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ORIGINAL ARTICLE

The Relationship between Environmental Particulate Matter and ECG Parameters, SpO2, Creatine Kinase-MB, and Cardiac Troponin in Cardiovascular Patients with COVID-19

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Abstract

***Background*:** This study investigated the relationship between environmental particulate matter and electrocardiographic parameters, SpO2, creatine kinase-MB, and cardiac troponin in cardiovascular patients with COVID-19.
***Methods*:** This cross-sectional analytical study obtained information about the concentration of environmental particulate matter (PM10, PM2.5) from March 20, 2020, to March 20, 2023, from the Abadan Environmental Department. Clinical and laboratory information of cardiovascular patients with COVID-19 who were hospitalized at Ayatollah Taleghani Hospital in Abadan during this period was collected. The relationship between environmental particulate matter and SpO2, ECG parameters, troponin, and creatine kinase MB in these patients was investigated.
***Results*:** 58.5% of patients were female, with a mean age of 63.03 ± 15.21. The mean ejection fraction (EF) was 30.41 ± 22.69, and the mean peripheral capillary oxygen saturation (SpO2) was 87.56 ± 11.28, which were lower than normal. The study of the association of environmental particulate matter with clinical and laboratory diagnostic factors in cardiovascular patients with COVID-19 showed that the association between disease progression and troponin T with PM2.5 was significant, and no significant association with environmental particulate matter was observed in other factors. Also, mean PM2.5 was significantly higher on days when patients died, and it was significantly higher in patients with positive troponin T.
***Conclusion*:** These findings suggest that exposure to higher levels of PM2.5 may be associated with an increased mortality risk compared to recovery. However, further studies controlling for confounding variables must confirm the causal relationship.

Keywords: Particulate matter, Covid-19, Cardiovascular Diseases, Air Pollution, Troponin

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INTRODUCTION

Air pollution is one of the serious environmental problems that has many adverse effects on human health. One of these effects is its impact on cardiovascular diseases. Particulate matter can enter the respiratory system and bloodstream, affecting various body systems [1].

PM2.5 are fine airborne particles with a diameter equal to or less than 2.5 micrometers. These particles comprise complex substances that can cause respiratory and cardiovascular diseases [2]. PM2.5 particles are considered one of the most significant health-threatening factors, causing the death of several million people annually worldwide [3, 4].

Cardiac troponin T (cTnT), cardiac troponin I (cTnI), and creatine kinase-MB (CK-MB) are proteins that indicate injury to the heart muscle. Elevated levels of these markers strongly suggest damage to the myocardium [5]. It is reported that 72% of premature deaths due to air pollution are caused by ischemic heart disease and stroke, which

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confirms that the cardiovascular system is one of the most sensitive body systems affected by environmental air pollution [6].

Viral pneumonia is recognized as a typical clinical manifestation of COVID-19 [7]; however, infection with this virus can also lead to cardiovascular complications such as acute coronary syndrome (ACS), arrhythmia, thromboembolism, and, myocardial injury (8). Myocardial injury during COVID-19 is associated with a higher risk of mortality [9].

Given the harmful effects of air pollution on the cardiovascular system, and considering the COVID-19 pandemic and its cardiovascular complications, close monitoring of cardiac biomarkers is essential to reduce complications and mortality associated with COVID-19 and air pollution. This study investigated the relationship between environmental particulate matter and changes in SpO2, EKG, troponin, and CK-MB in cardiovascular patients with COVID-19 in Abadan.

METHODS

This study, was a cross-sectional analytical study, received information on the concentration of environmental particulate matter (PM2.5, PM10) from March 20, 2020, to March 20, 2023, from the Abadan Environmental Department and the Khuzestan Environmental Organization. Information on 303 cardiovascular patients with COVID-19 who were hospitalized at Ayatollah Taleghani Hospital in Abadan (a city in Khuzestan Province, located in southwestern Iran) (Figure 1) during the study period was evaluated. PM concentrations were examined on the days patients were admitted to the hospital. The relationship between mean annual PM concentrations (PM2.5, PM10) and SpO2, ECG parameters (Q-T interval, P-R interval, and ST Segment), troponin, and creatine kinase MB (CK.MB) in these patients was examined. By referring to the Hospital Information System (HIS) of Abadan University of Medical Sciences and reviewing medical records, clinical and laboratory information of cardiovascular patients with COVID-19 hospitalized in Ayatollah Taleghani Hospital in Abadan from March 20, 2020, to March 20, 2023, was obtained. Patients with COVID-19 were diagnosed using computed tomography scans that indicated lung involvement or nasopharyngeal swabs that tested positive for SARS-CoV-2 through reverse transcription polymerase chain reaction (RT-PCR). Information was collected using a checklist. The inclusion criteria in this study were cardiovascular patients with COVID-19 who were hospitalized in Ayatollah Taleghani Hospital in Abadan from March 20, 2020, to March 20, 2023, and their clinical and laboratory information was available. The exclusion criteria for the study included cardiovascular patients with COVID-19 who were hospitalized at Ayatollah Taleghani Hospital in Abadan from March 20, 2020, to March 20, 2023. However, the clinical and laboratory information in their files was incomplete.

This study reports descriptive statistics for quantitative and qualitative variables separately. For quantitative variables such as age, length of stay (days), and laboratory indices (such as CPK, SpO2, EF, CK-MB, etc.), mean, and

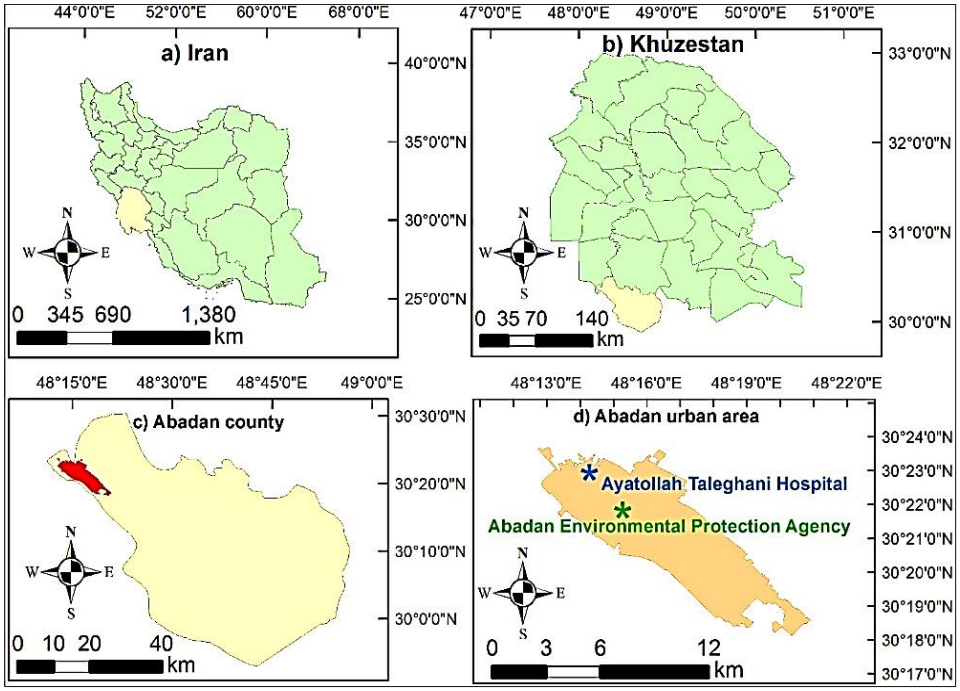

Figure 1. Geographical location of (b) Khuzestan province, Abadan (c) county and (d) city in (a) Iran

standard deviation (Mean ± SD) are calculated and resented. Qualitative variables such as gender, diagnosis, progression status (died or improved), and other demographic and clinical characteristics, as well as frequency (N) and percentage (%), are reported.

Statistical analyses were performed to investigate the association between particulate matter concentrations (PM10 and PM2.5) with laboratory and clinical diagnostic factors and assess statistical differences between groups. Normality of continuous variables was assessed using the Shapiro–Wilk test and by visually examining histograms and Q–Q plots. To compare the mean concentrations of PM10 and PM2.5 among different groups, such as those based on progression status, disease diagnosis, and the presence or absence of specific conditions, we utilized independent t-tests or one-way analysis of variance (ANOVA). The results are reported alongside the corresponding P values. Additionally, Pearson's correlation coefficient (r) was employed to assess the relationship between quantitative variables, such as SpO2, EF, and CPK, and the concentrations of PM10 and PM2.5. All analyses were performed at a significance level of 0.05, with P values less than 0.05 indicating a significant difference or relationship. All statistical calculations were performed using SPSS version 16.

RESULTS

The mean PM10 concentration is 135.74 ± 118, which is unhealthy for sensitive groups, and the mean PM2.5 concentration is 23.32 ± 16.5. Table 1 shows the frequency distribution of demographic and clinical characteristics of cardiovascular patients with COVID-19 participating in the study. According to the results in terms of gender, 58.5% of patients were female (177 patients), and 41.6% were male (126 patients). The mean age of the patients was 63.03 ± 15.21.

In terms of disease diagnosis, 33.7% of patients had atherosclerotic heart disease, 20.8% had heart failure, 34.7% had cardiac arrest, and 10.9% had embolism and phlebitis. In terms of disease progression, 75.6% of patients died.

The mean creatine phosphokinase was 158.60 ± 229.04, and the mean creatine kinase was 41.74 ± 71.07, which was within normal limits. However, the mean ejection fraction (EF) was 30.41 ± 22.69, which was lower than normal. The mean peripheral capillary oxygen saturation (SpO2) was 87.56 ± 11.28, which was lower than normal. The mean blood pressure was 120.56 ± 26.32. The mean heart rate was 91.85 ± 16.22, which, given that most patients are elderly, is higher than normal. The mean respiratory rate was 21.33 ± 45.3, slightly higher than usual. Additionally, the mean P-R interval was 0.136 ± 0.037 seconds, and the mean Q-T interval was 0.356 ± 0.050 seconds; both values are within the normal. According to the results, ST depression (STD) was reported positive in 15 patients (5%), ST elevation (STE) in 38 patients (12.5%), and T. Invert in 29 patients (9.6%).

Table 1. Demographic, laboratory, and clinical characteristics of patients (N = 303)

Variable		Mean or N	SD or %
Age, year **Mean ± SD:** 63.03 ± 15.21	≤ 50 years	59	19.5%
	51 – 70 years	149	49.2%
	≥ 71 years	95	31.4%
Sex	Female	177	58.4%
	Male	126	41.6%
Diagnosis	Atherosclerosis	102	33.7%
	Heart failure	63	20.8%
	Cardiac arrest	105	34.7%
	Embolism and	33	10.9%
Hospitalization (day)		8.53	7.57
Progress	Deceased	229	75.6%
	Recovery	74	24.4%
Underlying disease			
HLP	No	192	63.4%
	Yes	111	36.6%
MI	No	255	84.2%
	Yes	48	15.8%
CVA	No	249	82.2%
	Yes	54	17.8%
DM	No	160	52.8%
	Yes	143	47.2%
HTN	No	172	56.8%
	Yes	131	43.2%
IHD	No	256	84.5%
	Yes	47	15.5%
CHF	No	272	89.8%
	Yes	31	10.2%
CABG	No	273	90.1%
	Yes	30	9.9%
ICU add	No	113	83.7%
	Yes	22	16.3%
Intubation	No	228	75.2%
	Yes	75	24.8%
Troponin T	Negative	287	94.7%
	Positive	16	5.3%
CPK		158.60	229.04
CK.MB		41.74	71.07
EF		30.41	22.69
SpO_2		87.56	11.28
BP		120.26	26.32
RR		21.33	3.45
pulse rate		91.85	16.22
PR interval		0.136	0.037
QT interval		0.356	0.050
STD	No	288	95.0%
	Yes	15	5.0%
STE	No	265	87.5%
	Yes	38	12.5%
T. Invert	No	274	90.4%
	Yes	29	9.6%

SD: Standard deviation; HLP: Hyperlipidemia; MI: myocardial infarction; CVA :cerebrovascular accident; DM: diabetes mellitus; HTN: hypertension; IHD: ischemic heart diseas; CHF: coronic heart failure; CABG: coronary artery bypass graft; CPK: creatine phosphokinase; CK.MB:creatine kinase mb; EF:ejection fraction; SpO_2: oxygen saturation; BP: blood pressure; RR: respiratory rate; STD: ST depression; STE: ST elevation

The results of tables 2 and 3, in order to investigate the relationship between PM and diagnostic factors in patients, showed that the relationship between disease progression and troponin T with PM2.5 was significant ($P < 0.05$). Also, according to the results, the mean PM2.5 in days with patients who died was significantly higher ($P = 0.022$), and the mean PM2.5 in days with patients with positive troponin T was also significantly higher ($P < 0.001$).

63.03 ± 15.21. 75.6% of patients died. This was likely because many of the patients were older and had co-existing COVID-19 and heart disease, all of which are significant risk factors for mortality. The study of the relationship between particulate matter and clinical and laboratory diagnostic factors in cardiovascular patients with COVID-19 showed that only the relationship between disease progression and troponin T with PM2.5 was significant, and

Table 2. Association between PM10 and PM2.5 with laboratory and clinical characteristics of patients

Variable		PM 10		PM 2.5	
		Mean (SD)	p	Mean (SD)	p
Diagnosis	Atherosclerosis	107.72 (128.38)	0.649	19.77 (18.01)	0.068
	Heart failure	139.99 (172.82)		25.69 (17.27)	
	Cardiac arrest	119.67 (129.91)		25.70 (14.58)	
	Embolism and phlebitis	95.82 (76.45)		19.92 (15.24)	
Progress	Deceased	126.81 (148.11)	0.054	24.60 (17.12)	**0.022**
	Recovery	86.89 (70.40)		18.88 (13.25)	
Underlying disease					
HLP	No	107.38 (99.18)	0.126	21.75 (16.28)	0.061
	Yes	134.35 (177.34)		25.75 (16.57)	
MI	No	119.38 (141.34)	0.125	23.08 (16.46)	0.614
	Yes	111.33 (106.04)		24.49 (16.73)	
CVA	No	121.37 (142.33)	0.643	23.72 (16.615)	0.427
	Yes	103.87 (103.75)		21.62 (15.96)	
DM	No	116.00 (110.75)	0.063	24.55 (16.85)	0.200
	Yes	120.35 (160.88)		21.86 (15.98)	
HTN	No	113.36 (125.42)	0.549	23.41 (15.79)	0.927
	Yes	123.76 (147.95)		23.21 (17.38)	
IHD	No	120.10 (144.66)	0.367	23.50 (16.67)	0.669
	Yes	105.09 (55.63)		22.23 (15.44)	
CHF	No	113.90 (118.90)	0.164	23.61(16.64)	0.411
	Yes	153.10 (236.24)		20.80 (15.04)	
CABG	No	114.86 (138.76)	0.297	22.77 (16.57)	0.124
	Yes	143.79 (106.44)		28.03 (15.14)	
ICU add	No	122.42 (176.13)	0.640	22.17 (16.59)	0.309
	Yes	114.32 (89.90)		24.30 (16.38)	
Intubation	No	123.00 (147.43)	0.297	23.72 (16.53)	0.499
	Yes	101.88 (87.25)		22.07 (16.39)	
Troponin T	No	85.68 (83.93)	0.399	8.06 (10.66)	**< 0.001**
	Yes	119.63 (137.76)		24.09 (16.36)	
STD	No	118.67 (138.75)	0.729	23.02 (16.17)	0.227
	Yes	104.70 (46.90)		28.70 (21.47)	
STE	No	118.19 (138.59)	0.950	23.71 (16.79)	0.296
	Yes	116.51 (113.85)		20.30 (13.71)	
T.Invert	No	117.95 (142.33)	0.988	23.20 (16.76)	0.736
	Yes	118.36 (53.87)		24.35 (14.08)	

DISCUSSION

This study investigated the relationship between particulate matter and electrocardiographic parameters, SpO2, creatine kinase-MB, and troponin T in cardiovascular patients with COVID-19. According to the results, 58.5% of patients were female, and the mean age of the patients was

no significant relationship was observed with particulate matter in other factors. Also, according to the results, the mean PM2.5 on days related to deceased patients was significantly higher than on days related to recovered patients.

A study by Karimi et al. in 2022 found that for every 10 $\mu g/m^3$ increase in PM2.5 and PM10, COVID-19

Table 3. Association between PM10 and PM2.5 with clinical and laboratory diagnostic factors

Variable	PM_{10}		$PM_{2.5}$	
	r	P	r	P
CPK	- 0.030	0.688	-0.054	0.468
CK.MB	-0.023	0.771	-0.093	0.243
EF	-0.021	0.742	-0.004	0.948
SpO_2	-0.041	0.524	-0.008	0.896
BP	-0.035	0.584	-0.020	0.748
RR	-0.002	0.980	-0.051	0.425
pulse rate	0.037	0.557	-0.019	0.771
PR interval	0.077	0.239	-0.072	0.268
QT interval	0.001	0.997	-0.078	0.232

r: Pearson correlation coefficient; CPK: Creatine phosphokinase; CK.MB: Creatine kinase mb; EF: Ejection fraction; SpO_2: Oxygen saturation; BP: Blood pressure; RR: Respiratory rate; STD: St depression; STE: St elevation

hospitalizations increased by 8.5% and 4.8%, respectively. Additionally, a 10 µg/m³ rise in PM2.5 was associated with a 5.6% increase in COVID-19 mortality [10]. Curtis et al., in their 2021 study, also stated that it is estimated that a 1 µg/m3 increase in outdoor PM2.5 increases the rate of COVID-19 by 0.22 to 8%. The exact ways air pollutants contribute to an increase in COVID-19 infections are not entirely understood. However, these effects are likely linked to the inflammation, oxidation of the tissues, and changes in angiotensin-converting enzyme 2 levels in tissues [11]. Poppe et al., found that exposure to environmental particulate matter at levels of 10 µg/m³ increased the risk of acute ischemic coronary events by four and a half times, especially in patients with coronary artery disease who had undergone angiography. They concluded that short-term exposure to such pollution can trigger acute coronary events in these individuals [12]. Rich et al. showed that the induction of myocardial infarction by environmental particulate matter increases, and there is a greater chance of myocardial infarction associated with increased PM2.5 concentrations [13]. Setti's 2020 study indicated a link between PM10 and PM2.5 exposure and increased COVID-19 cases and mortality. Therefore, air pollution may contribute to the spread of COVID-19 [14]. Our study also found that mean PM2.5 was significantly higher in patients with positive troponin T. Aghajani et al., (2022) conducted a retrospective study, which indicated that hospitalized COVID-19 patients with abnormal troponin I levels have a 67% higher risk of death compared to those with normal troponin I levels [15]. A systematic review conducted by An et al., (2021) found that patients with elevated levels of creatine kinase-MB, myoglobin, cardiac troponin T, cardiac troponin I, high-sensitivity cardiac troponin I, and high-sensitivity cardiac troponin T were associated with greater disease severity in COVID-19 infections [16]. In a 2021 study conducted by Zhang, researchers examined the short-term effects of PM2.5 on cardiac troponin T (cTnT) in participants who underwent cardiac catheterization. For each interquartile range increase of 7.6 µg/m³ in PM2.5 from the previous day, cTnT concentrations rose by 7.7% [17].

A study by Wyatt et al., in 2022 found that a 10 µg/m³ increase in PM2.5 levels measured three days before to troponin I testing was linked to a 0.06 ng/mL rise in troponin I levels. The analysis revealed that this association was most pronounced among patients who were men, white, and residing in less urban areas [18]. In our study, we measured the relationship between PM and troponin T, and we found no correlation between PM and gender. Additionally, different urban areas were not compared.

Limitations of this study include a small sample size, a lack of information on PM concentrations on some days, a lack of comparison of data between polluted and non-polluted days, incompleteness of some patient information, the ecological nature of the exposure assignment, the lack of control for confounding variables, and the potential for underpowered subgroup analyses, which may affect the conclusions. Additionally, one limitation of this study is that it was conducted in a small area in southwestern Iran and therefore cannot be generalized to other communities. Future studies should examine a larger sample of patients in broader societies with different climates and compare factors on polluted and non-polluted days to reach more accurate conclusions.

CONCLUSION

The results showed that the mean PM2.5 concentration on days associated with deaths was significantly higher than on days associated with recovery. This significance suggests that the observed difference between the mean PM2.5 concentration in these two groups is likely, not random, and can be considered a factor associated with health outcomes. There was also a relationship between troponin T and the

mean PM2.5 concentration, indicating that the mean PM2.5 concentration on days associated with troponin positivity was significantly higher than that associated with troponin negativity. This significance suggests that higher PM2.5 levels are associated with a second troponin positivity. These findings suggest that exposure to higher PM2.5 levels may be associated with an increased mortality risk compared with recovery. However, further studies controlling for confounding variables must confirm a causal relationship.

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Ethical Approval: This study received approval from the Ethics Committee of Abadan University of Medical Sciences (Ethical Approval ID: IR.ABADANUMS.REC.1402.017).

Conflict of interest: The authors confirm that there are no conflicts of interest.

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