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Pulmonary Thromboembolism in Smokers and Non-smokers; Risk Factors and Anatomic Distribution of Emboli in CT Angiographies

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Abstract

Introduction: Recognition of risk factors in different high-risk groups such as smokers in comparison with non-high risk groups would help to develop good preventive strategies for pulmonary thromboemboli (PTE). The purpose of this study was to investigate and compare clinical findings and risk factors in smoker and nonsmoker patients with pulmonary thromboembolism and assessing anatomical variant in pulmonary computerized tomography angiographies.

Methods: In this descriptive study 260 consecutive patients suspected to have PTE underwent pulmonary computerized tomography angiographies in a training hospital since 2015 to 2018. Patient with documented PTE were enrolled. Clinical finding and risk factors were determined and compared between them.

Results: From 260 patients 172 subjects (66.15%) had PTE and enrolled in the study. Fifty-six (32%) were smoker and 116 (68%) non-smoker. The smoker group was younger and male gender was more predominant. Oxygen saturation and inspired oxygen partial pressure differed between smokers and non-smokers ($P < 0.05$). The predisposing factors of thromboembolism and anatomic distribution of emboli were the same in smokers and non-smokers.

Conclusion: Regarding different factors responsible for PTE in smokers and non-smokers, clinical presentation and anatomic distribution of PTE are comparable.

INTRODUCTION

Pulmonary thromboembolism (PTE) is a respiratory disease affecting main pulmonary artery or the branches with clot migration from deep veins in leg or other body sites leading to numerous adverse effects [1, 2]. Definite prevalence is not yet clear but the incidence is increasing. Due to complications such as unexplained deaths, prompt diagnosis and treatment is important [3]. The prevalence rates have shown a 6.5-fold growing trend in a ten-year period with mortality rate of 10.8 per 100000 operations [4]. Despite reduction of thromboembolism prevalence since 2004 by preventive approaches to reduce clot migration from venous to arterial system, still there is high fatality rate and therapeutic costs [5]. Success rate of preventive approaches for PTE is 46 to 56 percent [6]. In this era recognition of risk factors is useful to reduce morbidity and mortality rate [7-11]. The contributing role of smoking is not yet clear and regarding high smoking rate in the community, determination of its impact is crucial. Recognition of risk factors in different high-risk groups

such as smokers in comparison with non-high risk groups would help to develop good preventive strategies for PTE. The purpose of this study was to investigate and compare clinical findings and risk factors in patients with pulmonary thromboembolism in smoker and nonsmoker and assessing outcomes according to expansion and anatomical variant in pulmonary computerized tomography (CT) angiographies.

METHODS

In this descriptive study, 260 consecutive patients suspected to have PTE by pulmonary CT angiographies (with PTE protocol and 64-multi-slice device) in a training hospital were enrolled from 2015 to 2018. All CT angiographies were interpreted independently and blindly by two radiologists. Massive PTE was defined by involvement of more than 50 percent of the main, left or right pulmonary arteries in pulmonary CT angiography or unexpected low blood pressure (systolic≤90 mmHg and diastolic≤60mmHg in normotensive patient). Smoking definition was use of 100 cigarettes or more lifelong. Oxygen saturation and PiO_2 were defined by atrial blood gas at arrival of patients and without O_2 supplement. The heart rate and blood pressure were also recorded in patients after 10 minutes of admissions at rest, when the patients were in sitting or supine position. Exclusion criteria were incomplete data and current treatment with anti-coagulants.

Clinical data and risk factors were extracted from existing medical documents and recorded in a checklist.

Statistics

Data was expressed as mean ± standard deviation (SD) and the categorical variables as number (percentages). The patients divided into two groups according to smoking habits. Chi-square test or Fisher was performed to compare categorical variables in the two groups. Continuous variables were compared by independent Student *t* test or Mann-Whitney U test, based on data distribution.

RESULTS

According to history and physical examination, during the study period, 260 patients who were suspicious to PTE underwent CTA. The mean age of patients was 54 ± 13 years. Also, 58% (n = 150) were male and 42% were smoker (n = 109).

In 172 patients PTE was documented who enrolled in the study. The mean age of patients was 55 ± 12 years (20 to 86 years), 90(52%) were male and 56 (32%) smoker. Most of them had dyspnea, tachypnea and tachycardia. Embolus to the right pulmonary artery was more prevalent compared to the left side. Only 3(1.74) patients died due emboli or comorbidities. Fifty-six (32%) of patients were smoker. Patients divided into two groups according to smoking habits. The average age of the smoker group was 52 ± 23 years, who were significantly younger than the non-smoker group (58 ± 24 years; P = 0.02). Male gender was more prevalent in the smokers (P = 0.001). Oxygen saturation and inspired oxygen partial pressure (PiO_2) were significantly lower in the smokers compared to non-smokers. Embolus to the right pulmonary artery was non significantly more prevalent in the both groups. Also, the distribution of emboli was the same in the both groups (Table 1).

DISCUSSION

This study showed that in patients with PTE, smokers were younger and had lower O_2 saturation. However most of other clinical factors were the same as non-smokers.

Smoking as an important health issue not only affects smokers but also results in some problems in second-hand smokers [12]. Smoking results in inhalation of nicotine, tar products and other numerous chemicals leading to oxidative stress and harmful effects on cardiovascular system and many other organs [13, 14]. Determination of adverse effects due to smoking is important to prevent such consequences.

Previous studies have mentioned undetermined effect of smoking on PTE [15, 16] showing further importance of our study. Also inflammatory effects of smoking and some effects on fibrinogen and suspected role in platelet activation are some proposed factors in this era [17].

The study by Ageno et al. [18] revealed that smoking is a risk factor for venous thromboembolism but it was not assessed in our study. A review study by Corrigan et al. demonstrated that older age, smoking, malignancy, cancer and immobility were most important risk factors for PTE [10]. In a study in Iran among 71 patients, solitary role of smoking in thrombosis was rejected [19]. Courtney et al. [2] revealed that in symptomatic outpatients with highly suspicion of pulmonary emboli some factors such as family history, pleuretic chest pain, and non-cancerous thrombophilia led to higher probability of PTE or deep venous thrombosis (DVT). Zhu et al. analyzed 1850 patients with pulmonary emboli. They found that right pulmonary artery was affected in 58.2% and emboli were detected in the left pulmonary artery in 41.8% [20]. The current study also showed that the right pulmonary involvement was more prevalent in patients with PTE; however, the distribution of emboli was not different between both smokers and non-smokers.

This study had some limitations. We only included patients who survived the pulmonary embolism. Also this examination was performed using a 64 multislice CT device that may have lower accuracy compared to higher resolution devices.

In conclusion, this study showed that regarding different factors responsible for PTE in smokers and non-smokers, risk factors, clinical presentation and anatomic distribution of PTE are comparable.

Table 1: Comparison of Variables among Patients with PTE

Variable	All PTE N = 172	Smoker n=56 (32)	Non-smoker n=116 (68)	P Value
Age, years	55 ± 12	52 ± 23	58 ± 24	0.02
Sex				0.001
Male	90 (52)	40 (71)	50 (44)	
Female	82 (48)	16 (29)	66 (56)	
O2 Saturation	87 ± 8	84 ± 5	mean 89 ±4	0.001
PIO2	78 ± 18	72 ± 11	83+-13	0.001
Tachypnea	123 (71.51)	43 (76.78)	80 (68.96)	0.9
Tachycardia	112 (65.11)	40 (71.42)	82 (70.68)	1.0
Dyspnea	139 (80.81)	46 (82.14)	93 (80.17)	1.0
Pleuretic chest pain	36 (20.93)	16 (28.57)	20 (17.24)	0.4
Hemoptysis	11 (6.39)	3 (5.35)	8 (6.89)	1.0
Syncope	3 (1.74)	1 (1.78)	2 (1.72)	0.8
Fever (> 38.5)	8 (4.65)	3 (5.35)	5 (4.31)	0.9
Blood pressure (mmhg)	129.81 ± 14	133.82 ± 15	128.81 ± 14	0.8
Massive PTE	9 (5.23)	4 (7.14)	5 (4.31)	0.9
Right pulmonary artry PTE	78 (45.34)	23 (41.07)	55 (47.41)	0.8
Left Pulmonary artry PTE	65 (37.79)	20 (35.71)	45 (38.79)	0.9
Bilateral Pulmonary artry PTE	29 (16.86)	13 (23.21)	16 (13.79)	0.9
Intubation	7 (4.06)	3 (5.35)	4 (3.44)	0.2
Death	3 (1.74)	1 (1.78)	2 (1.72)	1.0
Proximal PTE	24 (13.95)	8 (14.28)	16 (13.79)	0.3
Middle PTE	94 (54.65)	28 (50)	66 (56.89)	0.6
Distal PTE	54 (31.39)	20 (35.71)	34 (29.31)	0.3
OCP in female	7 (8.53), n = 82	2 (12.5), n = 16	5 (7.57), n = 66	0.2
Immobility	9 (5.23)	2 (3.57)	7 (6.03)	0.7
Current admission	12 (6.97)	5 (8.92)	7 (6.03)	0.5
Current surgery	38 (22.09)	17 (30.35)	21 (18.10)	0.07
Background Cancer	17 (9.88)	8 (14.28)	9 (7.75)	0.1
Trauma	20 (11.62)	8 (14.28)	12 (10.34)	0.4
Current travel	10 (5.81)	3 (5.35)	7 (6.03)	1.0
Family history of thrombosis	Among 106 cases, 3 (2.83)	2 (3.57)	1 (1)	0.001

Results are shown as mean ± standard deviation or number (%).
PTE: Pulmonary thromboemboli; OCP: Oral contraceptive

Conflict of Interest

Authors declare that they have no conflict of interest.

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