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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Acute pulmonary embolism treatment with rivaroxaban results in a shorter duration of hospitalisation compared to standard therapy: an academic centre experience

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Abstract

Background: Depending on the severity of clinical condition, acute pulmonary embolism (APE) is treated with unfractionated heparin (UFH), low-molecular weight heparin (LMWH), oral anticoagulants or, in the most severe form, with fibrinolytic agents. Following APE, patients require prolonged anticoagulant therapy for 3–6 months or in some cases indefinitely. Treatment options in this period include vitamin K antagonists (VKA) and non-VKA oral anticoagulants (NOAC) including rivaroxaban. The most recent European Society of Cardiology guidelines on the diagnosis and management of APE recommend use of NOAC in patients at a low-to-moderate risk of early mortality (a class I B recommendation). Rivaroxaban may be used in haemodynamically stable patients since the first day of therapy and was approved for this indication in Poland in December 2012.

Aim: To evaluate the rate of rivaroxaban use, characterise patients with APE treated with rivaroxaban, and evaluate potential reduction of the duration of hospitalisation in patients treated with rivaroxaban compared to those receiving VKA.

Methods: We evaluated hospital and postdischarge treatment in 215 consecutive APE patients (105 men, 110 women) at the mean age of 65.0 (range: 19.5–91.9) years. The study included patients hospitalised from January 2013 to November 2014, i.e. in the period immediately following approval of rivaroxaban for the treatment of APE in Poland. In the acute phase, patients were treated with LMWH, UFH, or rivaroxaban, and the treatment was continued with VKA, LMWH, or rivaroxaban. The timing of initiation of oral therapy depended on the haemodynamic stability of the patient.

Results: Our study group of 215 APE patients included 157 (73%) moderate-risk patients, 51 (24%) low-risk, and 7 (3.3%) high-risk patients. Treatment was initiated with UFH or LMWH in 208 (96.7%) patients, and with rivaroxaban in 7 (3.3%) patients. In 33 (16.5%) patients, rivaroxaban was started after up to 3 days of heparin therapy. Chronic therapy prescribed at discharge included VKA in 64 (30.5%) patients, rivaroxaban in 82 (39%) patients, and LMWH in 64 (30.5%) patients. Five patients died during hospital, for the total mortality of 2.3%. Acute high-risk PE was diagnosed on admission in 2 of these patients, and moderate-risk PE in 3 patients. Treatment in this group included enoxaparin in 4 patients and UFH in 1 patient. Patients who were discharged on rivaroxaban stayed in hospital for a significantly shorter time compared to patients discharged on VKA (6 [2–22] vs. 8 [2–17] days, $p = 0.0005$). Duration of hospital stay was significantly shorter in APE patients with sPESI of 0 who were treated with rivaroxaban compared to those with sPESI of 0 treated with VKA (5 [2–11] vs. 6 [2–12] days, $p = 0.002$). A significant difference in the duration of hospital stay was also noted in patients with sPESI of ≥ 1 treated with rivaroxaban compared to those treated with VKA (7 [3–22] vs. 9 [3–17] days, $p = 0.015$). Patients with sPESI of ≥ 1 treated with rivaroxaban were hospitalised for a significantly longer time compared to those with sPESI of 0 treated with rivaroxaban (7 [3–22] vs. 5 [2–11] days, $p = 0.00005$).

Conclusions: Rivaroxaban therapy is a useful therapeutic option in patients with APE. Compared to standard therapy, use of rivaroxaban has been associated with a significant reduction of the duration of hospital stay.

Key words: acute pulmonary embolism, rivaroxaban, vitamin K antagonists, risk stratification

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INTRODUCTION

Venous thromboembolism (VTE) is the third most common cardiovascular disease. Annual incidence is 1–2 per 1000 [1, 2]. Acute pulmonary embolism (APE) is the most dangerous clinical manifestation of VTE which may be fatal. APE patients who are not at high risk of early mortality (i.e., haemodynamically stable) have been initially treated with unfractionated heparin (UFH) or low-molecular-weight heparins (LMWH), followed by vitamin K antagonist (VKA) therapy in most patients. However, long-term VKA treatment, continued for several months, is associated with a number of inconveniences and difficulties for both patients and physicians who care for them.

Non-VKA oral anticoagulants (NOAC) are free from many limitations of VKA and are a valuable alternative treatment option in patients with APE. Rivaroxaban, a direct factor Xa inhibitor, may be used in haemodynamically stable patients from the first day of therapy. In Poland, it was approved for this indication in December 2012. Dabigatran, a direct thrombin (factor IIa) inhibitor, may be used after at least 5 days of parenteral therapy and was approved for this indication in Poland in June 2014. The most recent European Society of Cardiology (ESC) guidelines on the diagnosis and management of APE recommend use of NOAC in APE patients at a low-to-moderate risk of early mortality (a class I B recommendation for both drugs) [3].

The aim of the study was to evaluate the rate of rivaroxaban use, characterise patients with APE treated with rivaroxaban in a tertiary care academic centre, and evaluate potential reduction of the duration of hospitalisation in patients treated with rivaroxaban compared to those receiving VKA.

METHODS

The study included 215 consecutive patients (105 men, 110 women) at the mean age of 65.0 (range 19.5–91.9) years with APE diagnosed and treated in a tertiary care academic centre. We included patients hospitalised from January 2013 to November 2014, i.e. in the period immediately following approval of rivaroxaban for the treatment of APE in Poland. The diagnosis of APE was confirmed by multislice computed tomography (Toshiba Aquilion) which showed the presence of thrombi in at least segmental pulmonary arteries. Acute high-risk pulmonary embolism (PE) was defined as shock or hypotension due to APE on admission (systolic blood pressure < 90 mm Hg or systolic pressure fall by ≥ 40 mm Hg for > 15 min if not due to new arrhythmia, sepsis, or hypovolemia). Acute moderate-risk PE was defined as systemic systolic blood pressure on admission ≥ 90 mm Hg with echocardiographic or computed tomography evidence of right ventricular (RV) overload and/or elevated serum levels of biomarkers reflecting RV damage/overload (troponin T, N-terminal pro-B-type natriuretic peptide [NT-proBNP]). Acute low-risk PE was diagnosed in haemodynamically stable patients without evidence of RV overload in imaging studies and with normal levels of biomarkers of RV damage/overload. In accordance with the ESC guidelines, the simplified PE severity index (sPESI) was calculated in all patients to estimate the severity of APE [3]. APE was diagnosed if symptoms were present for less than 14 days.

We excluded pregnant (n = 2) and lactating (n = 4) women, patients with an active bleeding in whom anticoagulant therapy was absolutely contraindicated (n = 3), and patients participating in clinical studies (n = 4). Detailed history was taken and physical examination was performed on admission in all patients. Within 24 h from admission, all patients underwent transthoracic echocardiography using a Philips iE 33 system (Andover, MD, USA) with a 2.5–3.5 MHz probe. Peak tricuspid regurgitant jet velocity was measured using continuous wave Doppler in the apical 4-chamber view. Tricuspid regurgitation peak gradient was calculated using the simplified Bernoulli equation — $dP = 4v^2$, where dP is the pressure gradient between cardiac chambers in mm Hg, and v is the measured peak tricuspid regurgitant jet velocity. Tricuspid annulus plane systolic excursion was calculated using the M-mode in the apical 4-chamber view.

Serum NT-proBNP level was measured by high-sensitive electrochemiluminescence assay with streptavidin-coated magnetic microparticles using an automated immunodiagnostic analyser Elecsis 2010 (Roche Diagnostics GmbH).

Serum troponin T level was measured using electrochemiluminescence test (Troponin T hs STAT, Roche Diagnostics GmbH). Levels > 0.014 ng/mL were considered abnormal.

Study participants were treated in accordance with the ESC guidelines [3]. We used intravenous UFH guided by activated partial thromboplastin testing or subcutaneous enoxaparin or nadroparin with dosing adjusted to body weight and renal function, followed by VKA (warfarin, acenocoumarol) treatment guided by the international normalised ratio (INR). Another treatment option was rivaroxaban 15 mg bid. The timing of oral therapy initiation depended on haemodynamic stability of the patient and low likelihood of cardiovascular decompensation. Chronic rivaroxaban therapy was considered in all APE patients.

We evaluated drug treatment during hospitalisation and at discharge.

In addition, we analysed deaths and cases of severe bleeding. In accordance with the International Society on Thrombosis and Haemostasis (ISTH) guidelines, severe bleeding was defined as bleeding resulting in haemoglobin level drop by at least 20 g/L or requiring transfusion of at least 2 units of packed red blood cells [4].

Statistical analysis

Normally distributed variables were reported as mean values and standard deviation, and other variables were reported as median values and range (minimum, maximum). Normal variable distribution was verified using the Shapiro-Wilk test.

Normally distributed variables were compared using the Student t test. The assumption of homogeneity of variance was verified using the Brown-Forsythe test. Non-normally distributed variables were compared using the Mann-Whitney U test. All calculations were performed using the Statistica 10 package.

RESULTS

The study group included 215 consecutive patients (105 men, 110 women) at the mean age of 65.0 (range 19.5–91.9) years with the diagnosis of APE. High-risk APE was diagnosed in 7 (3.3%) patients, moderate-risk APE in 157 (73%) patients, and low-risk APE in 51 (24%) patients.

Clinical, echocardiographic, and biochemical characteristics of the study group are shown in Table 1. Initial treatment included heparin in 208 (96.7%) patients (UFH in 28 patients and LMWH in 180 patients) and rivaroxaban started on the first day of treatment in 7 (3.3%) patients. Rivaroxaban was also initiated in 33 (16.5%) patients after up to 3 days of heparin treatment. Fibrinolytic treatment was used during the hospitalisation in 4 (1.9%) high-risk patients. In 2 high-risk patients, fibrinolysis was absolutely contraindicated due to a recent major surgery and UFH was used. Embolectomy was performed in 1 patient with a right atrial mobile thrombus.

Chronic treatment prescribed at discharge included VKA initiated during the hospitalisation in 64 (30.5%) patients, rivaroxaban in 82 (39.0%) patients, and LMWH in 64 (30.5%) patients. Indications for LMWH included an active malignancy in 33 patients, a suspicion of malignancy requiring further invasive diagnostic procedures in 12 patients, and a surgery performed recently or contemplated within next several weeks in 19 patients.

The main reasons to use VKA as a chronic treatment were patient preferences (60 patients) and stage IV or higher chronic kidney disease with estimated glomerular filtration rate < 30 mL/min/1.73 m^2 (4 patients), constituting a contraindication to rivaroxaban treatment.

Five deaths (2.3% patients) occurred during the hospitalisation, including 2 high-risk patients and 3 moderate-risk patients. Four of these patients were treated with enoxaparin and one was treated with UFH. The cause of death was an advanced malignancy in 4 cases, and an intractable lower respiratory tract infection in 1 case. None of these fatal cases was associated with a severe bleeding.

Figure 1 shows the management of patients with APE. Table 2 summarises the severity of PE in patients treated with VKA.

Patients who were discharged on rivaroxaban stayed in hospital for a significantly shorter time compared to patients discharged on VKA (6 [2–22] vs. 8 [2–17] days, p = 0.0005) (Fig. 2).

Duration of hospital stay was significantly shorter in APE patients with sPESI of 0 who were treated with rivaroxaban compared to those with sPESI of 0 treated with VKA (5 [2–11] vs. 6 [2–12] days, p = 0.002) (Fig. 3).

A significant difference in the duration of hospital stay was also noted in patients with sPESI of ≥ 1 treated with rivaroxaban compared to those treated with VKA (7 [3–22] vs. 9 [3–17] days, p = 0.015).

Patients with sPESI of ≥ 1 treated with rivaroxaban were hospitalised for a significantly longer time compared to those with sPESI of 0 treated with rivaroxaban (7 [3–22] vs. 5 [2–11] days, p = 0.00005).

Patients with sPESI of ≥ 1 treated with VKA were hospitalised for a significantly longer time compared to those with sPESI of 0 treated with VKA (9.9 ± 3.2 vs. 6.8 ± 2.4 days, p = 0.000037).

Three cases of a significant bleeding were noted in the study group during the hospital treatment. All these patients were treated with enoxaparin and had an increased risk of bleeding complications (1 patient due to concomitant dual antiplatelet therapy, and 2 patients with malignancies in the early postoperative period)

Table 1. Clinical, echocardiographic, and biochemical characteristics of patients with acute pulmonary embolism

Parameter	Value
Heart rate [bpm]	85.0 (53–160)
SBP [mm Hg]	130 (60–186)
eGFR [mL/min/1.73 m^2]	76.3 ± 30
Concomitant conditions:	
Chronic obstructive pulmonary disease	12 (5.6%)
Chronic heart failure	34 (15.8%)
Malignancy	32 (14.9%)
sPESI	1 (0–6)
Troponin T level [ng/mL]	0.021 (0–1.8)
Elevated troponin T level	127 (59%)
NT-proBNP level [pg/mL]	436.5 (12–35,000)
TRPG [mm Hg]	32 (9–126)
TAPSE [mm]	22 (5–70)

eGFR — estimated glomerular filtration rate; NT-proBNP — N-terminal pro-B-type natriuretic peptide; SBP — systolic blood pressure; sPESI — simplified pulmonary embolism severity index; TAPSE — tricuspid annulus plane systolic excursion; TRPG — tricuspid regurgitation peak gradient

DISCUSSION

Non-VKA oral anticoagulants have been approved for the treatment of APE, and their efficacy and safety have been proven in numerous clinical trials. In the EINSTEIN-DVT and EINSTEIN-PE studies, rivaroxaban 15 mg bid for 3 weeks followed by 20 mg once daily was used as an alternative to standard therapy. The EINSTEIN-PE study included 4832 patients, and rivaroxaban was not inferior to standard therapy

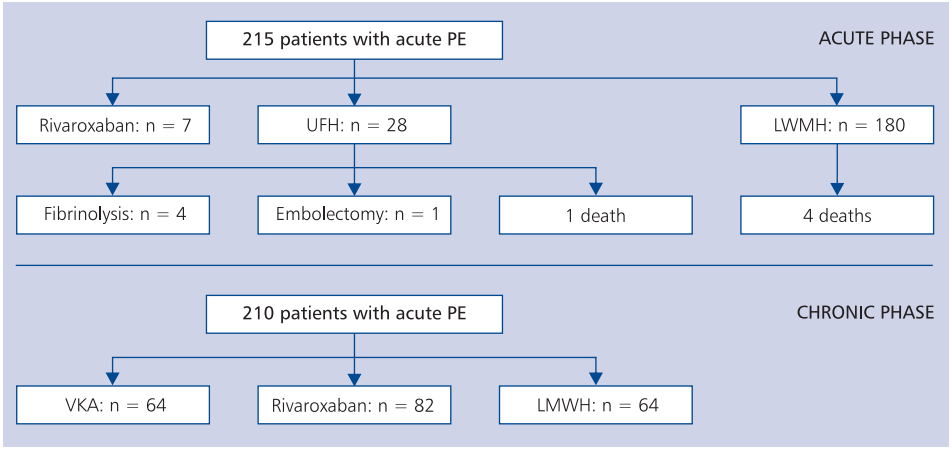

Figure 1. Acute and chronic management of patients with pulmonary embolism (PE); LWMH — low-molecular weight heparin; UFH — unfractionated heparin; VKA — vitamin K antagonist

Table 2. Severity of acute pulmonary embolism (APE) in patients treated with vitamin K antagonists (VKA) and rivaroxaban

Drug	Low-risk APE	Moderate-risk APE	P	sPESI 0	sPESI ≥ 1	P
VKA	16 (31.4%)	46 (29.9%)	0.84	37 (37%)	27 (24.5%)	0.05
Rivaroxaban	22 (43.1%)	59 (38.3%)	0.54	45 (45%)	37 (33.6%)	0.09

sPESI — simplified pulmonary embolism severity index

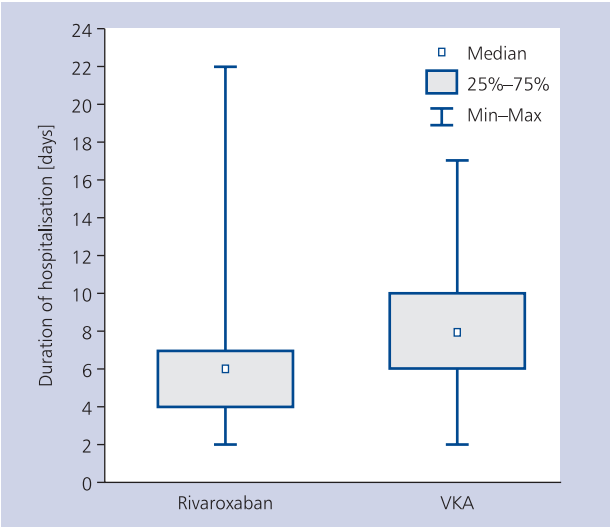

Figure 2. Comparison of the duration of hospitalisation among acute pulmonary embolism patients treated with rivaroxaban or vitamin K antagonist (VKA)

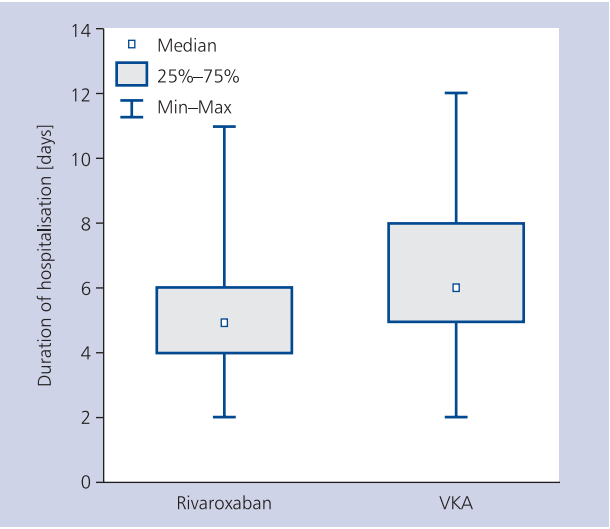

Figure 3. Comparison of the duration of hospitalisation among acute pulmonary embolism patients with simplified pulmonary embolism severity index (sPESI) = 0 treated with rivaroxaban or vitamin K antagonist (VKA)

in preventing VTE recurrences (hazard ratio [HR] 1.12, 95% confidence interval [CI] 0.75–1.68). Major or clinically significant bleeding occurred at the same rate in both groups but major bleeding was less frequent in the rivaroxaban group [5]. In the RE-COVER study, the rate of recurrent symptomatic VTE was compared between dabigatran (n = 1274) and warfarin (n = 1265) groups. Dabigatran was not inferior to warfarin in terms of the primary study endpoint occurrence (HR 1.10, 95% CI 0.65–1.84). No significant difference in the rate of major bleeding was seen, and the overall bleeding rate was lower in the dabigatran group [6]. Efficacy and safety of dabigatran was confirmed in the RECOVER II study [7].

Similarly, large clinical trials confirmed efficacy and safety of treating APE patients with apixaban and edoxaban which are less available in Poland compared to the 2 drugs discussed above.

In the AMPLIFY study, apixaban 10 mg bid for 10 days followed by 5 mg once daily was compared with the conventional therapy in 5395 patients, including 1836 with APE. Apixaban was not inferior in terms of treatment efficacy, defined as recurrent symptoms or death related to VTE (relative risk [RR] 0.84, 95% CI 0.60–1.18). Major bleeding was less frequent in the apixaban group (RR 0.31, 95% CI 0.17–0.55) [8].

The Hokusai-VTE study compared edoxaban 60 mg once daily with the conventional therapy in 8240 patients with acute VTE, including 3319 with APE. Edoxaban was not inferior to warfarin in terms of the occurrence of the primary endpoint, defined as recurrent VTE symptoms or death (HR 0.89, 95% CI 0.70–1.13). The rate of the primary safety endpoint was lower in the edoxaban group (HR 0.81, 95% CI 0.71–0.94, p = 0.004 for superiority) [9].

Current guidelines for the diagnosis and management of APE recommend NOAC as an alternative to standard therapy with parenteral anticoagulation followed by VKA (a class I B recommendation) in haemodynamically stable patients [3]. Increasing use of NOAC prompts to evaluate other aspects of this therapy in addition to its effectiveness and safety.

In the present study, we evaluated 215 patients treated due to APE in a tertiary care academic centre in 2013–2014. Most patients (208, or 96.7% of the study group) were initially treated with LMWH or UFH, and 4 (1.9%) patients required fibrinolytic treatment. Rivaroxaban was started on the first day of therapy in 7 (3.3%) patients. During the hospitalisation, VKA or rivaroxaban was initiated in patients initially treated with heparin (UFH or LMWH), with an advice to continue this therapy in the outpatient settings. At discharge, rivaroxaban was prescribed in 82 (39%) patients and VKA in 64 (30.5%) patients.

In our study group, we did not find differences in the rate of rivaroxaban use between low- and moderate-risk patients, and those with sPESI of 0 or ≥ 1. Similar results were obtained for VKA (Table 2). No deaths were noted among patients treated with rivaroxaban. These findings indicate utility of rivaroxaban also in moderate-risk APE patients, in addition to the low-risk group.

A major aim of the present study was to evaluate the duration of hospitalisation of APE patients treated with rivaroxaban or VKA. In the overall study group, patients prescribed rivaroxaban at discharge were hospitalised for a significantly shorter time compared to those treated with VKA (6 vs. 8 days, p = 0.0005). We also found that patients with sPESI of 0 who were treated with rivaroxaban were discharged after a shorter time compared to those treated with VKA (5 vs. 6 days, p = 0.002), Patients with sPESI of ≥ 1 were discharged on average after 7 days in the rivaroxaban group compared to 9 days in the VKA group (p = 0.015). Matsuo et al. [10] evaluated the duration of hospital stay of 97 patients with VTE, including 78 patients treated with rivaroxaban and 19 patients treated with UFH followed by warfarin. These authors showed a significantly shorter duration of hospitalisation among patients treated with rivaroxaban (10 [6–15] vs. 15 [9–22] days, p = 0.016). In the Polish literature, no data have been previously available on the duration of hospitalisation among APE patients treated with NOAC. In addition to shorter hospital stay which translates to economic benefits of rivaroxaban therapy, higher patient satisfaction was also shown among patients treated with this drug compared to the standard therapy. In a group of 2397 participants of the EINSTEIN PE study, Prins et al. [11] showed higher patient satisfaction in the rivaroxaban group compared to the VKA group. Importantly, reduced duration of hospitalisation is associated with lower treatment costs. In an analysis of EINSTEIN DVT and EINSTEIN PE studies, Bamber et al. [12] showed that rivaroxaban therapy was more cost-effective compared to the standard therapy.

An additional aim of the present study was to evaluate bleeding complications in APE patients treated with rivaroxaban. Our results confirm safety of rivaroxaban therapy in patients with APE. No significant bleeding complications were observed in these patients during in-hospital treatment.

Our study is the first comparison of consecutive APE patients treated with rivaroxaban vs. standard therapy in the Polish literature. Our findings require confirmation in further prospective multicentre studies.

Limitations of the study

A single centre nature of our study was a limitation but the sample of 215 patients allowed achieving the study goals. In the present study, we evaluated the utility of 1 NOAC, rivaroxaban, in the treatment of APE. Studies are needed to evaluate other NOAC in the clinical practice settings in Poland. A low number of high-risk APE patients (7 patients, or 3.3% of the study group) was also a limitation but rivaroxaban treatment is used mostly in low- and moderate risk APE patients. In addition, our study sample was too small to evaluate treatment safety.

CONCLUSIONS

Rivaroxaban therapy is a useful therapeutic option in patients with APE. Compared to standard therapy, use of rivaroxaban has been associated with a significant reduction of the duration of hospital stay.

Conflict of interest: *none declared*

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Leczenie riwaroksabanem w porównaniu ze standardową terapią powoduje skrócenie okresu hospitalizacji pacjentów z ostrą zatorowością płucną: doświadczenia ośrodka akademickiego

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Streszczenie

Wstęp: W ostrej fazie, w zależności od zaawansowania klinicznego, ostra zatorowość płucna (APE) jest leczona heparyną niefrakcjonowaną (UFH) lub drobnocząsteczkową (LMWH), antykoagulantami doustnymi, a w swej najcięższej postaci — fibrynolitycznie. Pacjenci po przebytej APE wymagają przedłużonego leczenia przeciwkrzepliwego, które należy stosować przez 3–6 miesięcy, a u niektórych pacjentów bezterminowo. Do leków stosowanych w tym okresie należą doustne antykoagulanty:

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antagoniści witaminy K (AVK) lub leki niebędące antagonistami witaminy K (NOAC), a wśród nich riwaroksaban. W najnowszych zaleceniach Europejskiego Towarzystwa Kardiologicznego dotyczących rozpoznawania i leczenia APE rekomenduje się stosowanie NOAC u chorych z grupy pośredniego i niskiego ryzyka wczesnego zgonu (klasa zaleceń I B). Riwaroksaban można stosować u stabilnych hemodynamicznie pacjentów od pierwszego dnia terapii; został on zarejestrowany w Polsce w tym wskazaniu w grudniu 2012 roku.

Cel: Celem pracy była ocena częstości stosowania riwaroksabanu i charakterystyka grupy chorych z APE leczonych tym lekiem, a także ocena potencjalnego skrócenia czasu hospitalizacji u pacjentów poddanych terapii riwaroksabanem w porównaniu z osobami otrzymującymi VKA.

Metody: Analizowano leczenie szpitalne i poszpitalne 215 kolejnych pacjentów (105 mężczyzn, 110 kobiet) w wieku 65,0 (19,5–91,9) lat z APE. U każdego chorego poza badaniem przedmiotowym wykonywano badania echokardiograficzne i laboratoryjne (stężenie biomarkerów odzwierciedlających uszkodzenie/przeciążenie prawej komory: troponina T, NT-proBNP), a także oceniano ryzyko 30-dniowego zgonu w celu oszacowania stopnia ciężkości APE (skala sPESI). Do badania włączono chorych hospitalizowanych w okresie 01.2013–11.2014, a więc bezpośrednio po zarejestrowaniu w Polsce riwaroksabanu do leczenia APE. APE wysokiego ryzyka wczesnego zgonu rozpoznawano, gdy przy przyjęciu stwierdzano wstrząs lub hipotonię spowodowaną APE. APE pośredniego ryzyka rozpoznawano wówczas, gdy systemowe ciśnienie skurczowe przy przyjęciu wynosiło ≥ 90 mm Hg oraz stwierdzano echokardiograficzne, tomograficzne lub laboratoryjne cechy przeciążenia/uszkodzenia prawej komory. APE niskiego ryzyka rozpoznawano u normotensyjnych chorych bez cech przeciążenia/uszkodzenia prawej komory. W ostrej fazie pacjenci przyjmowali UFH, LMWH lub riwaroksaban. Terapię kontynuowano za pomocą VKA, UFH lub riwaroksabanu. Czas rozpoczęcia terapii lekami doustnymi zależał od stabilności hemodynamicznej chorego.

Wyniki: W badanej grupie 215 chorych z APE u 157 (73%) pacjentów rozpoznano APE pośredniego ryzyka, u 51 (24%) niskiego ryzyka, a u 7 (3,3%) stwierdzono APE wysokiego ryzyka. 208 (96,7%) chorych leczono wstępnie UFH lub LMWH, u 7 (3,3%) od pierwszego dnia terapii włączono riwaroksaban, w 33 (16,5%) przypadkach po maksymalnie 3 dniach leczenia heparynami zastosowano riwaroksaban. W 4 (1,9%) przypadkach w trakcie hospitalizacji zastosowano leczenie fibrynolityczne. Po wypisaniu ze szpitala jako terapię przewlekłą u 64 (30.5%) pacjentów zalecono stosowanie VKA włączanych w trakcie hospitalizacji, u 82 (39%) riwaroksabanu, a u 64 (30.5%) UFH. W trakcie hospitalizacji zmarło 5 chorych, śmiertelność całkowita wynosiła 2,3%. U 2 pacjentów przy przyjęciu rozpoznano APE wysokiego ryzyka, a u 3 — APE pośredniego ryzyka. W tej grupie 4 chorych było leczonych enoksaparyną, a 1 za pomocą UFH. Przyczyną zgonów u 4 osób była zaawansowana choroba nowotworowa, a u 1 pacjentki niepoddające się leczeniu zapalenie dolnych dróg oddechowych. W przypadku żadnego ze zgonów nie stwierdzono poważnego krwawienia. Pacjenci, którzy zostali wypisani do domu z zaleceniem przyjmowania riwaroksabanu, byli hospitalizowani istotnie krócej niż chorzy, którzy otrzymali VKA (6 [2–22] *vs*. 8 [2–17] dni, p = 0,0005). W grupie chorych z APE i sPESI = 0 pkt. leczonych riwaroksabanem czas hospitalizacji był istotnie krótszy niż u badanych z sPESI = 0 pkt. leczonych VKA (5 [2–11] *vs*. 6 [2–12] dni, p = 0,002). U badanych z sPESI ≥ 1 pkt leczonych riwaroksabanem i VKA wykazano również istotną statystycznie różnicę długości trwania hospitalizacji (7 [3–22] *vs*. 9 [3–17] dni, p = 0,015). Stwierdzono istotnie dłuższy czas hospitalizacji pacjentów z sPESI ≥ 1 pkt leczonych riwaroksabanem w porównaniu z badanymi z sPESI = 0 pkt. leczonych tym lekiem (7 [3–22] *vs*. 5 [2–11] dni, p = 0,00005). Ponadto w badanej grupie chorych zaobserwowano 3 przypadki poważnych krwawień. Pacjenci z tej grupy stosowali leczenie enoksaparyną.

Wnioski: Leczenie riwaroksabanem jest przydatną opcją terapeutyczną u chorych z APE. W porównaniu ze standardową terapią zastosowanie riwaroksabanu wiąże się z istotnym skróceniem okresu hospitalizacji.

Słowa kluczowe: ostra zatorowość płucna, riwaroksaban, antagoniści witaminy K, stratyfikacja ryzyka

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