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Effect of air pollution, air pressure and air temperature on new onset pulmonary thromboembolism: A case-control study

Hava kirliliği, hava basıncı ve hava sıcaklığı değişikliklerinin yeni başlangıçlı pulmoner tromboembolizm üzerine etkisi: Olgu kontrol çalışması

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Ethics Committee Approval: The study was approved by the institutional Ethical Committee of Evliya Celebi Training and Research Hospital (2017-KAEK-86/05-37). All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.
Etik Kurul Onayı: Çalışma, Evliya Çelebi Eğitim ve Araştırma Hastanesi Kurumsal Etik Kurulu (2017-KAEK-86/05-37) tarafından onaylandı. İnsan katılımcıların katıldığı çalışmalardaki tüm prosedürler, 1964 Helsinki Deklarasyonu ve daha sonra yapılan değişiklikler uyarınca gerçekleştirilmiştir.

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Abstract

Aim: Air pollution affects many people globally and there are allegations and studies that it leads to serious health problems, such as pulmonary thromboembolism. In this study, we investigated the possible relationship between air conditions and pulmonary thromboembolism.

Methods: This study was carried out by archive scanning. Patients with acute dyspnea who were shown to have PE by contrast-enhanced CT were included in the analysis. Patients with a history of trauma, malignancy, recent surgical intervention, or immobility were excluded from the study. On the day of complaints, Particulate matter 10 (PM10), sulfur dioxide (SO_2), air temperature and air pressure values were obtained online from the relevant institution of Environment and Urban Ministry. These 215 patients' data were then evaluated statistically.

Results: Results suggest that the incidence of pulmonary embolism was higher on days when PM10 (P<0.001) and air pressure levels were high (P<0.001). However, SO_2 and temperature were not directly associated with the frequency of pulmonary embolism (P=0.422, P=0.778).

Conclusion: In light of this study, it can be said that air quality may have different consequences on human health. Elevated PM10 and air pressure levels can affect the circulatory system negatively and aggravate thromboembolism.

Keywords: Particulate matter, PM10, SO_2, Air pressure, Pulmonary thromboembolism, Air pollution

Öz

Amaç: Hava kirliliği, dünyada pek çok kişiyi etkiliyor ve pulmoner tromboembolizm gibi ciddi sağlık sorunlarına yol açtığına dair iddialar ve araştırmalar var. Bu çalışmada, hava koşulları ile pulmoner tromboembolizm arasındaki olası ilişkiyi araştırdık.

Yöntemler: Bu çalışma arşiv taramasıyla gerçekleştirildi. Kontrastlı BT ile PE olduğu gösterilen akut nefes darlığı olan hastalar analize dahil edildi. Travma, malignite, yakın zamanda cerrahi müdahale veya hareketsizlik öyküsü olan hastalar çalışma dışı bırakıldı. Şikayet günü Çevre ve Şehircilik Bakanlığı'nın ilgili kurumundan Partikül madde 10 (PM10), kükürt dioksit (SO2), hava sıcaklığı ve hava basıncı değerleri online olarak alındı. Bu 215 hastanın verileri daha sonra istatistiksel olarak değerlendirildi.

Bulgular: Bulgular, pulmoner emboli insidansının PM10 (P<0,001) ve hava basıncı düzeylerinin yüksek (P<0,001) olduğu günlerde daha yüksek olduğunu göstermektedir. Ancak, SO2 ve sıcaklık, pulmoner emboli sıklığı ile doğrudan ilişkili değildi (P=0,422, P=0,778).

Sonuç: Bu çalışmanın ışığında hava kalitesinin insan sağlığı üzerinde farklı sonuçları olabileceği söylenebilir. Yüksek PM10 ve hava basıncı seviyeleri dolaşım sistemini olumsuz etkileyebilir ve tromboembolizme neden olabilir.

Anahtar kelimeler: Partikül madde, PM10, SO_2, Hava basıncı, Pulmoner tromboembolizm, Hava kirliliği

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Introduction

Air pollution, a global problem that is common in developing countries, can cause serious health problems. According to the World Health Organization data, air pollution causes 700000 premature births in the world every year [1]. It is also reported as a risk factor for mortality and morbidity from cardiovascular and cerebrovascular events [2]. One of the possible mechanic pathways is enhanced thrombosis, hypercoagulability, and vascular endothelial damage. In addition, trauma, immobilization, and malignancy are among the risk factors. Hypercoagulation is a hallmark of thromboembolism, which can manifest as deep venous thrombosis (DVT) or pulmonary embolism (PE). PE is one of the major sudden death causes.

The idea that PM and DVT/PE may be related has recently emerged and studies have shown that there is indeed an interaction. Although studies are limited and some findings are conflicting, there is growing evidence that air pollution is a risk factor for cardiovascular and cerebrovascular events. Studies have been conducted on the effects of meteorological and air pollution parameters on the cardiovascular system and it is stated that the risk of cardiovascular disease may vary depending on the seasons [3]. It has also been reported that the incidence of PE increases when weather conditions are poor or rainy [4], but there is no consensus on this finding [5]. In this study, we investigated whether air pollution, pressure, temperature, and PE are related.

Materials and methods

Patients

After approval of local institutional of Evliya Celebi Training and Research Hospital Ethical Committee (2017-KAEK-86/05-37), the study was conducted by scanning the files of 215 patients, including 97 females and 118 males. The patients were selected among those admitted to the emergency department or chest diseases clinic with sudden onset dyspnea and diagnosed with acute PE by contrast-enhanced computed tomography (C-CT) between January 2012 and March 2019. The control group consisted of patients without PE, who applied with the same complaints. Demographic characteristics of the patients included in the study are listed in Table 1. Patients with a history of PE, malignancy, immobility, hematologic disorders, and recent trauma were excluded from the study. The history of the patients' complaints were recorded from the anamnesis.

Air quality parameters

PM10 and SO_2 values for the period of study were available at www.havaizleme.gov.tr (National air quality monitoring network). The air pressure and air temperature of those days were obtained from www.mgm.gov.tr (General Directorate of Meteorology). Air quality measurement takes different parameters such as PM2.5, O3, NO2 and CO into account. However, since only PM10 and SO_2 measurements were performed regularly in the city where the study was conducted, the study was carried out by considering these two parameters only.

Statistical analysis

We used Statistical Package for the Social Sciences (IBM SPSS Statistic Inc. version 21.0, Chicago, IL, USA) for statistical analysis. Continuous and ordinal variables were expressed as mean (standard deviation) and nominal variables were expressed as frequency and percentage. Kolmogorov-Smirnov test and Shapiro-Wilk tests of normality were used to identify distribution of variables. Chi Square test was used to compare two groups for nominal variables. Mann-Whitney U test was used to compare two groups of non-normally distributed continuous variables, while Student's t test was used to compare two groups of normally distributed continuous variables. The relationship between the air pollutants and PE was evaluated by binary logistic regression analysis. For all tests, a *P*-value of <0.05 was considered statistically significant. Receiver-operating characteristic (ROC) curve was applied for the prediction of PE and the area under the curve was calculated for PM10 and air pressure levels.

Results

Demographic characteristics of the patients are presented in Table 1. A total of 215 patients in the PE (+) group (54.8% male, mean age: 59.7 (6.3) years), and 150 patients in PE (-) group (59.3 male, mean age: 60.2 (5.4) years) were evaluated. Most patients in PE (+) group (66.8%) had concomitant deep vein thrombosis (DVT) (*P*<0.001), and they were either active or previous smokers (65.8%), which was a predisposing factor for PE (*P*=0.014). The other parameters such as gender (*P*=0.189), age (*P*=0.354), BMI (*P*=0.453), HT (*P*=0.181) and coronary heart disease (*P*=0.190) were not related with PE.

Air quality had a remarkable effect on PE development. PM10 levels were higher in the PE (+) group (*P*<0.001), and air pressure levels were significantly different between the two groups (*P*<0.001), while SO_2 and air temperature levels were similar (*P*=0.422, *P*=0.778 respectively) (Table 2).

Table 1: Demographic features of the patients

	PE (+) (n=215)	PE (-) (n=150)	*P*-value
Male (%)	54,8	59,3	0.189*
Age	59.7 (6.3)	60.2 (5.4)	0.354#
BMI (kg/m^2) mean (SD)	25.6 (7.4)	26.4 (4.6)	0.453a
Hypertension (%)	36.7	29.5	0.181*
Coronary Heart Disease (%)	23.8	17.6	0.190*
Deep Venous Thrombosis (%)	66.8	10.2	<0.001*
Smoking (%)			
Current or in the past	65.8	52.5	0.014*

* Pearson Chi- Square, # Student's *t* test, a Mann-Whitney U test

Table 2: Air quality and the other parameters for study groups

	PE (-) group n=150	PE (+) group n=215	*P*-value*
PM 10	78.36 (2.82	100.64 (2.65	<0.001*
SO2	9.79 (0.74	11.43 (0.78	0.422
Air Pressure	902.55 (0.45	905.15 (0.34	<0.001*
Air Temperature	17.038 (0.93	17.27 (0.79	0.778

PM10: Particulate matter 10, SO2: Sulphur dioxide, * Mann Whitney U test

Logistic regression analysis performed for air pollutants showed that the incidence of PE was correlated with PM10 (*P*<0.001, OR [Odds Ratio]: 1.022, 95% CI [Confidence interval]: 1.012-1.032) and air pressure (*P*=0.009, OR:1.147, CI: 1.032-1.272), and was not correlated with SO_2 (*P*=0.908, OR:1.002, CI: 0.971-1.034) and air temperature (*P*=0.629, OR:992, CI: 0.961-1.025) (Table 3).

Table 3: Binary Logistic regression analysis to identify predictors of PE

Variables	*P*-value	Exp(B) Odds Ratio	95% CI Lower Upper
PM10	<0.001	1.022	1.012 – 1.032
SO2	0.908	1.002	0.971 – 1.034
Air Pressure	0.009	1.147	1.035 – 1.272
Air Temperature	0.629	.992	0.961 – 1.025

ROC curve analysis determined a cut-off level of 84.5 for PM10 (Area under the curve (AUC): 0.668, 95% CI: 0.613-723, Long rank $P<0.001$, 64.2% sensitivity and 61.3% specificity) (Figure 1) and 903.5 for air pressure (AUC: 0.670, 95% CI: 0.614-725, Long rank $P<0.001$, 64.2% sensitivity and 63.3% specificity) (Figure 2) for predicting PE.

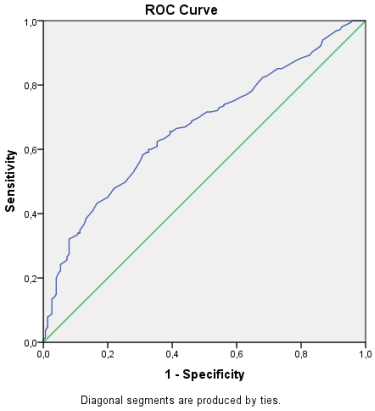

Figure 1: ROC Curve for PM10 and Pulmonary embolism

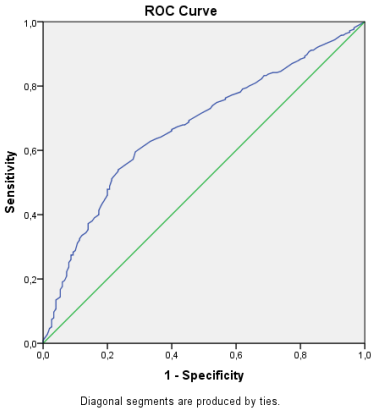

Figure 2: ROC Curve for Air Pressure and Pulmonary embolism

Discussion

The National Morbidity, Mortality and Air Pollution study, which examines the impact of air pollution on health, showed that air pollution above 20-µg / m^3 increases the risk of cardiovascular disease [6]. In another study involving 691 patients, air pollution increased myocardial infarction risk [7]. Myocardial infarction is the secondary systemic effect of pollutants. After pollutant inhalation, pulmonary inflammation occurs first. After translocation of the polluting agent from the lung to blood, secondary systemic effects, such as myocardial infarction, are observed.

Only PM10 and SO2 values could be measured by the National Air Monitoring Center in Kütahya. According to our study, the short-term exposure to an elevated level of air pollutants is a risk factor for PE. Therefore, our study involved these two pollutants. However, carbon monoxide, nitrogen dioxide and sulfur dioxide are also reported to cause air pollution and cardiovascular events [8,9]. This is the major lack of this study.

The air pressure is 1013 mbar at sea level, and values greater than 1013 mbar indicate high pressure. The source of the air pressure is atmospheric gas density, and it decreases with altitude increase. Changes in air temperature and climate affect gas density, causing changes in average air pressure. In the literature, fatal PE cases have also been reported in air pressure changes [10,11]. Kütahya air pressure values for the days included in the study ranged from 894 to 918 mbar (904 (0.25)). In our study, the mean air pressure was significantly higher in the PE (+) group, There are studies indicating that PE frequency increases in cold weather [11,12]. This may be due to exposure to higher concentrations of N_2O and O_3 in cold weather because of decreased gas expansion [12]. However, there are also arguments suggesting that cardiovascular diseases are more common in hot weather [13]. This is a result of dehydration and increased blood viscosity [1]. In our study, the mean air temperature was 17.1°C (0.6) with no significant differences between the groups. The air is also more polluted due to the fuel used for heating in winter. Although higher incidences of PE were reported during winter, there is no consensus in the literature regarding the relationship between air temperature and PE. We also could not detect any relationship between temperature and PE.

Although SO_2 is one of the basic air pollutants [14,15], there are limited series showing the association between SO_2 and PE. A few studies reported that SO_2 was positively correlated with serious cardiovascular disease [16,17,18]. The values measured on the days included in the study were normal. The fact this study was not conducted in an industrial city and the lack of thermal power plants have enabled SO_2 values to remain at normal levels. Consequently, there was no significant relationship between PE and SO_2.

Ambient PM is named according to particle size. Particles between 2.5µg-10µg are called PM10. In various studies, it is stated that especially <2.5µg PMs are more toxic, cause increased inflammatory response and platelet activation and are therefore more dangerous [19,20]. In addition, PM's sympathetic system activation, inflammatory and vasoconstrictive effects begin within the first 12 hours, which later increase coagulation [21]. In our study, PM2.5 was not measured, and our analysis was limited to PM10, which is also one of the main pollutants used in air quality measurement. Diseases caused by high levels of PM10 have been extensively studied in the literature, especially its negative impact on the cardiovascular system [22-24]. In our research, we found that PM10 values significantly correlated with the number of PE cases. Thus, we can easily state that air pollution causes PE.

Limitation

This study was conducted by examining the data of an uncrowded city. Larger studies involving different cities may yield clearer results.

Conclusion

Our study identified that elevated air pollutants are risk factors for PE. Elevated PM10 levels and high air pressure are correlated with acute PE cases. It will be useful to be aware of this relationship and take measures, especially in industrial cities.

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