Effects of exposure to air pollution on acute cardiovascular and respiratory admissions to the hospital and early mortality at emergency department

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Abstract

Background. Particulate matter (PM) and NO_2 induce pathophysiological changes which contribute to an increased incidence of acute cardiovascular (CV) and respiratory (Rp) events.

Objectives. To analyze the relationship between air quality and the frequency of admissions to the emergency department (ED) due to the CV diseases and Rp causes.

Materials and methods. The study analyzed the reasons for admissions to the ED during the cold periods from January 2017 to January 2020. These data were combined with the average daily concentrations of NO_2, $PM_{2.5}$ and PM_{10}, and the individual air quality indexes (IAQIs) for these pollutants.

Results. Our analyses have shown that 3468 (11.4%) and 1053 (3.46%) of all 30,419 analyzed patients were admitted to the ED for CV and Rp reasons, respectively. Cardiovascular patients were significantly more often admitted to the ED when the IAQI for NO_2 was worse than very good, and the IAQI for $PM_{2.5}$ or PM_{10} was worse than good. In such periods, diagnoses such as ischemic heart disease (IHD) or syncope were statistically more common and the risk of admission of a patient with a diagnosis such as IHD, heart failure (HF), syncope, stroke, or transient ischemic attack (TIA) was increased. Registered deaths occurred significantly more often among patients admitted on days with moderate or worse than moderate air quality determined in relation to PM_{10} in comparison to days with very good or good air quality (0.35% and 0.23%, respectively, $p = 0.04$).

Conclusions. Air quality significantly affects the admissions to the ED for CV and Rp reasons and has an impact on mortality.

Key words: cardiovascular diseases, air pollution, emergency department, particulate matter, respiratory tract diseases

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Background

Industrial progress leads to an uncontrolled increase in air pollution. The most toxic component of smog is particulate matter (PM) containing PM_{10}, $PM_{2.5}$ and $PM_{0.1}$ fractions. In urban conditions, combustion of fossil fuels remains the primary source of PM and is complemented by products of weathering and decomposition that contain both inorganic (e.g., Pb, Cr, Ni) and organic (e.g., endotoxins, fungal spores, plant debris) compounds.[1–5]

At the cellular level, PM increases oxidative stress, generates free radicals, causes tissue damage, and triggers an inflammatory response.[6–8] These noxious pathophysiological changes exacerbate the impact of air pollution on the human body. They are reflected through an increase of heart rate and blood pressure, deterioration of left ventricular diastolic function, induction of myocardial remodeling, progression of atherosclerosis, and activation of platelets and coagulation system; the overall greater risk of thromboembolism and myocardial ischemia is increased. Inflammation accounts for the majority of these changes through damage and dysfunction of blood vessel endothelium.[9–14] Consequently, all factors contribute to an increased incidence of acute cardiovascular (CV) events.[15]

Although air quality in Poland is one of the worst in Europe, data on the impact of air pollution on health remain limited.[16,17] Albeit a handful of studies conducted on the Polish population have investigated the relationship of air pollution to overall mortality and the incidence of respiratory (Rp) diseases, its impact on CV causes was rarely addressed.

Objectives

To the best of our knowledge, this is the first such analysis carried out in one of the most polluted parts of Poland (Lower Silesia). Our primary goal was to analyze the relationship between the quality of local air and the frequency of all admissions to the emergency department (ED) of the regional tertiary referral center, including hospitalization due to CV or Rp causes and early ED mortality.[17] For this purpose, we tested the correlation between the abovementioned variables and determined the relative risk of admission to the ED for a given reason.

Materials and methods

Study design and setting

Our primary focus was to analyze the causes of admissions and reports to the ED of the University Teaching Hospital in Wrocław, Poland, during the cold months (December–February), from January 1, 2017, to December 31, 2020. The choice of such observation period was dictated by the peak of the heating season when the concentration of airborne toxins reaches its highest levels in year.[1] We compared the data on hospital admissions and air quality retrospectively. The study design is depicted in Fig. 1.

We analyzed data collected from the observation of 30,419 admissions, which accounted for all ED admissions in the studied period.

Variables and data sources

The primary reason for ED visit, along with any additional diagnoses, was established on the basis of medical records obtained from the ED and, if required, other departments to which the patients were referred in the course of the diagnostic and therapeutic processes. The final reason for the report was determined at the patient's discharge. Respiratory causes included the following diagnoses: asthma (exacerbation or symptomatic dyspnea), chronic obstructive pulmonary disease (COPD; exacerbation or symptomatic dyspnea), pneumonia, bronchitis, other lower respiratory tract inflammations, cough, and (unless, on the basis of documentation, it could be attributed to a known cardiogenic cause) shortness of breath. If at least 1 diagnosis on admission to the ED was one of the following, the patient classified as a CV patient: ischemic heart disease (IHD; including symptomatic chronic IHD), unstable angina or myocardial infarction (MI; along with ST/non-ST-elevation MI and unspecified MI), heart failure (HF; exacerbated or symptomatic HF), arrhythmia (Arrh.; atrial flutter, atrial fibrillation (AF), other types of supraventricular tachycardia, ventricular tachycardia, and symptomatic atrioventricular block), elevated blood pressure (EBP; symptomatic or systolic ≥180 mm Hg or diastolic ≥110 mm Hg), stroke or transient ischemic attack (TIA), syncope, venous thromboembolism (VTE; including deep vein thrombosis and pulmonary embolism), or sudden cardiac arrest (SCA). All deaths that occurred within 24 h of reporting to the ED were recorded regardless of the cause.

The mean values of NO_2, PM_{10} and $PM_{2.5}$ used in calculations were obtained from the arithmetic mean of daily automatic measurements performed by the representative air quality monitoring stations at Wiśniowa Street and Korzeniowskiego Street (NO_2, PM_{10} and $PM_{2.5}$), and Bartnicza

Table 1. Individual air quality index (IAQI) for each pollutant

IAQI	Daily average		
	NO_2 [μg/m^3]	$PM_{2.5}$ [μg/m^3]	PM_{10} [μg/m^3]
Very good	0–40	0–13	0–20
Good	40.1–100	13.1–35	20.1–50
Moderate	100.1–150	35.1–55	50.1–80
Sufficient	150.1–230	55.1–75	80.1–110
Bad	230.1–400	75.1–110	110.1–150
Very bad	>400	>110	>150

PM – particulate matter.

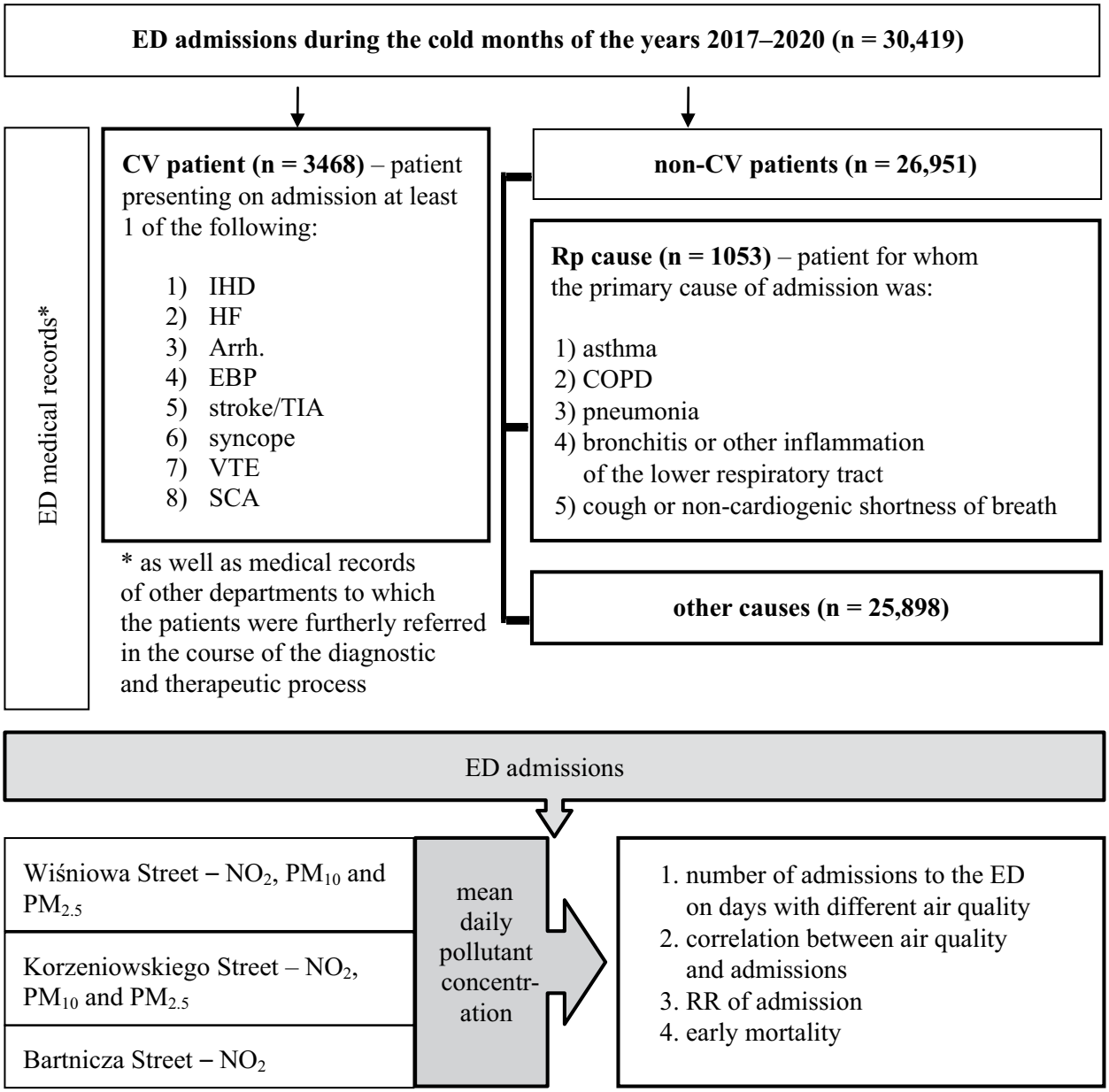

Fig. 1. Flowchart of study design

COPD – chronic obstructive pulmonary disease; ED – emergency department; CV – cardiovascular; Rp – respiratory; IHD – ischemic heart disease; HF – heart failure; Arrh. – arrhythmia; EBP – elevated blood pressure; TIA – transient ischemic attack; VTE – venous thromboembolism; SCA – sudden cardiac arrest; PM – particulate matter; RR – relative risk.

Street (NO_2 only) in Wrocław. In the calculations, the individual air quality index (IAQI), described as air quality index determined on the basis of a single pollutant (NO_2, PM_{10} or $PM_{2.5}$), were referred to as presented below (Table 1). The IAQI is compliant with the standards used to calculate the air quality index established by the Polish General Directorate for Environmental Protection.[18]

Statistical analyses

We analyzed the relationship between the number of reports due to CV and Rp reasons and factors causing air pollution ($PM_{2.5}$, PM_{10} and NO_2). The explanatory variables were continuous variables. We used the Shapiro–Wilk test to check the normal distribution of the compared variables, out of which only the NO_2 variable approximated a normal distribution. The analysis of Kendall rank correlation coefficient (r) between all the reasons for registration to the ED on individual days and the average level of NO_2, $PM_{2.5}$ and PM_{10} on the day of admission (lag = 0) was performed, considering the time delay up to 3 days (lag = 1, lag = 2, lag = 3).

The Mann–Whitney U (M–W) test was performed to compare deaths and admissions on days belonging to 2 different categories of days grouped based on IAQIs. The admissions on days with very good or good IAQIs for $PM_{2.5}$ or PM_{10} were compared with admissions on days

with moderate or worse air quality. In the analyzed period, the IAQI for NO_2 did not qualify to moderate or worse air quality; therefore, the admissions on days with very good IAQI were compared to the admissions on days with good IAQI for this pollutant. The mortality on days with particular air quality was compared in an identical manner.

In the groups of days with specific IAQI, we determined an average daily number of admissions to the ED due to particular reasons. To calculate the relative risk (RR) of hospitalization for a reported reason in relation to the average daily concentration of NO_2, $PM_{2.5}$ or PM_{10} on the day of admission (lag = 0), a generally accepted formula was used. Baseline risk (RR = 1) was derived from the risk of registration to the ED for a particular cause in "very good" air quality conditions, defined as the average daily concentration of an individual pollutant ($NO_2 \leq 40$ µg/m^3, $PM_{2.5} \leq 13$ µg/m^3 or $PM_{10} \leq 20$ µg/m^3). For these calculations, the following categories: sufficient, bad and very bad – were combined to characterize air quality worse than moderate ($PM_{2.5} > 55$ µg/m^3 and $PM_{10} > 80$ µg/m^3). Separately, they described only a few days.

The results of the research were analyzed statistically using the STATISTICA v. 13.3 software (StatSoft Inc., Tulsa, USA). The result was considered statistically significant if $p \leq 0.05$. A retrospective study protocol was approved by the Bioethical Committee of the Wroclaw Medical University, Poland (approval No. KB-722/2019 provided on October 30, 2019).

Results

In the analyzed period, the air quality, determined separately by the average daily concentration of PM_{10}, $PM_{2.5}$ or NO_2 was most often very good or good (Fig. 2). The highest recorded average daily concentrations of NO_2, $PM_{2.5}$ and PM_{10} were 74 µg/m^3, 208 µg/m^3 and 240 µg/m^3, respectively. The average daily level of PM_{10} exceeded the informing level (100 µg/m^3) specified in the ordinance of the Polish Minister of the Environment 9 times, and it exceeded twice the alarming level (150 µg/m^3) set in the same ordinance.[19]

Out of 30,419 analyzed patients, 3468 (11.4%) were admitted to the ED for CV reasons (Table 2). If all admissions for CV reasons are taken as 100%, the most common CV diagnoses were as follows: EBP – 1127 (32.50%), Arrh. – 975 (28.11%), AF – 633 (18.25%), stroke or TIA – 786 (22.66%), HF – 395 (11.39%), and IHD – 330 (9.52%), and MI – 204 (5.88%) (Fig. 3). Apart from CV reasons for admission, respiratory causes as the reason for admission were reported 1053 times (3.46%).

The correlation coefficients between the number of daily admissions and the concentration of tested pollutants are presented in Table 3. The average daily number of admissions for the majority of registered CV and Rp causes is greater in periods of higher daily concentrations of pollutants (Table 4). The RR of reporting to the ED for Rp causes under conditions of different air quality based on IAQIs for the selected pollutant is presented in Table 5.

Table 2. Number of patients registered in the emergency department (ED) in individual months, percentage of CV cases and characteristics of monthly levels of pollutants

Month	All patients	CV patients (% of all patients)	NO_2 [µg/m^3] monthly median (±SD); IQR	$PM_{2.5}$ [µg/m^3] monthly median (±SD); IQR	PM_{10} [µg/m^3] monthly median (±SD); IQR
January 2017	3162	440 (13.92)	36.33 (±10.48); 14.33	51.50 (±33.53); 57.50	62.00 (±34.41); 59.00
February 2017	2740	233 (8.62)	29.17 (±14.18); 17.17	51.50 (±44.36); 51.25	56.00 (±48.23); 54.50
December 2017	3189	327 (11.39)	29.33 (±8.33); 11.00	17.00 (±9.66); 15.00	21.00 (±9.27); 11.00
January 2018	2917	359 (13.10)	28.67 (±8.80); 15.00	21.00 (±14.68); 25.00	25.00 (±14.17); 22.00
February 2018	2703	312 (8.54)	33.67 (±10.51); 15.50	37.50 (±26.00); 19.75	44.50 (±27.29); 23.00
December 2018	3270	325 (9.95)	23.33 (±8.84); 12.33	17.00 (±15.61); 22.50	20.00 (±17.11); 24.00
January 2019	3159	444 (13.92)	27.67 (±9.54); 17.34	19.00 (±25.96); 27.00	17.00 (±26.38); 27.00
February 2019	2870	337 (10.67)	32.00 (±9.66); 12.33	29.75 (±17.60); 17.50	30.50 (±17.27); 23.00
December 2019	3267	290 (9.23)	29.33 (±9.86); 15.00	22.00 (±12.58); 21.00	27.50 (±16.25); 28.00
January 2020	3142	401 (13.75)	30.67 (±7.98); 10.67	23.50 (±20.20); 21.00	27.00 (±23.06); 25.00
Total	30,149	3468 (11.50)	30.00 (±10.38); 13.67	26.00 (±26.59); 29.50	30.00 (±28.83); 30.00

CV – cardiovascular; SD – standard deviation; IQR – interquartile range; PM – particulate matter.

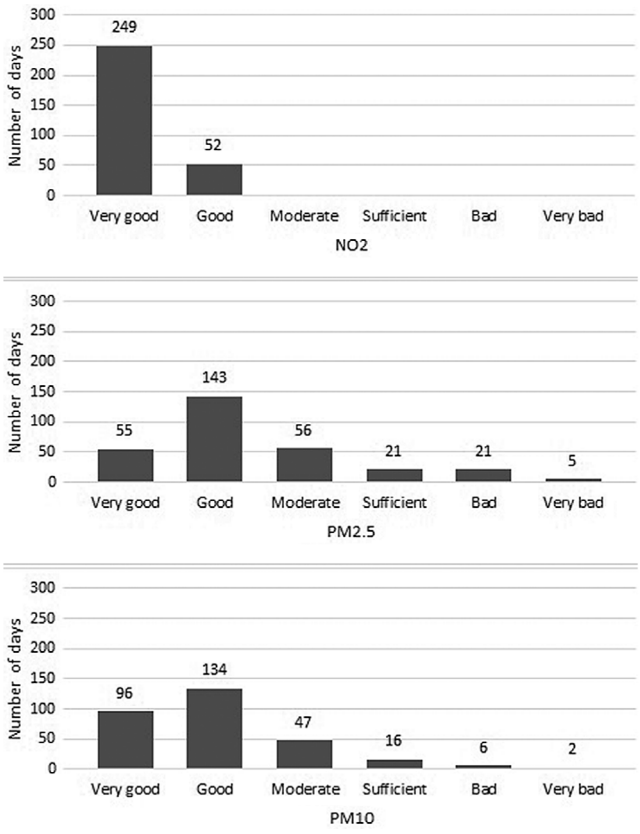

Fig. 2. The number of days in the analyzed period belonging to a given category of the individual air quality index (IAQI)

PM – particulate matter.

In the analyzed period, there were 79 (0.26% of all admitted patients) registered deaths which occurred within 24 h of admission to the ED. These deaths happened significantly more often among patients admitted to the ED on days with moderate or worse than moderate air quality, determined by PM_{10}, in comparison to days with very good or good air quality (0.35% and 0.23%, respectively, M–W test; p = 0.039; Z = 2.06). This relationship, however, did not occur in the case of other pollutants and IAQI categories. Figure 4 depicts the number of deaths per 100 patients admitted to the ED under different air quality conditions for individual pollutants.

Discussion

The main finding of our study is that CV patients were significantly more often admitted to the ED when the IAQI for NO_2 was worse than very good, and the IAQI for $PM_{2.5}$ or PM_{10} was worse than good. In those periods, diagnoses such as IHD and syncope were statistically more common. Moreover, the risk of admission to the ED due to a CV cause was increased for patients diagnosed with IHD, MI, HF, EBP, syncope, stroke, or TIA. Additionally, the negative impact of air pollution below the alarming level (PM_{10} = 150 μg/m^3) and the informing level (PM_{10} = 100 μg/m^3) on the frequency of CV reports is particularly noteworthy.[19]

In our study, the average daily number of admissions due to IHD was higher during periods with worse air quality. Likewise, the RR of reporting due to IHD including MI was increased in each analyzed category of the IAQI, regardless of the selected pollutant, reaching statistical significance

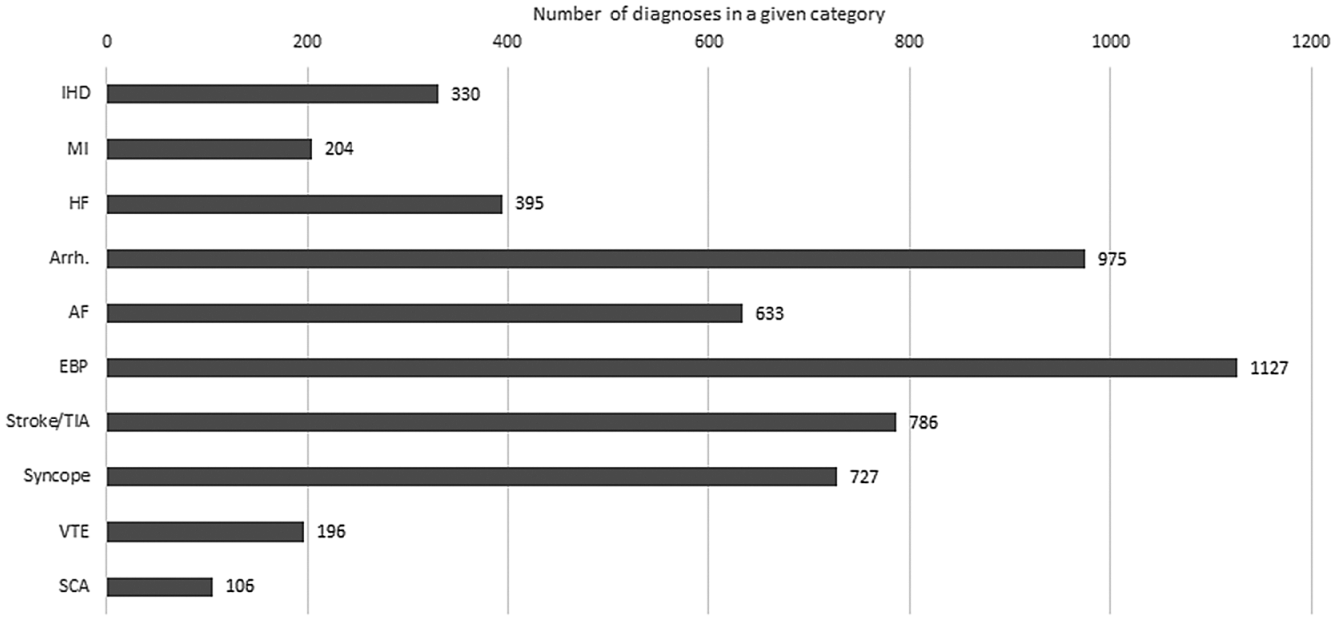

Fig. 3. The number of diagnoses in a given category among all patients registered in the ED for CV reasons. A patient on admission might have had more than 1 diagnosis

ED – emergency department; CV – cardiovascular; EBP – elevated blood pressure; Arrh. – arrhythmia; AF – atrial fibrillation; TIA – transient ischemic attack; HF – heart failure; IHD – ischemic heart disease; MI – myocardial infarction; VTE – venous thromboembolism; SCA – sudden cardiac arrest.

Table 3. Correlation of the daily number of admissions with a given diagnosis with the average daily concentration of NO_2, $PM_{2.5}$ and PM_{10} on the day of admission (lag = 0) and on the day preceding admission by 1, 2 or 3 days (lag = 1/2/3).

Cause of admission	Delay time [days]	NO_2 r; (p-value); [n]	$PM_{2.5}$ r; (p-value); [n]	PM_{10} r; (p-value); [n]
CV patients	lag = 0	**0.179; (<0.001); [301]**	**0.122; (0.002); [301]**	**0.095; (0.014); [300]**
	lag = 1	0.067; (0.084); [300]	**0.082; (0.033); [300]**	0.056; (0.151); [300]
	lag = 2	0.028; (0.464); [299]	0.052; (0.180); [299]	0.019; (0.617); [298]
	lag = 3	0.048; (0.220); [298]	0.059; (0.132); [298]	0.021; (0.593); [297]
IHD	lag = 0	**0.131; (<0.001); [301]**	**0.135; (<0.001); [301]**	**0.142; (<0.001); [300]**
	lag = 1	**0.096; (0.013); [300]**	**0.117; (0.002); [300]**	**0.096; (0.013); [299]**
	lag = 2	0.059; (0.129); [299]	**0.103; (0.008); [299]**	**0.078; (0.044); [298]**
	lag = 3	**0.077; (0.048); [298]**	**0.099; (0.011); [298]**	**0.090; (0.021); [297]**
MI	lag = 0	0.065; (0.094); [301]	**0.107; (0.006); [301]**	**0.126; (0.001); [300]**
	lag = 1	0.059; (0.124); [300]	**0.102; (0.008); [300]**	**0.088; (0.024); [299]**
	lag = 2	0.039; (0.319); [299]	**0.095; (0.014); [299]**	0.074; (0.055); [298]
	lag = 3	0.056; (0.153); [298]	0.065; (0.095); [298]	0.051; (0.189); [297]
HF	lag = 0	**0.113; (0.003); [301]**	**0.108; (0.005); [301]**	**0.104; (0.007); [300]**
	lag = 1	**0.095; (0.014); [300]**	**0.091; (0.019); [300]**	**0.081; (0.038); [299]**
	lag = 2	0.018; (0.641); [299]	0.042; (0.281); [299]	0.029; (0.448); [298]
	lag = 3	0.020; (0.602); [298]	0.056; (0.148); [298]	0.037; (0.341); [297]
Arrh.	lag = 0	**0.082; (0.033); [301]**	0.029; (0.450); [301]	0.018; (0.642); [300]
	lag = 1	0.020; (0.598); [300]	0.021; (0.580); [300]	0.023; (0.545); [299]
	lag = 2	−0.031; (0.417); [299]	−0.037; (0.337); [299]	−0.039; (0.322); [298]
	lag = 3	−0.017; (0.666); [298]	−0.017; (0.668); [298]	−0.015; (0.709); [297]
AF	lag = 0	**0.089; (0.022); [301]**	0.066; (0.089); [301]	0.067; (0.082); [300]
	lag = 1	0.034; (0.374); [300]	0.057; (0.143); [300]	0.069; (0.075); [299]
	lag = 2	−0.007; (0.848); [299]	−0.010; (0.803); [299]	0.003; (0.947); [298]
	lag = 3	−0.002; (0.965); [298]	−0.013; (0.731); [298]	−0.006; (0.868); [297]
EBP	lag = 0	**0.084; (0.031); [301]**	0.075; (0.052); [301]	0.014; (0.722); [300]
	lag = 1	−0.012; (0.761); [300]	0.044; (0.261); [300]	−0.024; (0.543); [299]
	lag = 2	−0.010; (0.794); [299]	0.039; (0.319); [299]	−0.024; (0.544); [298]
	lag = 3	0.033; (0.392); [298]	0.075; (0.053); [298]	0.005; (0.904); [297]
Stroke/TIA	lag = 0	**0.142; (<0.001); [301]**	**0.116; (0.003); [301]**	**0.115; (0.003); [300]**
	lag = 1	**0.090; (0.020); [300]**	**0.092; (0.017); [300]**	**0.101; (0.009); [299]**
	lag = 2	0.043; (0.264); [299]	0.055; (0.153); [299]	0.069; (0.074); [298]
	lag = 3	0.047; (0.223); [298]	0.078; (0.046); [298]	0.072; (0.063); [297]
Syncope	lag = 0	**0.151; (<0.001); [301]**	**0.109; (0.005); [301]**	**0.109; (0.005); [300]**
	lag = 1	0.059; (0.127); [300]	0.069; (0.075); [300]	0.059; (0.129); [299]
	lag = 2	0.033; (0.400); [299]	0.047; (0.229); [299]	0.033; (0.391); [298]
	lag = 3	−0.008; (0.830); [298]	−0.007; (0.852); [298]	−0.013; (0.731); [297]
VTE	lag = 0	0.058; (0.136); [301]	0.015; (0.691); [301]	0.013; (0.733); [300]
	lag = 1	0.016; (0.684); [300]	−0.001; (0.979); [300]	−0.003; (0.947); [299]
	lag = 2	−0.007; (0.867); [299]	0.013; (0.730); [299]	0.024; (0.529); [298]
	lag = 3	0.002; (0.953); [298]	0.015; (0.698); [298]	0.009; (0.823); [297]
SCA	lag = 0	−0.015; (0.696); [301]	0.017; (0.656); [301]	0.025; (0.696); [300]
	lag = 1	0.047; (0.221); [300]	**0.081; (0.036); [300]**	**0.081; (0.037); [299]**
	lag = 2	0.033; (0.390); [299]	0.060; (0.123); [299]	0.033; (0.397); [298]
	lag = 3	0.009; (0.825); [298]	0.037; (0.343); [298]	0.024; (0.540); [297]
Rp causes	lag = 0	**0.227; (<0.001); [301]**	**0.192; (<0.001); [301]**	**0.162; (<0.001); [300]**
	lag = 1	**0.191; (<0.001); [300]**	**0.191; (<0.001); [300]**	**0.158; (<0.001); [300]**
	lag = 2	**0.147; (<0.001); [299]**	**0.150; (<0.001); [299]**	**0.115; (0.003); [298]**
	lag = 3	0.069; (0.075); [298]	**0.080; (0.039); [298]**	0.042; (0.284); [297]

The values of Kendall rank correlation coefficient (r) in bold show statistical significance ($p < 0.05$). CV – cardiovascular; IHD – ischemic heart disease; MI – myocardial infarction; HF – heart failure; Arrh. – arrhythmia; AF – atrial fibrillation; EBP – elevated blood pressure; TIA – transient ischemic attack; VTE – venous thromboembolism; SCA – sudden cardiac arrest; Rp – respiratory; PM – particulate matter.

in most cases. As shown in Table 5, the RR of reporting due to MI was significantly increased even on days with very good air quality defined by the IAQI for PM_{10} (good IAQI for PM_{10} occured in almost 45% of days in the analyzed period). Data supporting our observations can be found in numerous studies from various parts of the world; however, the specific values often differ significantly regarding the strength of the studied relationships.[15,20–22] Recently, 2 studies conducted in Poland have investigated the relationship between air pollution and hospitalization for acute coronary syndromes. In one of them, carried out in Bialystok, PM_{10} concentrations exceeding 50 μg/m^3 corresponded with a higher number of hospitalizations on the day of exposure.[23] Those findings aligned with

Table 4. Average and median daily number of particular diagnoses depending on the individual air quality index on the day of admission (lag = 0)

Daily average (median)	Individual air quality index								
	NO_2			$PM_{2.5}$			PM_{10}		
	very good	good	p-value (Z value)	very good or good	moderate or worse	p-value (Z value)	very good or good	moderate or worse	p-value (Z value)
Number of days	249	52	–	198	103	–	230	71	–
CV patients	11.06 (11.00)	13.75 (13.00)	**<0.001 (3.75)**	10.97 (10.00)	12.58 (12.00)	**0.003 (2.95)**	11.01 (10.00)	13.17 (13.00)	**<0.001 (3.43)**
IHD	1.01 (1.00)	1.50 (1.00)	**0.028 (2.19)**	0.98 (1.00)	1.31 (1.00)	**0.022 (2.28)**	0.99 (1.00)	1.44 (1.00)	**0.022 (2.29)**
MI	0.63 (0.00)	0.88 (0.50)	0.187 (1.32)	0.63 (0.00)	0.78 (0.00)	0.169 (1.38)	0.62 (0.00)	0.86 (1.00)	0.086 (1.71)
HF	1.23 (1.00)	1.69 (1.00)	0.051 (1.95)	1.13 (1.00)	1.66 (1.00)	**0.003 (2.93)**	1.21 (1.00)	1.65 (1.00)	**0.013 (2.48)**
Arrh.	3.18 (3.00)	3.50 (3.00)	0.538 (0.62)	314 (3.00)	3.44 (3.00)	0.283 (1.07)	3.10 (3.00)	3.70 (3.00)	**0.037 (2.08)**
AF	2.08 (2.00)	2.23 (2.00)	0.528 (0.63)	2.01 (2.00)	2.28 (2.00)	0.179 (1.34)	1.98 (2.00)	2.49 (2.00)	**0.024 (2.56)**
EBP	3.61 (3.00)	4.38 (4.00)	**0.031 (2.16)**	3.64 (3.00)	3.95 (4.00)	0.126 (1.53)	3.67 (3.00)	3.97 (3.00)	0.202 (1.28)
Stroke/TIA	2.53 (2.00)	3.02 (3.00)	0.063 (1.86)	2.45 (2.00)	2.92 (3.00)	**0.023 (2.28)**	2.42 (2.00)	3.23 (3.00)	**<0.001 (3.52)**
Syncope	2.27 (2.00)	3.10 (3.00)	**<0.001 (3.45)**	2.25 (2.00)	2.73 (3.00)	**0.008 (2.67)**	2.31 (2.00)	2.76 (3.00)	**0.028 (2.19)**
VTE	0.65 (0.00)	0.67 (0.00)	0.895 (–0.13)	0.66 (0.00)	0.64 (0.00)	0.621 (0.49)	0.65 (0.00)	0.66 (1.00)	0.580 (0.55)
SCA	0.35 (0.00)	0.37 (0.00)	0.790 (–0.27)	0.33 (0.00)	0.39 (0.00)	0.505 (0.67)	0.32 (0.00)	0.45 (0.00)	0.114 (1.58)
Rp causes	3.27 (3.00)	4.79 (4.00)	**<0.001 (4.34)**	3.14 (3.00)	4.27 (4.00)	**<0.001 (3.74)**	3.12 (3.00)	4.85 (4.00)	**<0.001 (5.02)**

The categories „moderate or worse" include days when the individual air quality index was moderate, sufficient, bad, or very bad. The fields containing categories in which admissions for a given reason occur significantly more often (Mann–Whitney test; $p < 0.05$) than on days when the individual air quality index is very good (in case of NO_2) or not better than good (in case of $PM_{2.5}$ and PM_{10}) are in bold. CV – cardiovascular; IHD – ischemic heart disease; MI – myocardial infarction; HF – heart failure; Arrh. – arrhythmia; AF – atrial fibrillation; EBP – elevated blood pressure; TIA – transient ischemic attack; VTE – venous thromboembolism; SCA – sudden cardiac arrest; Rp – respiratory; PM – particulate matter.

Table 5. The RR of registering the given diagnosis on days with different air quality

RR	Individual air quality index													
	NO_2		$PM_{2.5}$						PM_{10}					
	good NO_2 = 40–100 µg/m³		good $PM_{2.5}$ = 13–35 µg/m³		moderate $PM_{2.5}$ = 35–55 µg/m³		worse than moderate $PM_{2.5}$ > 55 µg/m³		good PM_{10} = 20–50 µg/m³		moderate PM_{10} = 50–80 µg/m³		worse than moderate PM_{10} > 80 µg/m³	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
CV patients	**1.24**	1.15–1.34	1.04	0.95–1.14	1.11	1.00–1.24	**1.31**	1.18–1.46	1.02	0.95–1.10	**1.17**	1.06–1.28	**1.29**	1.15–1.45
IHD	**1.48**	1.15–1.90	1.05	0.77–1.44	1.17	0.81–1.69	**1.69**	1.19–2.41	1.31	1.00–1.71	**1.50**	1.07–2.09	**2.15**	1.49–3.10
MI	1.39	1.00–1.93	1.20	0.80–1.81	1.20	0.74–1.94	**1.74**	1.10–2.77	**1.67**	1.17–2.38	**1.72**	1.11–2.68	**2.35**	1.44–3.84
HF	**1.37**	1.08–1.73	1.27	0.93–1.72	**1.79**	1.28–2.51	**1.78**	1.26–2.53	1.11	0.87–1.41	**1.40**	1.05–1.88	**1.54**	1.08–2.20
Arrh.	1.10	0.94–1.28	0.94	0.79–1.11	1.02	0.83–1.24	1.13	0.92–1.39	0.93	0.80–1.07	1.12	0.93–1.34	1.22	0.97–1.53
AF	1.07	0.88–1.31	1.11	0.89–1.39	1.23	0.95–1.60	1.27	0.97–1.66	1.02	0.84–1.22	**1.27**	1.01–1.60	1.28	0.96–1.71
EBP	**1.21**	1.05–1.40	1.07	0.91–1.27	1.09	0.90–1.33	**1.26**	1.03–1.53	0.99	0.86–1.13	1.09	0.92–1.30	1.04	0.83–1.30
Stroke/TIA	1.19	1.00–1.42	1.11	0.91–1.36	1.16	0.92–1.47	**1.50**	1.19–1.90	1.08	0.92–1.28	**1.36**	1.11–1.67	**1.49**	1.16–1.91
Syncope	**1.36**	1.14–1.61	1.09	0.88–1.34	**1.33**	1.05–1.68	**1.30**	1.01–1.67	1.11	0.93–1.32	1.18	0.95–1.48	**1.50**	1.16–1.95
VTE	1.04	0.72–1.49	1.04	0.71–1.53	0.94	0.59–1.51	1.12	1.70–1.80	0.91	0.66–1.26	1.03	0.68–1.56	0.86	0.48–1.52
SCA	1.04	0.64–1.71	0.95	0.56–1.61	0.95	0.50–1.80	1.38	0.75–2.54	0.95	0.60–1.51	1.42	0.82–2.43	1.26	0.62–2.57
Rp causes	**1.46**	1.27–1.68	1.15	0.96–1.38	**1.40**	1.14–1.71	**1.71**	1.40–2.09	1.04	0.90–1.20	**1.62**	1.37–1.92	**1.54**	1.24–1.90

RR = 1 for admission with a given diagnosis in conditions of „very good" air quality. The categories: „$PM_{2.5}$ > 55 µg/m³" and „PM_{10} > 80 µg/m³" include days when the individual air quality index was sufficient, bad or very bad. The values in bold show statistical significance ($p < 0.05$). RR – relative risk; 95% CI – 95% confidence interval; CV – cardiovascular; IHD – ischemic heart disease; MI – myocardial infarction; HF – heart failure; Arrh. – arrhythmia; AF – atrial fibrillation; EBP – elevated blood pressure; TIA – transient ischemic attack; VTE – venous thromboembolism; SCA – sudden cardiac arrest; Rp – respiratory; PM – particulate matter

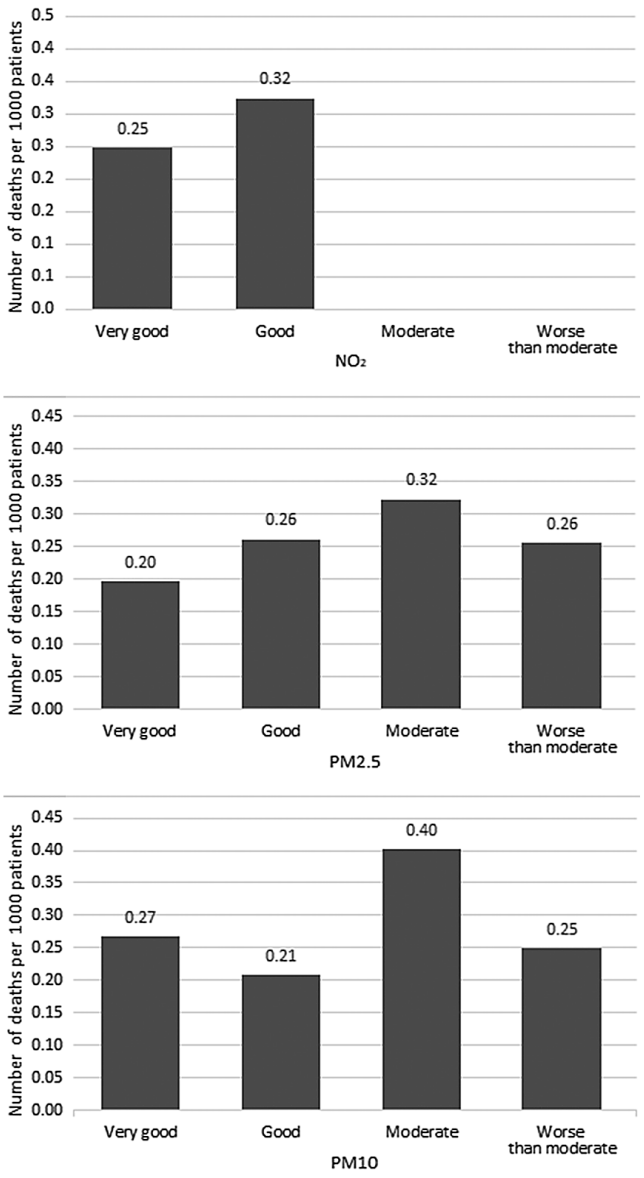

Fig. 4. Number of deaths per 1000 registered patients in a given category of the individual air quality index (IAQI)

PM – particulate matter.

the study conducted in Kraków, where a short-term increase in $PM_{2.5}$ concentration was associated with a higher number of daily hospital admissions due to MI, while for PM_{10}, the effect was statistically significant only with a concomitant decrease in temperature.[24] It should not be forgotten that our work was based on the records from cold periods, hence the temperature differences are undoubtedly responsible for part of the observed effect.

Furthermore, we have shown that the number of patients who reported to the ED resulting from HF was higher on days with poorer air quality, regardless of the pollutant (Table 4). The observed relationship of exposure to polluted air with the diagnosis of HF is stronger in comparison to the results obtained by the majority of researchers. A meta-analysis published in 2013 revealed the negative impact of short-term exposure to polluted air on hospitalization or deaths due to HF.[25] The RR of admission or death from HF due to increase in NO_2 [ppb], $PM_{2.5}$ and PM_{10} [μg/m^3] concentrations by 10 units on the exposure day (lag = 0) rose by about 1% for each of these pollutants, although these relationships became more pronounced when the exposure lasted longer.[25,26]

We observed a significantly higher RR of admission due to the EBP in the conditions of good air quality determined in relation to the average daily NO_2 concentration and worse than moderate air quality determined in relation to PM_{10}. The admissions due to EBP showed a correlation of a weak significance with the concentration of NO_2 on the day of admission, and no correlation with any of the tested pollutants on the preceding days. Nevertheless, literature provides a solid basis that both short-term and chronic exposure to polluted air increase the risk of hospitalization due to EBP and further promote the development of chronic hypertension in exposed individuals.[27–29] It should be noted, however, that a particularly common cause of an increase in blood pressure is the omission of an antihypertensive drug dose, which appears unrelated to atmospheric pollution.[30,31]

The exposure to polluted air or lead is estimated to account for approx. 33% of strokes worldwide.[32] In our study, the risk of stroke or TIA increased significantly (RR = 1.5; 95% confidence interval (95% CI): 1.19–1.90) either when $PM_{2.5}$ exceeded the level of 55 μg/m^3 or when the mean daily PM_{10} was in the moderate air quality category (RR = 1.36; 95% CI: 1.11–1.67). Most likely, this could also be observed at much lower concentrations of PM.[22] Other researchers have noted a significant increase in the risk of admission for these reasons, even at the level of 15 μg/m^3, 24 h before the admission.[20] Both long-term and short-term exposure to polluted air increased the risk of stroke, with the effects being more pronounced for ischemic strokes than hemorrhagic strokes.[32,33] Our findings indicate that the effect is the strongest on the day of stroke occurrence (lag = 0) and decreases over time (Table 3), which aligns with the literature.[32,34]

We found limited data on the relationship between air pollution and the occurrence of syncope. In a study which investigated the reasons for reports to emergency medical services, a significant correlation was established between the reported syncope/loss of consciousness and the concentration of $PM_{2.5}$ on the day of reporting, with a moving average of 2–5 days.[35] In our study, this relationship was among the strongest observed (Table 3,5). However, it seems like this effect could be partly explained by the arrhythmic potential of some pollutants. Fainting could also be a symptom of many other CV diseases, well documented as associated with polluted air. It is also worth noting that the exposure to $PM_{2.5}$ leads to an increased cerebral vascular resistance and, hence, decreased cerebral blood flow.[36] It is possible that some of the reports classified as syncope might have been an episode of TIA. The difficulty in distinguishing syncope from loss of consciousness for

any other reason (when based mainly on patient history) increases the likelihood of analytic bias of the frequency of such reports. Given the limited literature on the subject and the single-center nature of our study, the indications for further research are to analyze the relationship between air pollution and the occurrence of syncope, and to investigate the etiopathogenesis of this phenomenon.

Limitations

Our study has several limitations. The manual method of collecting medical records and creating databases is relatively susceptible to human error, e.g., incorrect interpretation of the disease history or misclassification of cases as individual categories due to ambiguous descriptions. We did not record the location where main symptoms occurred; therefore, some patients admitted to the University Teaching Hospital might have arrived from other, sometimes distant, regions of Poland. In addition, it is noteworthy that weekday and weather corrections were not taken into account and the impact of pollutants other than NO_2, $PM_{2.5}$ and PM_{10} was not assessed. Although this study analyses average daily concentrations of NO_2 and PM, it does not allow to distinguish the effect of short-term from chronic exposure to polluted air. The obtained data are an approximation of how polluted air impacts human health, covering the period of at least a few days before admission, which could explain why the relationships we observed, albeit generally consistent with the available literature, seem stronger than in most of the studies examining short-term exposure to polluted air, and better correspond with the results of long-term exposure. Nevertheless, due to differences in the methodology and presentation of results, a precise comparison of the quoted data was not possible.

Conclusions

Air pollution significantly affects the frequency of admissions to ED for CV and Rp reasons. This impact is observable below the alarming or informing level, and in the case of MI, remains significant even with good air quality in relation to daily PM_{10} concentration. Due to the potentially serious health consequences of aspirating air pollutants, it is crucial to immediately take extensive measures in order to reduce anthropogenic emissions of NO_2, $PM_{2.5}$ and PM_{10}, educate particularly vulnerable patients and limit their exposure to polluted air.

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