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Symptomatic artery disease as a predictor of in-hospital and 6-month prognosis in patients with acute pulmonary embolism

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Abstract

Background: The association of previous arterial disease with outcome of patients with pulmonary embolism (PE) is not well considered.

Aim: To investigate association between the positive history of symptomatic arterial disease and in-hospital and 6 -month outcome in patients with acute PE.

Methods: Among 237 consecutive patients with PE admitted in the intensive care unit of university hospital during 6 years, 40 patients had positive history of arterial disease (24 had coronary disease – myocardial infarction or revascularization, 15 had stroke and 1 of them had peripheral arterial disease). In-hospital and six-month mortality was compared between patients with and without previous symptomatic arterial disease. The six-month major bleeding events were also compared between groups.

Results: In-hospital and six-month mortality was significantly higher in patients with positive history of arterial disease (30.0 % vs 9.1 %, p=0.001 and 35.0 % vs 13.7 %, p=0.02, respectively). Hazard ratios for in-hospital and six-month mortality adjusted to age and gender were significantly higher in patients with positive history of arterial disease (HR=3.668 95 %CI 1.766-7.618, p<0.001, and HR=2.948, 95 %CI 1.545-5.626, p=0.001). Positive history of arterial disease was also associated with increased risk of major bleeding (22.5 % vs 11.5 %, p=0.078) during 6 months follow-up (HR=2.230 95 %CI 1.031-4.823, p=0.042).

Conclusion: The positive history of symptomatic arterial disease is associated with increased risk for in-hospital and six-month mortality and major bleeding in PE patients.

Key words pulmonary embolism, symptomatic arterial disease, prognosis, bleeding

Introduction

The incidence of venous thrombo-embolism (VTE), comprising deep vein thrombosis (DVT) and pulmonary embolism (PE), increases exponentially with advancing age.[1] Co-existence of concomitant diseases and use of co-medications, impaired hepatic or renal function, and changes in pharmacokinetic and pharmacodynamic profiles make VTE patients especially vulnerable.[2]

Intermediate and long-term survival in VTE patients is poor, and the elderly are especially prone to bleeding complications from anticoagulation or reperfusion treatment. It is unknown whether differences in VTE severity exist between the patients with or without symptomatic arterial disease (they are more frequently present in elderly patients), and whether its presence impacts on in-hospital or 6-month clinical outcomes.[3-7]

In few heart diseases left-side cardiac thrombosis is frequent and could lead to arterial embolism. Similar mechanism may be responsible for right-side cardiac thrombosis and consequently be a direct source of pulmonary embolism. Yasuoka et al. showed a higher incidence of perfusion defects in lung scan in patients with spontaneous echocontrast in the right atrium than in those without it (40% vs.7 %).[8] Pesavento et al. assessed the prevalence of heart diseases in 11.236 consecutive patients older than 60 years discharged from hospitals with a diagnosis of pulmonary embolism. They found a higher prevalence of all-cause heart diseases (OR 1.26; 95 % CI, 1.13–1.40) in patients with a diagnosis of pulmonary embolism alone (secondary or unprovoked) compared with those discharged with a diagnosis of pulmonary embolism associated with deep vein thrombosis, generating the hypothesis that some specific heart diseases in older patients could be a possible source of pulmonary emboli.[9]

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Table 1. Clinical characteristics of the patients

	Positive history of symptomatic arterial disease N=40	No history of symptomatic arterial disease N=197	P
Age (years)	71±12	57±17	<0.001
Females – n, %	18, 45	99, 50.3	0.605
Chronic diseases – n,%			
COPD	2, 5.0	8, 4.1	0.678
CHF	17, 42.5	18, 9.1	<0.001
CKD	18, 45.0	41, 20.8	0.002
CLD	1, 2.5	6, 3.0	1.000
Diabetes mellitus – n,%	13, 32.5	19, 9.6	<0.001
Arterial hypertension – n,%	31, 77.5	67, 34.0	<0.001
Atrial fibrillation – n,%			
Paroxismal	10, 25.0	16, 8.1	<0.001
Permanent/Persistent	5, 12.5	6, 3.0	
Risk factors for VTE – n,%			
Active malignancy	6, 15.0	27, 13.7	0.804
Familial history of VTE	2, 5.0	23, 11.7	0.269
Active smoking	8, 20	38, 19.3	1.000
Major surgery in 21 days	6, 15.0	20, 10.2	0.405
Obesity	6, 15.0	52, 26.4	0.159
Admission parameters			
Heart rate (beat/min)	100±20	102±22	0.773
SAP (mmHg)	115±27	121±26	0.167
pO_2 (mmHg)	65.8±20	71.9±18.3	0.062
Positive cTnT – n,%	13 out of 30, 43.3	45 out of 122, 36.9	0.535
BNP – median, IQR pg/ml	312.0, 152.6-629.4	112.0, 38.4-268.8	<0.001
RVSP – mean±SD mmHg	51.2±16.2	46.9±19.6	0.193
RV/LV>0.9 – n, %	19 out of 37, 51.4	98 out of 188, 52.1	1.000
sPESI>0 – n,%	33, 82.3	123, 62.4	0.017
Risk – n, %			
Low	10, 25.0	69, 35.0	0.155
Intermmediate	20, 50.0	101, 51.3	
High	10, 25.0	27, 13.7	

COPD – chronic pulmonary diasese, CHF – chronic heart failure, CKD – chronic kidney disaese (GFR<60 ml/min), CLD – chronic liver disease with insufficiency, SAP – systolic arterial pressure, cTnT – cardiac troponin T, BNP – brain natriuretic peptide, RVSP – right ventricle systolic pressure, RV/LV – right ventricle/ left ventricle diameter on multidetector computed tomography, sPESI – simplified pulmonary embolic score index.

Elevated cardiac troponin levels indicate increased mortality in PE. Serum NT-proBNP was proved to be a marker of left ventricular (LV) overload and was used for reliable differentiation between cardiac and pulmonary origin of dyspnoea. However, elevated NT-proBNP levels can be found in APE.[9-13] Interestingly, low levels of brain natriuretic peptide can identify patients with good prognosis.[13-15]

We aimed to explore if the symptomatic peripheral arterial disease (including coronary artery disease) is the predictor of early mortality (in-hospital and 6-month) in patients hospitalized with acute objectively confirmed VTE.

Methods

We included in our prospective study 237 consecutive patients with PE. These patients were admitted in the intensive care unit of university hospital during 6 years. Forty patients had positive history of arterial disease (24 had coronary disease – myocardial infarction or revascularization, 15 had stroke and 1 of them had peripheral arterial disease). In-hospital and six-month mortality was compared between patients with and without previous symptomatic arterial disease. The six-month major bleeding events were also compared between groups. The observed risk factors for VTE were active malignancy, familial history of VTE, active smoking, major surgery in 21 days and obesity.

The admission parameters that were measured were: heart rate, systolic arterial pressure, right ventricle systolic pressure, right ventricle/ left ventricle diameter on multidetector computed tomography, biomarkers, oxygen saturation. Simplified pulmonary embolic score index (sPESI) was calculated and patients' risk was stratified into three groups according to it.

Patients were screened for the following co-morbidities: chronic pulmonary disease, chronic heart failure, chronic kidney disease (GFR<60 ml/min), chronic liver disease with insufficiency, diabetes mellitus, arterial hypertension and paroxysmal and permanent/persistent atrial fibrillation.

Group comparisons for continuous variables with a normal distribution were performed using the t-test and the data were described as means with standard deviations (SD), group comparisons for continuous variables with a skewed distribution were performed using the Wilcoxon rank-sum test and the data were presented as

Table 2. Treatment of patients

	Positive history of symptomatic arterial disease N=40	No history of symptomatic arterial disease N=197	P
Anticoagulant therapy at admission			
NFH	30, 75.0	163, 82.7	0.196
LMWH or fondaparinux	9, 22.5	24, 12.2	
DOAC	1, 2.5	10, 5.1	
Oral anticoagulant therapy[1] – n,%			
Vitamin K antagonist			
AntiXa inhibitors	11, 36.7	55, 30.9	0.255
Dabigatran	10, 33.3	87, 48.9	
Thrombolytic therapy – n,%	9, 30.0	36, 20.2	
	22, 55.0	109, 55.3	1.000

[1]25 patients died before oral anticoagulant therapy was introduced.

median values with inter-quartile ranges (IQRs), and group comparisons for discrete variables were performed using the x2 or Fisher exact test and the data were presented as frequencies and percentages. For identifying clinical factors associated with in-hospital mortality, univariate Cox's regression analysis reporting hazard ratios (HRs) with 95% confidence intervals (CIs) was performed. P-values are two-tailed. The data were analyzed using SPSS version 17.

Results

Patients with PE and symptomatic arterial disease were significantly older 71 vs. 57 years. Genders were similarly and evenly distributed in both groups of patients (Table 1). Investigated co-morbidities were more prevalent in patients with PE and symptomatic arterial disease (except for COPD and chronic liver disease) (Table 1). Risk factors for PE were evenly distributed in both groups of patients. Parameters explored at the hospital admission were similar in both groups of PE patients, only BNP were significantly higher in the group of patients with symptomatic arterial disease and PE. Simplified pulmonary embolic score index was similar in both groups - the majority of patients had intermediate risk score according to it (Table 1). Half of patients in both groups received thrombolytic therapy (Table 2). Almost third of patients received DOAC for chronic anticoagulation, the second third received antiXa inhibitors and the last third was on oral vitamin K antagonists (Table 2). In-hospital and six-month mortality was significantly higher in patients with positive history of arterial disease (30.0% vs 9.1%, p=0.001 and 35.0% vs. 13.7%, p=0.02, respectively). Hazard ratios for in-hospital and six-month mortality adjusted to age and gender were significantly higher in patients with positive history of arterial disease (HR=3.668 95%CI 1.766-7.618, p<0.001, and HR=2.948, 95%CI 1.545-5.626, p=0.001) (igures 1, 2). Positive history of arterial disease was also associated with increased risk of major bleeding (22.5% vs 11.5%, p=0.078) during 6 months follow-up (HR=2.230 95%CI 1.031-4.823, p=0.042) (Figure 3).

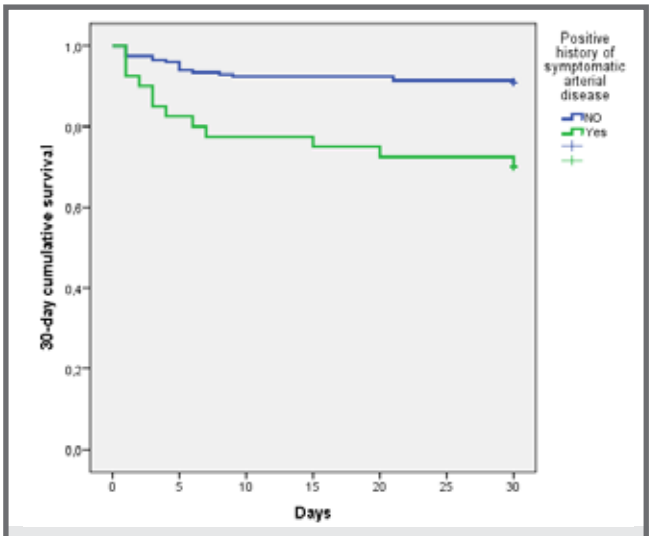

Figure 1. There was significant difference in 30-day mortality between group with positive history of arterial disease and without (log rank p<0.001). 30-day mortality were 30.0% in patients with positive history of symptomatic arterial disease and 9.1% in patients without positive history of arterial disease.

Discussion

We found that individuals, who were discharged with the diagnosis of PE and with the symptomatic peripheral arterial disease, had higher in-hospital and six-month mortality as well as higher rate of major bleeding.

Patients with symptomatic arterial disease were older and had higher BNP at admission. There was no significant difference in risk factors for PE or frequency of other co-morbidities among those two groups of PE patients. Older age and excessive myocardial wall stress (high BNP) were more prevalent in patients with symptomatic arterial disease.

Indeed, older age (a well-known risk factor for atherosclerosis) has long been identified as an independent risk factor for venous thrombosis.[16-19]. A few case-control and prospective studies have found an association between venous thromboembolic disorders and chronic arterial disease of the legs, hyperlipidemia, or hypertension.[19-22]

Furthermore, atherosclerosis is associated with activation of both platelets and blood coagulation as well as an increase in fibrin turnover, which can lead to throm-

botic complications. A role of this prothrombotic state in promoting venous thrombosis is plausible, on the basis of the assumption that activated platelets and coagulation factors appear in the slow flowing venous system.[23-30] The risk of arterial thrombosis in patients with major cardiovascular risk factors is most likely mediated by the presence not only hypercoagulability but the inflammatory state as well. Both increased inflammation and coagulation also may predispose these patients to develop venous thromboembolic events.[30]

It is generally believed that the genesis of venous thromboembolism (VTE) differs from atherosclerotic cardiovascular disease.[31] Prandoni et al. suggest either that atherosclerosis can induce venous thrombosis or that the two conditions share common risk factors.[20]

Recent studies suggest that VTE and cardiovascular disorders may share common risk factors and that in some patients at risk for atherosclerosis, VTE might occur as the first symptomatic cardiovascular event. Yet, 2 large cohort studies challenged this hypothesis by showing that the presence of atherosclerosis was not predictive of an increased risk of VTE.[32,33]

Few studies have shown that, surprisingly enough, the use of statins reduces the risk of venous thromboembolism. Since users of non-statin lipid-lowering agents were not at lower risk of venous thrombosis, the reduction in venous thromboembolism observed in statin users might be explained by the ability of statins to improve endothelial function and prevent the development and destabilization of atherosclerotic lesions.[34-36]

Ageno et al. [34] in their meta-analysis suggest that major risk factors for atherothrombotic disease also are significantly associated with VTE. These findings strengthen the hypothesis that cardiovascular risk factors also may be directly involved in the pathogenesis of VTE and corroborate the results of the studies that challenged the common view that atherosclerosis and VTE are 2 completely distinct disease entities.[34]

The clinical implications of our findings are potentially important. A significant proportion of VTE, between 26% and 47%, is currently classified as apparently unprovoked in the absence of major known risk factors such as cancer, trauma, surgery or medical illness, or pregnancy.[37] Recognition of cardiovascular risk factors, if proven to be relevant for VTE, may substantially lower these numbers and support new strategies for both primary and secondary prevention of venous thrombosis and PE. Indeed, in contrast to hereditary thrombophilias, these risk factors can be ameliorated with appropriate therapy and lifestyle changes. The role of weight loss and antiplatelet and lipid-lowering therapy needs to be specifically assessed. Studies evaluating the role of aspirin in the secondary prevention of VTE are currently underway, and preliminary evidence exists that statins may be protective against VTE.[38] The obvious mechanism for this protective effect is through improving lipid profiles, but another possibility is that statins may have a direct effect on endothelial function and coagulation.[39] Finally, the reported increased risk of atherothrombosis in these patients further stresses the need for routine evaluation and management of cardiovascular risk. Further we could seek for new strategies for improvement prognosis in PE patients whent the disease is fully developed.

Becattini et al. found that patients with a first episode of pulmonary embolism have a high incidence of both venous and arterial events in the 3 years after the index episode. Cardiovascular events are more common in patients with idiopathic pulmonary embolism than in patients with pulmonary embolism associated with transient risk factors as is also the case for arterial events. Interestingly, cardiovascular events were the most common cause of death in patients with pulmonary embolism. These results differ from those of the few other previous studies on the long-term clinical course of pulmonary embolism, where cancer was the main cause of death.[40]

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Sažetak

Simptomatska arterijska bolest kao prediktor hospitalne i šestomesečne prognoze kod obolelih od akutne plućne tromboembolije

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Uvod: *Povezanost postojećeg arterijskog oboljenja i ishoda kod obolelih od plućne tromboembolije (PTE) nije dovoljno istražen. Cilj rada je utvrđivanje udruženosti pozitivne anamneze o simptomatskoj arterijskoj bolesti i intrahospitalne i šestomesečne prognoze kod bolesnika sa akutnom PTE.*

Metode: *Od 237 bolesnika primljenih u intenzivnim negama tokom 6 godina, 40 bolesnika je imalo pozitivnu anamnezu za arterijsko oboljenje (24 je imalo koronarnu bolest- infarkt miokarda ili revaskularizaciju miokarda, 15 je imalo šlog i 1 je imao perifernu arterijsku bolest). Intrahospitalna i šestomesečna prognoza su poređene kod bolesnika sa i bez simptomatske arterijske bolesti. Incidenca šestomesečnog major krvarenja je takođe poređena među grupama.*

Rezultati: *Intrahospitalni i šestomesečni mortalitet je bio značajno veći kod bolesnika sa pozitivnom anamnezom za perifernu arterijsku bolest (30.0% vs. 9.1%, p=0.001 i 35.0% vs. 13.7%, p=0.02). Verovatnoća za intrahospitalni i šestomesečni mortalitet ujednačen za godine starosti i pol je bila značajno viša kod bolesnika sa pozitivnom anamnezom o arterijskom oboljenju (HR=3.668 95%CI 1.766-7.618, p<0.001, i HR=2.948, 95%CI 1.545-5.626, p=0.001). Pozitivna anamneza o arterijskoj bolesti je takođe bila udružena sa povišenim rizikkom od major krvarenja (22.5% vs. 11.5%, p=0.078) tokom 6 meseci praćenja (HR=2.230 95%CI 1.031-4.823, p=0.042).*

Zaključak: *Pozitivna anamneza o postojanju simptomatskog arterijskog oboljenja je udružena sa povišenim rizikom od intrahospitalnog i šetomesečnog mortaliteta, kao i sa rizikom od major krvarenja kod bolesnika sa PTE.*

Ključne reči: *plućna tromboembolija, simptomatska arterijska bolest, prognoza, krvarenje*