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THE IMPACT OF CHRONIC EXPOSURE TO AIR POLLUTION ON ELECTROCARDIOGRAPHIC PARAMETERS

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Abstract

Objectives: Chronic exposure to air pollution caused by particulate matter (PM) with aerodynamic diameters of <10 μm (PM_{10}) and <2.5 μm ($PM_{2.5}$), dependent on "low emissions" resulting from the combustion of solid fuels in households, significantly increases the risk of cardiovascular events. The aim of the study was to assess the impact of chronic exposure to air pollution in the place of residence on the occurrence of coronary artery disease, hypertension and the presence of electrocardiographic abnormalities in 24-hour Holter ECG recording. **Material and Methods:** After considering the exclusion criteria, 100 consecutive patients of the cardiology outpatient clinic were enrolled in the study, including 50 patients living for ≥10 years in Warsaw districts with the lowest average concentrations of $PM_{2.5}$ (group I) and 50 living in the districts with the highest recorded exposure (group II). All patients underwent clinical and physical examination, 12-lead ECG, 2D cardiac echo, and Holter ECG. To avoid the impact of acute exposure, the study was carried out in May – the month with statistically the lowest recorded $PM_{2.5}$ concentrations. **Results:** In the group of patients exposed to higher concentrations of $PM_{2.5}$ in the place of residence, coronary artery disease and arterial hypertension were significantly more frequent, while in the Holter ECG examination, ventricular arrhythmias, conduction disturbances and ST-segment and T-wave changes were independently associated with exposure to air pollution. **Conclusions:** Chronic exposure to air pollution in the place of residence contributes to the occurrence of chronic coronary syndrome and hypertension. Chronic exposure to air pollution seems to be a significant factor increasing the incidence of ventricular arrhythmia, conduction disturbances and ST-segment depression episodes in Holter monitoring. Int J Occup Med Environ Health. 2022;35(3):353–60

Key words:
air pollution, ECG, coronary disease, ventricular premature complexes, premature supraventricular beats, Holter ECG

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INTRODUCTION

According to the World Health Organization (WHO) data [1], 90% of the world population breathes air that exceeds acceptable pollution levels. In Poland, due to the dominant share of coal burning for household heating, the highest in Europe concentrations of benzo[a]pyrene as well as particulate matter (PM) with aerodynamic diameters of <10 μm (PM_{10}) and <2.5 μm ($PM_{2.5}$) are recorded. Their small size means that after getting into the respiratory system, they freely overcome tissue barriers and, together with bloodstream, get into all organs of the body.

Based on the analysis of the main causes of death in Poland, air pollution is responsible for about 10% of all deaths from coronary artery disease and strokes [2]. Acute exposure to dust pollution increases the risk of exacerbation of heart failure [3] or heart attack. In previous studies of the long-term effect of air pollution on cardiovascular disease, investigators averaged exposures across a city and then compared health effects between cities [4].

Long-term observations of measurements of air pollution concentrations, supported by mathematical modeling, indicate that within a city there are districts with significantly different average concentrations of PM_{10} and $PM_{2.5}$. However, the gradients of exposure to pollutants within cities affect the risk of death from cardiovascular causes [5] and may be associated with subclinical atherosclerosis [6]. The impact of long-term exposure in the place of residence for electrocardiographic parameters in groups of patients living in different districts of the same city has been poorly studied to date.

Aim of the study

The aim of this study was to assess the impact of chronic exposure to air pollution in the place of residence on the occurrence of coronary arterial disease, hypertension and the presence of electrocardiographic abnormalities in 24-hour Holter ECG recording.

MATERIAL AND METHODS

The study involved 100 consecutive patients of the cardiology outpatient clinic, including 50 consecutive subjects aged 21–88 (age M±SD 62±14 years), living for ≥10 years in districts with the lowest average $PM_{2.5}$ concentrations (group I) and 50 consecutive people aged 29–89 (age M±SD 65±13 years), living for ≥10 years in districts with the highest $PM_{2.5}$ concentrations (group II).

Clinical and physical examinations, 2D cardiac echo (VIVID 7, GE-Healthcare, Horten, Norway), standard resting ECG (ASPEL AsCard GREY v.07.305, ASPEL, Zabierzow, Poland) and 24-hour Holter ECG recording (Lifecard CF, Reynolds Medical, Washington, USA) were performed in each patient.

To avoid the impact of acute exposure on the parameters assessed, the study was conducted over a period of 1 month (May) with the lowest statistically recorded $PM_{2.5}$ concentrations in the year.

Exclusion criteria: patients with congenital and acquired significant valvular diseases, including mitral valve prolapse, early repolarization syndrome, unstable coronary syndrome, left ventricular (LV) dysfunction with diminished ejection fraction (EF, <50%), LV hypertrophy (intra-ventricle septum of >12 mm), signs of heart failure over NYHA II class, dyselectrolytemia, diabetes or active thyroid disease, or exposed to dust, chemicals or other potential harmful substances in their workplace, were excluded from the study.

Chronic coronary syndrome (CCS) was defined according to the actual European Society of Cardiology guidelines [7].

Smoking status: according to the guidelines of the National Center for Health Statistics of Centers for Disease Control and Prevention, the following categories of the smoking status were used [8]:

– current smoker – an adult who had smoked ≥100 cigarettes in his or her lifetime and who currently smokes cigarettes;

– former or ex-smoker – an adult who had smoked ≥100 cigarettes in his or her lifetime but who had quit smoking at the time of interview, with a division into those who had quit smoking >12 and <12 months before;
– never smoker – an adult who never smoked, or who had smoked <100 cigarettes in his or her lifetime.

For the purpose of statistical analysis, the "smoking" category included both current smokers and ex-smokers for <12 months.

Districts with the highest and lowest concentrations of $PM_{2.5}$ were selected on the basis of maps prepared by the Provincial Inspectorate for Environmental Protection based on direct measurements at stations located in individual districts of the city, with subsequent modeling taking into account the height of buildings, the location and height of sources of dust pollution emissions, industrial plants, meteorological data (air temperature, wind speed and direction, and air humidity) during the last 10 years. Within Warsaw, the lowest levels of $PM_{2.5}$ pollution were recorded in the following districts: Rembertów, Białołęka, Wilanów and Bielany, while the highest were recorded in: Wola, Włochy, and Targówek (Figure 1). Other districts were exposed to intermediate $PM_{2.5}$ concentrations or the recorded levels changed significantly in the following years. The average 10-year $PM_{2.5}$ concentrations in selected districts were 15–17 μg/m^3 and 25–35 μg/m^3, respectively.

The study was financed from local statutory funds.

The study protocol was approved by the local ethics committee (16/WIM/2016).

Statistical analysis

Continuous variables were presented as mean values and standard deviations. In the univariate analysis, the t-test and the χ^2 test were used for the analysis of unrelated groups and the independence of variables, respectively. The correlation of ECG abnormalities and clinical features, which in the univariate analysis were of borderline significance or showed a significant relationship with air pollution exposure ($p < 0.05$), were evaluated with using the multivariate analysis of variance (MANOVA). In all analyses, Statistica v. 12 (USA) was used.

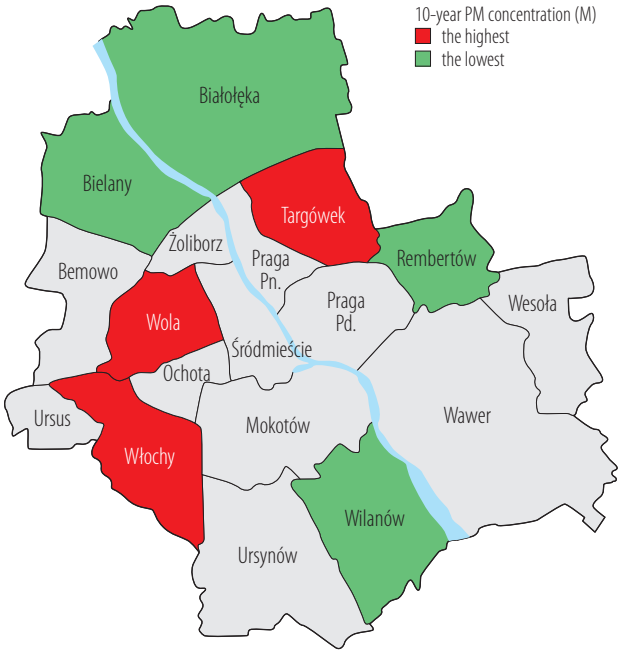

Figure 1. Warsaw districts, Poland

RESULTS

The compared groups did not differ in terms of age and gender. In group II, a history of CCS (including confirmed by angiography) and arterial hypertension was found significantly more often. In group I, during the visit, blood pressure (BP) was higher than indicated by the diagnosed hypertension. In all subjects, the measured BP value was <150/95 mm Hg. The comparative characteristics of the groups are shown in Table 1.

In the group of patients living in districts with elevated PM concentrations, the average daily heart rate (HR) was significantly lower (M±SD 74±8.5 vs. 70±9.3, p = 0.0270). In this group, there was a significantly more frequent occurrence of premature supraventricular contractions (PSVC), premature ventricular contractions (PVC), first-degree atrio-ventricular block, right bundle branch block,

Table 1. Characteristics of the studied groups of 100 consecutive patients of the cardiology outpatient clinic in Warsaw, Poland

Variable	Participants (N = 100)		p
	group I (N = 50)	group II (N = 50)	
Age [years] (M±SD)	62±15	66±14	0.1712
>60 years [n (%)]	32 (64)	37 (74)	0.3871
Gender (men) [n (%)]	26 (52)	27 (54)	1.0
Smoking [n (%)]			
current and ex-smoking for <12 months	7 (14)	15 (30)	0.0911
ex-smoking for >12 months	11 (22)	9 (18)	0.8026
Hypercholesterolemia and/or hypertriglyceridemia [n (%)]	23 (46)	16 (32)	0.2186
Chronic coronary syndrome [n (%)]	14 (28)	28 (56)	**0.0084**
confirmed by angiography	9 (18)	28 (56)	**0.0002**
Hypertension in anamnesis [n (%)]	7 (14)	23 (46)	**0.0011**
Increased blood pressure during the visit [n (%)]	19 (38)	16 (32)	0.6750
>1 of comorbidities (lipids disorder, chronic coronary syndrome, hypertension) [n (%)]	16 (32)	20 (40)	0.5320

Group I – patients living for ≥10 years in the districts of Warsaw with the lowest average $PM_{2.5}$ concentrations; group II – patients living for ≥10 years in the districts of Warsaw with the highest average $PM_{2.5}$ concentrations.
Bolded are statistically significant differences.

paroxysmal atrial fibrillation, and ST-segment and T-wave (ST-T) changes (Table 2). There were no significant differences between complex forms of PVC: pair (4% vs. 14%, p = 0.1622), non-sustained ventricular tachycardia (6% vs. 10%, p = 0.7124), the total number of recorded PVC or PSVC cases (M±SD 565±1514 vs. 682±2346, p = 0.858, and M±SD 1149±3600 vs. 1051±3461, p = 0.9123, respectively). The average EF was similar in the groups (Table 2). There were no differences between males and females in the groups of patients living in selected districts in terms of arrhythmia.

There were no differences in standard treatment between the groups, including β-blockers, angiotensin-converting enzyme inhibitors (ACEI)/angiotensin-receptor blockers or statins. The logistic analysis showed that independent risk factors are:

– for PVCs – the air pollution and smoking,
– for PSVCs – CCS,
– for conduction disturbances – air pollution, CCS,
– for the ST-T changes: air pollution, smoking habits and CCS (Table 3).

DISCUSSION

As reported by WHO, Poland is one of the most polluted countries in Europe. In recent years, public awareness of the importance of the impact of polluted air on health has increased significantly, but there is still a dominant belief in the sole effect on respiratory diseases. Due to the complex pathomechanism of inducing cardiovascular events, there may be difficulties in directly and indisputably demonstrating the effect of air pollution [9]. According to the Institute for Health Metrics and Evaluation estimates, air pollution is responsible for 11.12% of cardiovascular deaths [2], and the risk of cardiac complications is significantly greater than that of pulmonary complications [10].

Table 2. Electrocardiography (ECG) and echocardiographic parameters in relation to the place of the residence in 100 consecutive patients of the cardiology outpatient clinic in Warsaw, Poland

Parameter	Participants (N = 100)		p
	group I (N = 50)	group II (N = 50)	
Heart rate [bpm] (Holter) (M±SD)	74±8.5	70±9.3	**0.0270**
ST-segment depression [n (%)]			
standard ECG	9 (18)	13 (26)	0.4689
Holter	15 (30)	30 (60)	**0.0049**
Premature ventricular contractions (Holter) [n (%)]	16 (32)	30 (60)	**0.0091**
pair [n (%)]	2 (4)	7 (14)	0.1622
non-sustained ventricular tachycardia [n (%)]	3 (6)	5 (10)	0.7124
≥200/day (Holter) [n (%)]	7 (14)	28 (56)	**<0.0001**
if present (M±SD)	565±1514	682±2346	0.8580
Premature supraventricular contractions (Holter) [n (%)]	27 (54)	38 (76)	**0.0360**
≥200/day (Holter) [n (%)]	9 (18)	22 (44)	**0.0095**
if present (M±SD)	1149±3600	1051±3461	0.9123
Paroxysmal atrial fibrillation (Holter) [n (%)]	4 (8)	13 (26)	**0.0332**
Any atrioventricular and/or intraventricular conduction disturbances (Holter) [n (%)]	8 (16)	29 (58)	**<0.0001**
Atrio-ventricular block (Holter) [n (%)]			
first degree	3 (6)	14 (28)	**0.0078**
second degree (type 1)	1 (2)	2 (4)	1.0
second degree (type 2) and third degree	0	0	1.0
Branch block (Holter) [n (%)]			
left bundle	3 (6)	7 (14)	0.3173
right bundle	2 (4)	12 (24)	**0.0095**
Ejection fraction (M±SD)	57.8±3.4	56.7±3.6	0.1195

Holter – Holter monitoring.
Bolded are statistically significant differences.

The aim of this study was, among others, to assess the impact of long-term exposure in the place of residence on the incidence of risk factors for cardiovascular events, including cardiac arrhythmias. The main mechanism damaging the structures of the circulatory system seems to be oxidative stress, contributing to the release of generalized inflammation, which in turn leads to excessive blood clotting, impairment of blood vessel function and intensification of atherosclerotic processes. Ultimately, it leads to increased arrhythmia, destabilization of atherosclerotic plaques and the formation of heart attacks, strokes or other ischemic incidents within the arterial system [6]. The balance of the autonomic nervous system is disturbed by the interaction of dusts with neurotransmitters and their impact on dysfunction of neurohormonal regulation.

Table 3. Relationship between the studied parameters and the occurrence of electrocardiographic changes in 100 consecutive patients of the cardiology outpatient clinic in Warsaw, Poland

Parameter	Electrocardiographic changes							
	PVC		PSVC		ST-T changes		conduction disturbances	
	Wilks' λ	p	Wilks' λ	p	Wilks' λ	p	Wilks' λ	p
High exposure to air pollution	0.956622	**0.000013**	0.898395	0.070525	0.830520	**0.008264**	0.853858	**0.031839**
Smoking	0.789602	**0.030611**	0.873162	0.435961	0.806903	**0.038611**	0.836629	0.100276
Lipid disorders	0.785506	0.453836	0.867686	0.891226	0.787690	0.154772	0.819277	0.389202
Chronic coronary syndromes	0.789439	0.310624	0.9075540	**0.040004**	0.807859	**0.036181**	0.867868	**0.013299**
Hypertension	0.780853	0.940969	0.867776	0.866259	0.791955	0.111739	0.813491	0.779077

PSVC – premature supraventricular contractions; PVC – premature ventricular contractions; ST-T – ST-segment and T-wave changes.

A study by Kunzli et al. [11] demonstrates that long-term PM exposure is not only related to the degree, but also to the progression rate of atherosclerosis, which is now considered as an inflammatory disease with low-density lipoprotein cholesterol accumulated in the arteries. In an animal model, a repeated exposure to PM_{10} was associated with both systemic inflammation and its progression, which was correlated with the number of PM_{10} phagocytosed by macrophages [12]. The thickening of the wall and obliteration of the lumen provokes downstream ischemia of the tissues. In this study, in patients living in more polluted areas, CCS and hypertension were more frequent. Due to the fact that this is only an observational study with a small number of participants, it is indisputably difficult or even impossible to demonstrate the primary influence of the place of residence on cardiovascular diseases.

The possible influence of air pollution on potentially life-threatening tachyarrhythmias can be caused by the decline in the autonomic nervous system activity, the activation of pulmonary neural reflexes and a direct influence of pollutants on the cardiac ion channel.

Previous studies have shown that PM exposure is related to changes in autonomic function, as indicated by heart rate variability or increases in systolic BP during elevated exposure to PM [13–16]. In addition, patients with heart failure after implantation of cardioverter defibrillators had higher rates of discharges due to arrhythmias, associated with air pollution [17]. The results of the ARIA study have revealed that the incidence of episodes of ventricular tachycardia and ventricular fibrillation were correlated significantly with $PM_{2.5}$, but not with PM_{10}, and none of the gaseous pollutants were linked to the occurrence of ventricular tachycardia or ventricular fibrillation [18]. Contrary to this, Wang et al. [19] showed that significant effects of acute exposure to air pollution on arrhythmia were only detected in the cold season. Moreover, there was no evidence of $PM_{2.5}$, PM_{10}, CO and O_3 increasing the arrhythmia risk. The only influence was noted for SO_2 and NO_2. In the study by Dales et al. [20], no relations between acute changes in air pollution and severe arrhythmias in a group of patients with implantable cardioverter defibrillators were found but, contrary to the Polish situation, the recorded PM concentrations were relatively low.

This study confirms the chronic effect of air pollutants ($PM_{2.5}$) on electrocardiographic parameters. The authors observed significantly more frequent abnormalities, such as ventricular and supraventricular arrhythmias, conduction disturbances and ST-segment depression in Holter monitoring, in subjects living in districts with the highest air pollution as compared to subjects living in the least polluted districts. The main question was whether the diag-

nosed coronary artery disease was the effect of air pollution, or whether its presence resulted in a higher rate of arrhythmias. The logistic regression analysis showed that chronic exposure to air pollution was a significant factor in the occurrence of PVC, regardless of CCS. This study found no differences in the medications used, including B-blockers, ACEI, and statins. The authors did not reveal any influence of gender on the analyzed disturbances, nor did they find any difference between EF. It is, therefore, possible that in the patients involved in the study, chronic exposure to air pollution might be responsible for the presence of CCS, arterial hypertension, and arrhythmia as much.

Krishnan et al. [3] and Hornath et al. [21] indicate that $PM_{2.5}$ may impair endothelial function which leads to reduced nitrogen oxide production and the weakening of artery relaxation. Such a mechanism may result in myocardial blood flow dysfunction, subendocardial ischemia and secondary ST-T abnormalities during Holter monitoring, as well as in periodic changes in intraventricular conduction abnormalities.

Interestingly, smoking, including passive smoking, clearly increases the risk of adverse cardiovascular events due to air pollution. Current smokers have a 14%, 31%, and 57% higher risk of arrhythmia, heart failure, and sudden heart death, respectively, compared to non-smokers [22]. In this study, smoking (current or ex-smokers for <12 months) was also related to the presence of PVC and ST-T changes. It is interesting that CCS influenced neither PSVC nor PVC. A possible explanation can be the fact that all analyzed patients were with preserved EF, so the impact of CCS was limited. The mechanisms of the harmful effects of air pollution on the cardiovascular system discussed above can have a greater influence on people already suffering from heart failure, arrhythmia or lung disease. Therefore, measures aimed at reducing the impact of air pollution on the increased risk of adverse cardiovascular events should be implemented in a wide spectrum of the population, including both primary prophylaxis in healthy people and in those with already diagnosed cardiovascular diseases, the frequency of which increases with an aging population.

Limitations of the study

The hormonal status of the participants (especially women), their level of education, typical physical activity or chronic stress exposure, which may all affect the occurrence of arrhythmias, were not analyzed.

CONCLUSIONS

Chronic exposure to air pollution in the place of residence contributes to the occurrence of CCS and hypertension. It seems to be a significant factor increasing the incidence of ventricular arrhythmia, conduction disturbances and ST-segment depression episodes in Holter monitoring.

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