

Original Article

Impact of Air Pollution on Cardiac Arrhythmia in Patients With Implantable Cardiac Defibrillators

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ABSTRACT

Objective: Cardiac arrhythmia, a disruptor of heart rhythm, can increase hospitalizations and mortality. This study explored the impact of air pollution on arrhythmia incidence among patients with implantable cardioverter-defibrillators (ICDs) in Mashhad, Iran, and its potential influence on their quality of life and risk of out-of-hospital cardiac arrest.

Methods: In 2018, a study was conducted on patients with ICDs from Imam Reza Hospital. Patients were assigned to an air pollution monitoring station based on their residential address. Arrhythmia data were collected from device interrogations during follow-up visits. A checklist was used to record meteorological data, unhealthy weather days, and levels of carbon monoxide (CO), nitrogen dioxide (NO₂), sulfur dioxide (SO₂), and particulate matter (PM₁₀ and PM_{2.5}). The correlation between arrhythmia and air pollutants was analyzed using time-series and spatial data analyses.

Results: The study involved 194 patients (mean [SD] age =63.14 [12.34] years; 167 men and 27 women). The primary indication for ICD implantation was ischemic cardiomyopathy (70%), followed by dilated cardiomyopathy (18.6%). A total of 1163 cardiac arrhythmias were recorded, with 70% being ventricular and 30% supraventricular. A significant association was identified between the overall count of cardiac arrhythmias and the levels of NO₂ ($P = 0.034$), PM_{2.5} ($P = 0.03$), and CO ($P = 0.004$). Conversely, the concentrations of SO₂ and PM₁₀ did not have a substantial impact on total arrhythmia occurrences. These findings imply that air pollutants such as NO₂, PM_{2.5}, and CO could potentially jeopardize cardiac health.

Conclusions: We concluded that air pollutants, specifically NO₂, CO, and PM_{2.5}, influence the occurrence of cardiac arrhythmia in high-risk groups such as patients with ICDs. The findings highlight the urgent need for comprehensive strategies to mitigate air pollution to reduce the risk of cardiac arrhythmias in vulnerable populations. (*Iranian Heart Journal 2025; 26(4): 22-33*)

KEYWORDS: Implantable cardioverter-defibrillator, Cardiac arrhythmia, Air pollution, Air pollutants, Particulate matter

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Every year, air pollution contributes to the premature death of more than 7 million people worldwide.¹ Multiple lines of evidence have demonstrated a significant association between exposure to air pollution and the incidence of cardiovascular events.²

Approximately 60% of air pollution-related deaths in Europe are attributed to cardiovascular events. A study (2019) conducted in Europe provided compelling evidence that air pollution significantly reduces life expectancy, with an average reduction of 2.2 years in the region.³

During the 1990s, a series of studies revealed an association between elevated levels of air pollution and increased mortality rates from pulmonary and cardiovascular diseases. This pattern was observed in several cities, including Philadelphia, Detroit, and Michigan. On days with higher air pollution levels, a corresponding increase occurred in the number of deaths from these diseases.^{4,5}

Exposure to air pollution can induce alterations in cardiac rhythm, mediated by its effects on the cardiac autonomic nervous system.⁶ Cardiac arrhythmias represent a significant global health concern and are a leading cause of mortality worldwide. These irregular heart rhythms can lead to severe complications and are often fatal if left untreated.⁶

Implantable cardioverter-defibrillators (ICDs) are a crucial tool for managing heart disease and have been shown to significantly reduce mortality rates. By detecting arrhythmias and administering electrical therapy, these devices can effectively terminate potentially fatal arrhythmias.⁷ Numerous studies have explored the association between ventricular arrhythmia and air pollution in patients with ICDs; nonetheless, the results have been inconsistent, leading to ongoing debate within the scientific community.

In a notable study conducted in Iran, Ghorbani et al.⁸ demonstrated a significant association in Mashhad between increased air pollution levels and a rise in cardiac-related fatalities. This association was particularly pronounced with the concentration of particulate matter (particulate matter with a diameter of 10 μm or less [PM₁₀] and particulate matter with a diameter of 2.5 μm or less [PM_{2.5}]).

In 2004, the American Heart Association (AHA) issued a scientific statement indicating a significant association between both short-term and long-term exposure to environmental pollutants and an increased risk of cardiovascular events and mortality.²

The specific cardiovascular outcome most affected remains unclear due to the complex nature of these diseases. Nevertheless, evidence suggests that contributing factors include systemic inflammation, hemostatic pathway activation, vascular dysfunction, progressive atherosclerosis, plaque instability, and cardiac arrhythmias.²

The present study was conducted to investigate the association between air pollution levels and the occurrence of cardiac arrhythmias in patients with ICDs in Mashhad, Iran.

METHODS

This longitudinal descriptive analysis was conducted in a major Iranian metropolis known for its high levels of air pollution. The study participants were patients with ICDs who were referred for device programming to the Cardiac Center of Imam Reza Hospital in Mashhad, Iran. The study period spanned from fall 2018 through fall 2019. Patients were enrolled consecutively after meeting the inclusion criteria and providing informed consent, ensuring a comprehensive and representative sample.

Inclusion criteria:

1. Possession of an ICD for primary or secondary prevention of cardiac arrhythmias.
2. Residence near an air pollution monitoring station to ensure accurate exposure assessment.
3. A primary residence within the city of Mashhad to allow for consistent exposure to local air quality.

Exclusion criteria:

1. Absence of informed consent to participate in the study.
2. Incomplete data recorded in the ICD device memory, which could compromise the accuracy of the results.
3. Extended travel outside Mashhad during the study period. Specifically, participants who were absent from the city for more than 2 consecutive weeks were excluded to ensure consistent exposure to local air quality.

Data collection tools and methods

We collected comprehensive demographic data (including age, sex, and medical history such as diabetes, hypertension, and history of cardiac ischemia) from patients who were referred to the hospital for follow-up and ICD programming. The left ventricular ejection fraction was recorded for each patient. These data served as the primary sample for the study. Additionally, data on arrhythmia incidents over 1 year were meticulously recorded for patients residing in Mashhad. Patient home addresses were geocoded to latitude and longitude coordinates using Google Maps. The study was confined to individuals living within the defined urban perimeter of Mashhad.

Simultaneously, the daily average and monthly levels of urban environmental pollutants, including sulfur dioxide (SO₂ [ppb]), carbon monoxide (CO [ppm]),

nitrogen dioxide (NO₂ [ppb]), and particulate matter (PM_{2.5} and PM₁₀ [μg/m³]), were obtained from the Department of Environmental Pollutants Refinement of the Environment Organization. These pollutants play a crucial role in environmental health and can affect the cardiovascular system. PM₁₀ and PM_{2.5} are particulate matter with aerodynamic diameters of 10 μm and 2.5 μm or less, respectively. They originate from various sources and differ in composition. Combustion emissions, particularly from gasoline, oil, diesel fuel, or wood burning, are major contributors to atmospheric levels of both PM_{2.5} and PM₁₀. NO₂, a sharp-smelling gas, combines with fine particles to form the reddish-brown smog observed in polluted air. It is produced by direct emissions and through sunlight-driven reactions of nitric oxide (NO) with other atmospheric pollutants.⁹

SO₂ emissions primarily originate from the combustion of sulfur-rich fuels by vehicles and diesel-powered machinery, as well as from industrial activities such as fossil fuel extraction, oil refining, and metal processing.⁹ Carbon monoxide (CO) is a colorless, odorless gas produced by the incomplete combustion of fuels, including natural gas, gasoline, and wood. It is emitted from various sources, such as vehicles, power plants, wildfires, and waste incinerators.⁹

A comprehensive record of all detected arrhythmias, including ventricular and supraventricular types, was maintained for 1 year. Concurrently, pollutant concentrations were tracked every 24 hours across various locations in Mashhad for a minimum of 12 months, from fall 2018 through fall 2019.

Statistical Analysis

Data were categorized and analyzed using SPSS, version 21 (IBM Corp). The χ^2 test, *t*-test, and Mann-Whitney test were drawn upon for comparative analysis. The bivariate Moran

spatial correlation was utilized to measure the degree of spatial correlation between variables. The Moran Index (I) is a key indicator of spatial autocorrelation, assessing the extent to which geographically proximate objects exhibit similar attributes by measuring the pattern of distribution for specific values across a geographic space. A Moran I value approaching +1 denotes a clustered arrangement of similar values, while a value nearing -1 indicates dispersion. A Moran I value around 0 suggests the absence of a discernible spatial pattern, implying a random distribution. By aligning these 2 datasets, a daily correlation was established between the frequency of arrhythmias and the levels of each pollutant.¹⁰

Time-series analysis was conducted using the autoregressive integrated moving average (ARIMA) (1,1) model via the ARIMAX method, with a significance level of $P < 0.005$. The tmap package, version 3.3-3, was employed for mapping.

RESULTS

The current study enrolled 194 patients, all residents of Mashhad, who were referred to the Cardiac Center of Imam Reza Hospital for ICD programming. The cohort comprised 167 male participants (86.1%) and 27 female participants (13.9%). The mean (SD) age of the study subjects was 63.14 (12.34) years.

The primary indications for ICD implantation were ischemic cardiomyopathy, accounting for 70.1% of patients, and dilated cardiomyopathy, representing 18.6%. The prevalence of additional comorbidities was less than 5% (Table 1). Primary prevention was the reason for device implantation in 71.1% of the study population.

Table 1. Distribution frequency of disease etiology in the study population

Disease Etiology	Numbers	%
Arrhythmogenic right ventricular dysplasia	1	0.5
Brugada syndrome	1	0.5
Dilated cardiomyopathy	36	18.6
Hypertrophic cardiomyopathy	4	2.1
Ischemic cardiomyopathy	136	70.1
Long QT syndrome	1	0.5
Sudden cardiac arrest	9	4.9
Ventricular tachycardia	5	2.9
Total	193	100%

In this study, 20.4% of participants did not take any antiarrhythmic medication. The most common antiarrhythmic treatment was a β -blocker, used as monotherapy by 46.6% of patients. A combination of a β -blocker and another antiarrhythmic drug, predominantly amiodarone, was used by 15.5% of patients. The use of other antiarrhythmic medications was reported in fewer than 10% of patients.

The prevalence of ventricular arrhythmias was 59.6%, and the prevalence of supraventricular arrhythmias was 31.0%. Of the 1163 recorded arrhythmic events, ventricular arrhythmias constituted 70%, which included ventricular tachycardia (25%), nonsustained ventricular tachycardia (69.8%), and ventricular fibrillation (5.2%). The remaining 30% were supraventricular arrhythmias.

The mean (SD) left ventricular ejection fraction of the study participants was 26% (10%). Diabetes mellitus and hypertension were present in 34.0% and 31.5% of patients, respectively.

Cardiac arrhythmia and air pollutants

Our findings indicate a significant correlation between the incidence of both ventricular and supraventricular cardiac

arrhythmias and levels of specific air pollutants. Pollutants such as NO₂, CO, and PM_{2.5} demonstrated a direct, positive correlation with the average number of cardiac arrhythmias. Conversely, SO₂ levels

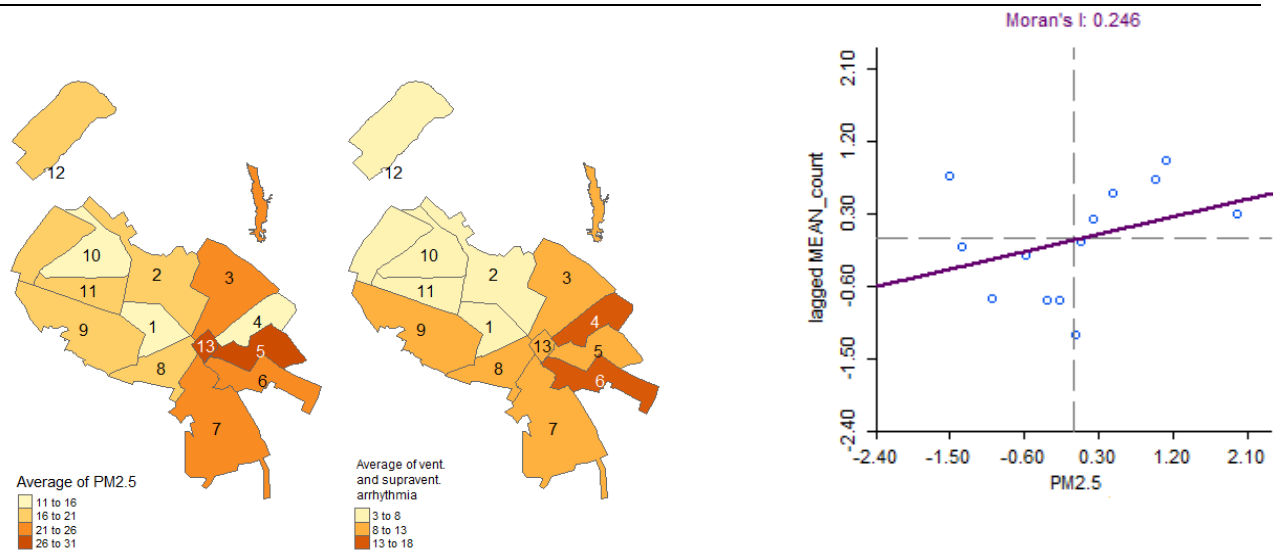
exhibited an inverse relationship with the average number of cardiac arrhythmias. Still, no significant correlation was observed between cardiac arrhythmias and PM₁₀ (Table 1 and Figure 1).

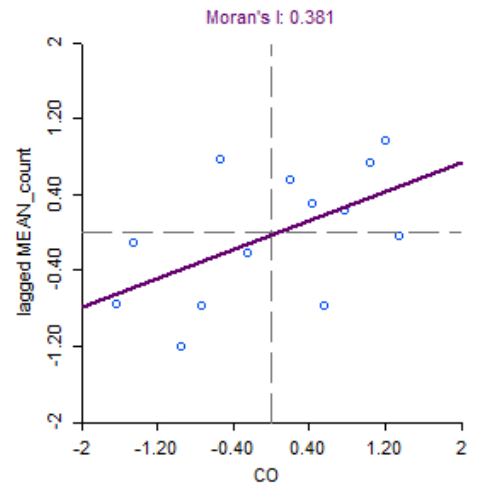
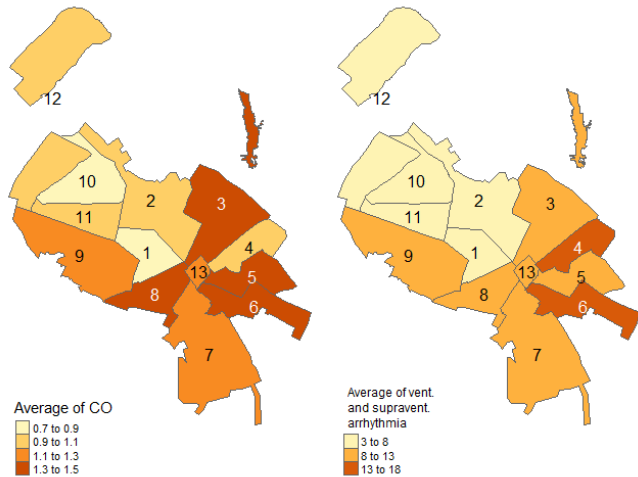
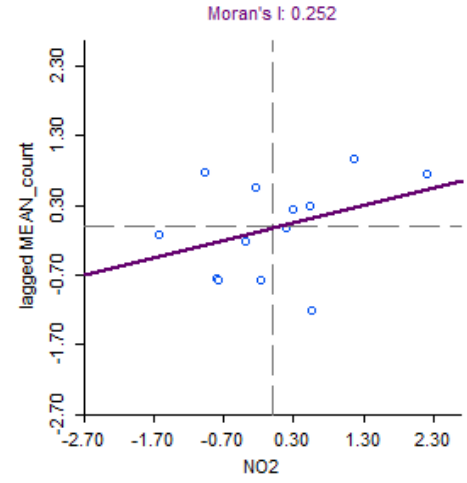
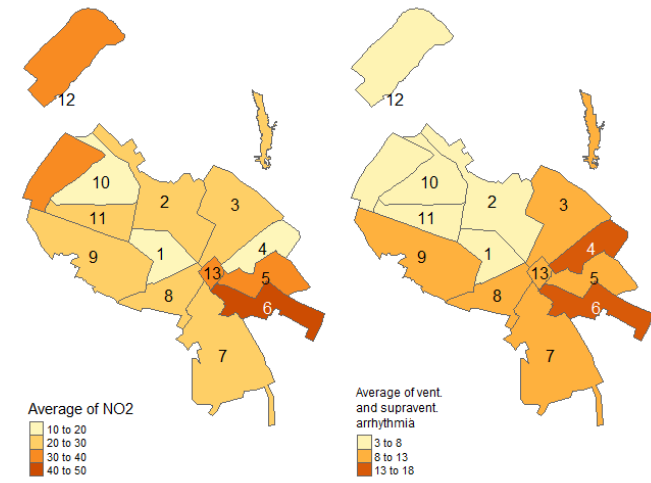
Table 2. Examining the correlation between ventricular and supraventricular arrhythmias and air pollutant levels

Type of Pollutant	PM ₁₀	PM _{2.5}	SO ₂	CO	NO ₂
<i>P</i> *	0.43	0.03	0.01	0.004	0.034
<i>r</i> **	-0.003	0.246	-0.274	0.381	0.252

* spatial regression analysis, ** statistical correlation coefficient of spatial data

PM₁₀: particulate matter with an aerodynamic diameter of 10 µm or less, PM_{2.5}: particulate matter with an aerodynamic diameter of 2.5 µm or less, SO₂: sulfur dioxide, CO: carbon monoxide, NO₂: nitrogen oxide





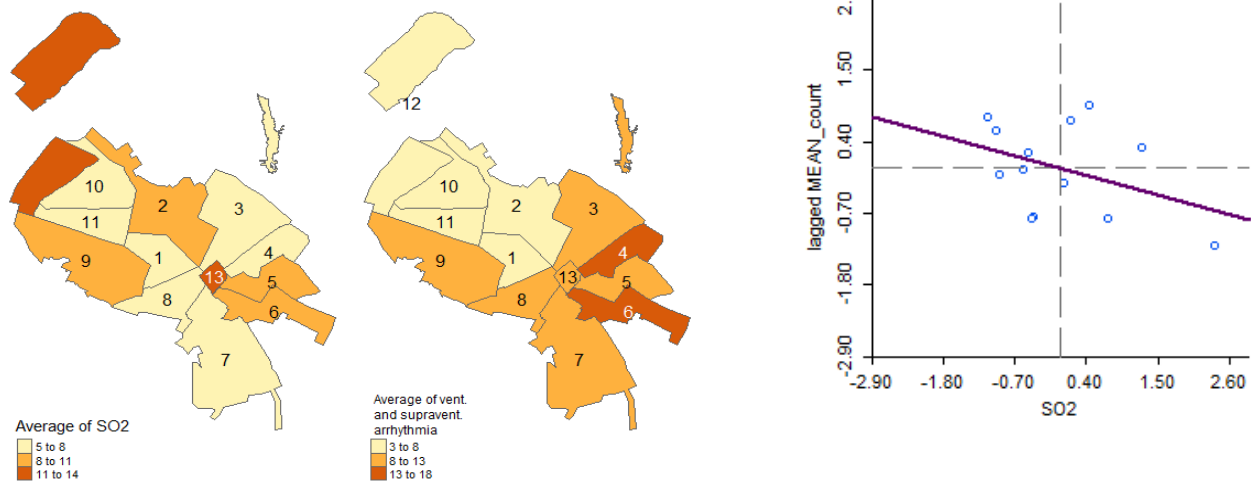


Figure 1. The images present the results of the assessment of the correlation between average ventricular and supraventricular arrhythmias and diverse pollutants across 13 districts of the Iranian city of Mashhad.

Navigating through time

Using the ARIMAX method, we employed an autoregressive integrated moving average (ARIMA) (1,1) regression model to analyze the correlation between the levels of NO_2 , CO , SO_2 , PM_{10} , and $\text{PM}_{2.5}$ and the incidence of cardiac arrhythmias. The analysis revealed a statistically significant association between the concentrations of NO_2 ($P = 0.005$) and $\text{PM}_{2.5}$ ($P = 0.014$) and the occurrence of cardiac arrhythmias. No significant correlation was detected for the other air pollutants (Figures 2 and 3).

Figure 2 depicts the correlation between the incidence of cardiac arrhythmia and the levels of NO_2 in Mashhad's air from

September 2018 through November 2019. The data reveal an upward trend in cardiac arrhythmia cases, indicating a positive correlation. The highest recorded incidence of cardiac arrhythmia occurred in September 2019, with 132 cases, followed by July 2019, with 127 cases.

Figure 3 illustrates the trend of cardiac arrhythmia cases in conjunction with fluctuations in $\text{PM}_{2.5}$ concentration in Mashhad's atmosphere from September 2018 through November 2019. The peak concentration of $\text{PM}_{2.5}$ was recorded in December 2018, reaching a level of $45.74 \mu\text{g}/\text{m}^3$.

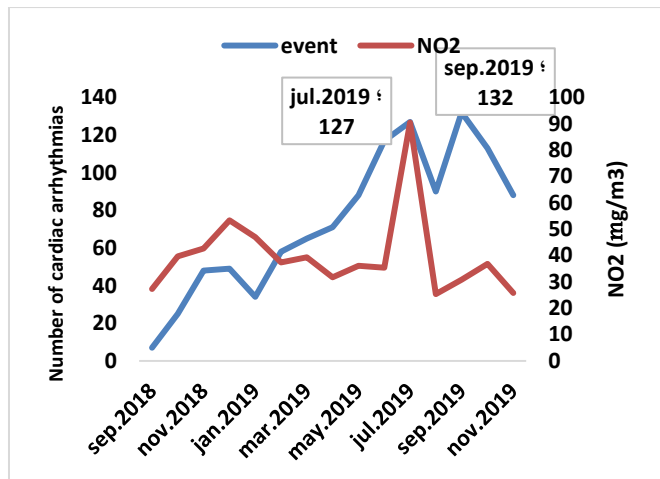


Figure 2. The image presents the results of the examination of the interplay between the incidence of cardiac arrhythmia and nitrogen dioxide (NO₂) concentrations in Mashhad's atmosphere from September 2018 through November 2019.

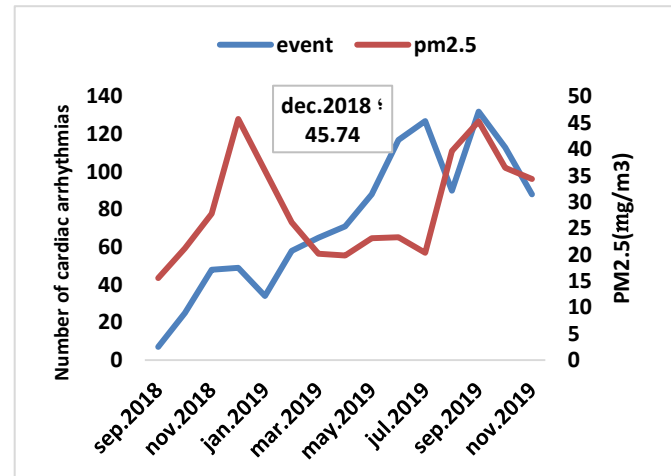


Figure 3. The image presents the results of the examination of the correlation between the incidence of cardiac arrhythmias and the PM_{2.5} air pollution level in Mashhad from September 2018 through November 2019.

DISCUSSION

The present study aimed to assess the association between air pollution levels and the incidence of cardiac arrhythmias in patients with ICDs in Mashhad, Iran. The analysis revealed 1163 recorded cardiac arrhythmic events, with more than 70% being ventricular arrhythmias (ventricular tachycardia, 25%; nonsustained ventricular tachycardia, 69.8%; and ventricular fibrillation, 5.2%) and the remaining 30% being supraventricular arrhythmias.

The investigation into the relationship between the peak daily pollutant levels and the total number of cardiac arrhythmias (both ventricular and supraventricular) on the same day demonstrated a significant association between increased air concentrations of NO₂ ($P = 0.034$), CO ($P = 0.004$), and PM_{2.5} ($P = 0.03$) and an increase in the total number of cardiac arrhythmias. Conversely, SO₂ ($P = 0.01$) was not associated with an increase in the total number of arrhythmias, and the daily concentration of PM₁₀ ($P = 0.43$) showed no correlation with the total number of arrhythmias.

A substantial body of evidence, consistent with the findings of this research, has indicated a potential association between air pollution and cardiac arrhythmias.¹¹ Particulate matter, particularly suspended particles with an aerodynamic diameter of less than 2.5 μm or 10 μm , has been identified as a potential risk factor for cardiovascular events, including cardiac arrhythmias.^{12, 13}

Although our study did not find a significant correlation between PM₁₀ concentration and the incidence of cardiac arrhythmias, numerous other studies have highlighted its role in their development. For instance, Link et al.¹⁴ suggested that PM₁₀ is associated with an increased risk of atrial fibrillation within a few hours of exposure in patients with implantable defibrillators. Rich et al.¹⁵ observed a positive association between the risk of ventricular arrhythmia and particulate matter concentration in patients with an ICD.

Moreover, in a study conducted by Moderato et al.¹⁶ in 2023, both short-term and medium-term exposure to PM_{2.5} and PM₁₀ were independently associated with an

increased risk of out-of-hospital cardiac arrest.

Tsai et al.¹⁷ reported a significant association between high levels of particulate matter (PM_{2.5} and PM₁₀) and the number of emergency department visits for cardiac arrhythmia on cold days (<23 °C).

Further analysis revealed a direct and significant correlation between the monthly average levels of NO₂ and PM_{2.5} and the monthly average number of total cardiac arrhythmias; however, this correlation was not evident for CO, SO₂, and PM₁₀.

Consistent with our findings, a substantial body of evidence has underscored the role of PM_{2.5} in increasing susceptibility to cardiac arrhythmias. Feng et al.¹⁸ proposed that the risk of various arrhythmias, including supraventricular premature beat, atrial tachycardia, ventricular tachycardia, and ventricular premature contraction, is associated with the concentration of PM_{2.5} in ambient air. A study conducted by Han et al.¹⁹ in China demonstrated a significant association between short-term exposure to NO₂ and the frequency of hospital visits for atrial fibrillation. Chiu et al.²⁰ showed that emergency department visits for cardiac arrhythmia increased following short-term exposure to PM_{2.5} on both hot and cold days. Zanobetti et al.²¹ found that the risk of ventricular ectopy increased following exposure to PM_{2.5} and black carbon in elderly patients.

Overall, more evidence supports the role of PM_{2.5} in the development of cardiac arrhythmia compared with other pollutants.²² Danesh Yazdi et al.²³ (2021) investigated the association between long-term exposure to air pollutants, including PM_{2.5}, NO₂, and ozone, and the incidence of cardiac arrhythmias leading to emergency department visits. The study, which included 63,006,793 individuals in the United States from 2000 through 2016, found that long-term exposure to PM_{2.5} significantly

increased the risk of all negative cardiovascular and pulmonary outcomes, including myocardial infarction, ischemic stroke, atrial fibrillation, and pneumonia. Specifically, each 1-μg/m³ increase in PM_{2.5} was associated with an increase of 1575 atrial fibrillation cases per year. An increase of 1 ppb in NO₂ was correlated with a 0.00129% increase in atrial fibrillation cases.²³

A study by Anderson et al.²⁴ (2021) on the impact of long-term exposure to air pollution on the incidence of atrial fibrillation, with a median follow-up of 18.5 years, showed that every 3.9-μg/m³ rise in the 3-year mean PM_{2.5} concentration was significantly associated with an increase in atrial fibrillation.

Pratla et al.²⁵ (2020) investigated the association between the occurrence of ventricular arrhythmias and air pollution in 176 patients with dual-chamber ICDs. They found that for each unit increase in PM_{2.5}, the rate of ventricular arrhythmia increased by 39% over 3 weeks. The study concluded that in high-risk populations, the PM_{2.5} level is associated with the rate of ventricular arrhythmia.²⁵

Our study revealed a notable inverse correlation between the prevalence of cardiac arrhythmias and the concentration of SO₂. This finding appears to be coincidental, as no known biological mechanism explains a protective effect of SO₂ against arrhythmias. Conversely, other research has indicated a significant positive association. For instance, in a 2017 study by Kim et al.,²⁶ a cohort of 160 patients with ICDs in Seoul was monitored over 5 years to examine the effect of air pollutant concentration on ventricular tachyarrhythmia. The findings revealed a significant positive correlation between sustained ventricular tachycardia or ventricular fibrillation and the atmospheric concentrations of SO₂, PM₁₀, NO₂, and CO.²⁶

Indeed, some studies present findings contrary to ours. By way of example, research by Delas et al.⁶ (2020) investigated the effects of ambient air pollution on ICD performance in Ontario, Canada. Their study did not establish an association between ambient air pollution and an increased risk of arrhythmia in patients with ICDs.⁶

Rich et al.²⁷ (2005) investigated the association between environmental pollutants and ventricular arrhythmias in patients with ICDs. Their findings suggested no association between acute exposure to pollutants (within 24 hours before an arrhythmia) and the incidence of arrhythmia.²⁷

Given these conflicting results, further research is necessary to definitively establish the role of gaseous pollutants in cardiac arrhythmia.

CONCLUSIONS

The present study aimed to assess the association between air pollution and the incidence of cardiac arrhythmias in patients with ICDs in Mashhad, Iran. We found a significant correlation between the total number of cardiac arrhythmias, particularly ventricular arrhythmias, and the same-day ambient air concentrations of NO₂, PM_{2.5}, and CO. Nonetheless, no such association was observed with SO₂ levels or PM₁₀. A direct and significant relationship was also noted between the monthly average levels of NO₂ and PM_{2.5}, and the monthly average number of total cardiac arrhythmias, but not with CO, SO₂, or PM₁₀.

In conclusion, these findings underscore the association between specific air pollutants (NO₂, CO, and PM_{2.5}) and the occurrence of cardiac arrhythmias in high-risk groups, such as patients with ICDs.

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cardiovascular diseases. Her expertise and insights significantly enriched this study and were pivotal to its successful completion.

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Conflict of Interest

The authors declare no conflicts of interest, financial or otherwise.

Limitations

This study examined the association between air pollution and cardiac arrhythmias, a complex issue with numerous contributing risk factors. The findings may not fully capture the intricate interactions among these factors due to the multifaceted nature of arrhythmia occurrence. Thus, this study has limitations, and further research is needed.

Ethics Approval and Consent to Participate

The study protocol was approved by the Medical Ethics Committee in Iran following a comprehensive scientific and statistical review (approval code: 980272; ethics code: IR.MUMS.MEDICAL.REC.1398.336). Informed consent was obtained from all participants.

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