacjentów bez typowego bólu wieńcowego. U 103 (35,3%) chorych stwierdzono podwyższone stężenie troponiny. Zmiany elektrokardiograficzne sugerujące niedokrwienie mięśnia sercowego (ujemne załamki T, obniżenie odcinka ST lub uniesienie odcinka ST) zaobserwowano u 208 (71,2%) chorych. W grupie pacjentów wysokiego ryzyka znamiennie częściej stwierdzano: obniżenie odcinka ST w odprowadzeniach V4–V6 (42,6% *v.* 23,9%; p = 0,02), uniesienie odcinka ST w odprowadzeniu V1 (46,7% *v.* 20%; p = 0,0002) i aVR (70,7% *v.* 40,1%; p = 0,0007) w porównaniu z chorymi z grupy niewysokiego ryzyka.

Wnioski: U 1/3 chorych z OZTP mogą występować wszystkie objawy (ból, podwyższone stężenie troponiny i zmiany elektrokardiograficzne) sugerujące rozpoznanie OZW. Zmiany elektrokardiograficzne sugerujące niedokrwienie mięśnia sercowego występują u 70% pacjentów. W grupie osób wysokiego ryzyka znamiennie częściej obserwowano obniżenie odcinka ST w odprowadzeniach V4–V6 oraz uniesienie odcinka ST w odprowadzeniach V1 i aVR.

Słowa kluczowe: ostry zator tętnicy płucnej, ostry zespół wieńcowy, EKG

Kardiol Pol 2011; 69, 3: 235–240

Adres do korespondencji:
dr n. med. Piotr Kukla, Oddział Internistyczno-Kardiologiczny, Szpital Specjalistyczny im. H. Klimontowicza, ul. Węgierska 21, 38–300 Gorlice, tel: +48 18 35 53 415, e-mail: kukla_piotr@poczta.onet.pl
Praca wpłynęła: 27.07.2010 r. **Zaakceptowana do druku:** 06.12.2010 r.