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Association between monocyte/HDL cholesterol ratio and air pollution in a highly polluted region: A disease-specific analysis

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Abstract

The monocyte/HDL cholesterol ratio (MHR) is increasingly recognized as a potential marker of inflammation, oxidative stress, and endothelial dysfunction, despite the lack of standardized reference values. This study investigates the association between air pollution and MHR and its relationship with chronic diseases. A retrospective cohort study was conducted with 600 patients admitted to Igdir State Hospital. Patients were grouped by Particulate Matter 10 (PM10) exposure levels (≥50 μg/m³ vs <50 μg/m³). Laboratory data and disease diagnoses were recorded and analyzed. MHR was significantly higher in patients with atherosclerotic cardiovascular disease (ASCVD) ($p<0.003$) and lower in patients with diabetes mellitus (DM) ($p<0.003$) on days with PM10≥50 μg/m³. Similar trends were observed on low-pollution days (PM10<50 μg/m³), with a significant positive association between MHR and ASCVD ($p<0.012$) and cerebrovascular accident (CVA) ($p<0.027$). A significant correlation was also found between MHR and HbA1c levels ($p<0.001$). MHR is positively associated with cardiovascular risk and may serve as a low-cost, accessible marker of inflammation in clinical practice.

Keywords: High-density lipoprotein cholesterol, air pollution, chronic diseases

Introduction

Monocytes and macrophages, which are formed from precursor cells, damage the vascular endothelial structure during inflammation, resulting in the synthesis of prooxidant and proinflammatory cytokines (1). High-density lipoprotein (HDL) cholesterol is an important mediator with antioxidant, antithrombotic, and anti-inflammatory properties as well as eliminating the harmful effects of low-density lipoprotein (LDL) cholesterol [1].

HDL helps lower inflammation and fights infections by changing how immune cells work, widening blood vessels, and supporting the barrier function of blood vessel linings [2]. HDL has a structure consisting of apolipoproteins and contains proteinase inhibitors [3]. Because of this structure, HDL cholesterol is linked to inflammation and helps lessen the negative effects of LDL cholesterol by reducing its harmful impact on blood vessel linings and stopping oxidation.

There are studies in the literature suggesting a negative relationship between HDL and monocytes. In addition, its anti-inflammatory properties disappear in patients with coronary artery disease (CAD) at low HDL concentrations [4]. In this context, HDL can be considered as a predictive marker for CAD.

Epidemiological studies and clinical trials have shown that air pollution causes toxic chemical exposure in the body, is one of the causes of preventable diseases and premature deaths, and increases cerebrovascular mortality and cardiovascular diseases, especially ischemia [5]. The relationship between myocardial infarction (MI), atherosclerosis, and coronary artery diseases in air pollution with an aerodynamic particle diameter value of 10 μg and PM10 value of 50 μg/m³ and above has been published in various articles [6–11].

According to data from the World Health Organization (WHO), Iğdır is ranked among the 10 most polluted cities in Europe in terms of air quality. A similar observation was supported by a previous study by Argun et al. (2019) [12], which examined urban factors contributing to air pollution in Iğdır.The aim of this study is to clearly investigate whether air pollution affects the monocyte/high-density lipoprotein (HDL) cholesterol ratio and how this

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association relates to specific chronic diseases, particularly cardiovascular and cerebrovascular conditions.

Material and Methods

Patients with cardiovascular and chronic diseases over the age of 18 years who applied to Igdir State Hospital between 20.10.2022 and 20.10.2023 were included in the study population. By taking aerodynamic particle diameter 10 μg (PM10) as the limit, the days with 50 and above were considered to have polluted air, and those below 50 were considered unpolluted air. The files of a total of 600 patients on these days were scanned from the hospital electronic system, and the patients' monocyte, HDL cholesterol, LDL cholesterol, and HbA1c values and disease diagnoses were determined. All procedures followed were carried out in line with the ethical standards of the committee responsible for human experiments (institutional and national) and the 1975 Declaration of Helsinki, which was revised in 2008. For this study, the Erzincan Binali Yıldırım University Clinical Research Ethics Committee has been approved by the presidency 1of the ethics committee with the decision numbered 03/13 in the session dated 13.10.2022 and numbered 03.

The PM10 data used in this study were obtained from official air quality monitoring stations operated by the Turkish Ministry of Environment, Urbanization, and Climate Change. These stations employ β-ray absorption and gravimetric methods for continuous measurement of particulate matter. Based on WHO and EU standards, a PM10 level of 50 μg/m³ was accepted as the threshold value. Days with PM10 levels ≥50 μg/m³ were classified as polluted, and those with <50 μg/m³ as clean. Seasonal distribution was not directly analyzed; however, PM10 levels in Iğdır are typically higher during winter months due to increased residential heating and lower atmospheric dispersion.

Patients under the age of 18, pregnant women, those diagnosed with familial hypercholesterolemia according to ICD-10 codes, and individuals with a history of hematological malignancy (acute myeloid leukemia, chronic myeloid leukemia, chronic lymphocytic leukemia, multiple myeloma, etc.) were excluded from the study.

Demographic parameters (age and gender) and laboratory parameters (monocyte count, HDL, monocyte/HDL cholesterol ratio (μL/mg/dL), LDL, and HbA1C) obtained from the emergency department, outpatient clinic, and intensive care unit were analyzed for both groups (Group 1: n=300, <50 μg/m3 PM10 and group 2: n=300, 50≥μg/m³ PM10). Patients were also examined for comorbidities and their relationship to the monocyte/HDL cholesterol ratio in both arms.

Individuals over the age of 18 years who applied to the emergency department, outpatient clinic, or intensive care unit of our institution and were also diagnosed with chronic diseases such as cardiovascular events, ischemic stroke, chronic bronchitis, asthma, chronic obstructive pulmonary disease, diabetes mellitus, hyperlipidemia, and congestive heart failure were included in the study.

Although patients were included regardless of their presenting complaints, efforts were made to analyze only laboratory values and disease diagnoses that were available consistently. However, since the patients were admitted for various reasons, potential confounding factors such as acute infections or comorbidities may have affected MHR levels.

Statistical Analysis

The data were analyzed using the IBM SPSS Statistics 25 © (Copyright SPSS Inc. 1989, 2017) software. The conformity of continuous variables to normal distribution was evaluated by the Kolmogorov-Smirnov test. Pearson Chi-square and Fisher's exact test were used in the analysis of categorical variables, and Yates correction was utilized. In independent two-group mean comparisons, the student t-test was performed when parametric test assumptions were met, and the Mann-Whitney U test was performed when these assumptions were not met; since parametric test assumptions were not met in more than two independent group comparisons, the Kruskal-Wallis test and post hoc Bonferroni correction were performed. A value of $p<0.05$ was considered statistically significant.

Result

The differences between the PM10 (particulate matter 10) group measured below 50 μg/m³ and the group measured at 50 μg/m³ and above were not statistically significant in terms of age, gender, monocyte, HDL cholesterol, monocyte/HDL cholesterol ratio, LDL, and HbA1C values ($p>0.05$) (Table 1).

When the distribution of diseases according to air pollution levels was analyzed, the incidence rates of bronchiolitis, arterial embolism, atherosclerotic cardiovascular disease (ASCVD), Diabetes Mellitus (DM), hypertension (HT), pneumonia, asthma,chronic obstructive pulmonary disease (COPD), cerebrovascular accident (CVA), upper respiratory tract infection (URTI), pulmonary embolism, and myocardial infarction (MI) did not differ statistically significantly according to air pollution level (Table 2).

On days with PM10 levels ≥50 μg/m³, the monocyte/HDL cholesterol ratio differed significantly in relation to atherosclerotic cardiovascular disease (ASCVD) and diabetes mellitus (DM). The median MHR was 13.42 (4.66–51.43) in 65 patients with ASCVD, compared to 11.37 (2.78–53.33) in 235 patients without ASCVD. In patients with DM, the median MHR was 10.83 (3.86–27.50), while it was higher—13.33 (2.78–53.33)—in those without diabetes. No statistically significant differences in MHR were observed among the other disease groups

On days when PM10 levels were below 50 μg/m³, the monocyte/HDL cholesterol ratio was significantly associated with atherosclerotic cardiovascular disease (ASCVD), diabetes mellitus (DM), and cerebrovascular accident (CVA). The median MHR was 13.14 (4.25–31.38) in patients with ASCVD and 11.17 (2.63–48.89) in those without. Similarly, the median MHR was lower in diabetic patients [10.77 (3.64–42.94)] than in non-diabetics [12.30 (2.63–48.89)]. For patients with CVA, the median MHR was 15.87 (9.67–36.60), compared to 11.39 (2.63–48.89) in those without CVA. The MHR did not show statistically significant variation in relation to other disease groups.

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The HbA1c values of the patients were divided into two groups, below 6.5 and above 6.5, and the monocyte/HDL cholesterol ratios were analyzed according to these groups and air pollution level. According to the analyses, the median monocyte/HDL ratio was calculated as 10.14 (4.16-24.81) in those whose HbA1c values were below 6.5 and 13.23 (3.75-31.25) in those whose HbA1c values were above 6.5 on days with a PM10 value below 50 µg/m3, and these differences were found to be statistically significant (p<0.001). The median monocyte/HDL ratio was found to be 11.37 (4.44-27.50) for people with HbA1c values below 6.5 and 11.55 (3.86-51.43) for those with HbA1c values above 6.5 on days when the PM10 value was 50 µg/m³ or higher, and these differences were statistically significant (Table 5).

The relationship between acute diseases (e.g., URTI, bronchiolitis, pneumonia, MI) and MHR was assessed in patients with chronic conditions (e.g., COPD, asthma, ASCVD, CVA, DM, HT). On days with PM10 <50 µg/m³, the median MHR was 11.61 (3.64–42.94) in those without acute illness, and 13.89 (5.41–24.47) in those with acute illness (p=0.213). On days with PM10 ≥50 µg/m³, the median MHR was 11.49 (2.78–53.33) in patients without acute illness and 11.66 (4.39–51.43) in those with acute illness (p=0.616). These differences were not statistically significant (Table 6).

Table 1. Relationship between air pollution level and independent variables

Variables	PM10 (µg/m³)			p
	<50 µg/m³ (n=300)	≥50 µg/m³ (n=300)	General (n=600)	
Age (years)	57.93±13.39 59 (20-102)	55.71±13.58 58 (18-92)	56.83±13.47 59 (18-102)	0.995
Gender				
Female	152 (50.7)	152 (50.7)	304 (50.7)	0.999
Male	148 (49.3)	148 (49.3)	296 (49.3)	
Monocyte	510.23±162.07 490 (140-1430)	517.00±173.36 470 (200-1320)	513.61±167.70 490 (140-1430)	0.836
HDL	43.30±12.15 41 (9-95)	43.41±12.27 42 (14-88)	43.35±12.20 42 (9-95)	0.924
Monocyte/HDL	12.95±6.25 11.72 (2.63-48.89)	13.12±6.82 11.75 (2.78-53.33)	13.04±6.54 11.75 (2.63-53.33)	0.969
LDL (n=6/10)	106.33±35.23 104 (63-158)	123.76±39.75 126 (42-174)	117.22±37.92 110 (42-174)	0.368
HbA1c (n=146/163)	7.53±2.09 6.7 (4.5-13.8)	7.54±2.19 6.9 (4.8-16.1)	7.54±2.14 6.7 (4.5-16.1)	0.889

Findings were given as mean ± SD, median (min-max), or n (% percentage). Mann-Whitney U test, Student t-test, Pearson Chi-square test

Table 2. Distribution of diseases by air pollution level

Variables	PM10 (µg/m³)			p
	<50 µg/m³ (n=300)	≥50 µg/m³ (n=300)	General (n=600)	
Bronchiolitis	7 (2.3)	5 (1.7)	12 (2.0)	0.771
Arterial embolism	1 (0.3)	2 (0.7)	3 (0.5)	0.999
ASCVD	80 (26.7)	65 (21.7)	145 (24.2)	0.153
DM	126 (42.0)	149 (49.7)	275 (45.8)	0.059
HT	95 (31.7)	92 (30.7)	187 (31.2)	0.791
Pneumonia	13 (4.3)	11 (3.7)	24 (4.0)	0.835
Asthma	10 (3.3)	5 (1.7)	15 (2.5)	0.296
COPD	6 (2.0)	7 (2.3)	13 (2.2)	0.999
CVA	14 (4.7)	19 (6.3)	33 (5.5)	0.474
URTI	23 (7.7)	30 (10.0)	53 (8.8)	0.314
Pulmonary embolism	1 (0.3)	2 (0.7)	3 (0.5)	0.999
MI	2 (0.7)	1 (0.3)	3 (0.5)	0.999

ASCVD: Arteriosclerotic cardiovascular disease, DM: Diabetes mellitus, HT: Hypertension, COPD: Chronic Obstructive Pulmonary Disease, CVA: Cerebrovascular Accident, URTI: Upper Respiratory Tract Infection, MI: Myocardial infarction, Pearson Chi-square and Fisher's Exact Test

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Table 3. Monocyte/HDL cholesterol ratio on days with PM10≥50 µg/m³ by diseases

Diseases	Situation	n	Mean±SD	Median	Min-Max	p
ASCVD	No	235	12.34±5.81	11.37	2.78-53.33	**0.003**
	Yes	65	15.94±9.14	13.42	4.66-51.43	
DM	No	151	14.51±8.19	13.33	2.78-53.33	**0.003**
	Yes	149	11.70±4.69	10.83	3.86-27.50	

ASCVD: Arterosclerotic cardiovascular disease, DM: Diabetes mellitus, monocyte/HDL cholesterol ratio on days with PM10≥50 µg/m³ by disease (only statistically significant results shown), Mann-Whitney U test

Table 4. Monocyte/HDL cholesterol ratio on days with PM10≥50 µg/m³ by diseases

Diseases	Situation	n	Mean±SD	Median	Min-Max	p
ASCVD	No	220	12.60±6.52	11.17	2.63-48.89	**0.012**
	Yes	80	13.92±5.37	13.14	4.25-31.38	
DM	No	174	13.64±6.57	12.30	2.63-48.89	**0.022**
	Yes	126	12.01±5.69	10.77	3.64-42.94	
CVA	No	286	12.76±6.12	11.39	2.63-48.89	**0.027**
	Yes	14	16.83±7.86	15.87	9.67-36.60	

ASCVD: Arterosclerotic cardiovascular disease, DM: Diabetes Mellitus, CVA: Cerebrovascular accident, monocyte/HDL cholesterol ratio on days with PM10<50 µg/mm3 by disease (only statistically significant results shown), Mann-Whitney U test

Table 5. Monocyte/HDL cholesterol ratio by air pollution and HbA1c values

Variables	n	Monocyte/HDL			p
		Mean±SD	Median	Min-Max	
Days with PM10<50 µg/m³					
HbA1c<6.5 (unit)	64	11.34±5.06	10.14	4.16-24.81	<0.001[a]
HbA1c≥6.5 (unit)	55	13.47±5.56	13.23	3.75-31.25	
Days with PM10≥50 µg/m³					
HbA1c<6.5 (unit)	74	12.05±4.84	11.37	4.55-27.50	<0.001[a]
HbA1c≥6.5 (unit)	64	12.37±6.79	11.55	3.86-51.43	

Kruskal-Wallis test, post hoc Bonferroni correction. [a]: All groups are different from one another (p<0.001). PM10: Particulate matter 10, HbA1c: Hemoglobin A1C

Table 6. Monocyte/HDL cholesterol ratio in those with a chronic disease according to air pollution and acute disease comorbidity

Variables	n	Monocyte/HDL			p
		Mean±SD	Median	Min-Max	
Days with PM10<50 µg/m³					
No acute diseases	233	12.74±5.88	11.61	3.64-42.94	0.213
Acute disease present	32	13.50±4.74	13.89	5.41-24.47	
Days with PM10≥50 µg/m³					
No acute diseases	236	12.85±6.40	11.49	2.78-53.33	0.616
Acute disease present	33	13.90±8.55	11.66	4.39-51.43	

Mann-Whitney U test. PM10: Particulate matter 10

Discussion

In our study, the monocyte/HDL cholesterol ratio (MHR) was found to be significantly higher in patients with atherosclerotic cardiovascular disease (ASCVD), regardless of air pollution levels. In patients with cerebrovascular accident (CVA), MHR was significantly elevated only on days with PM10 levels below 50 µg/m³. Additionally, patients with diabetes mellitus (DM) had significantly lower MHR values compared to non-diabetic individuals, both on polluted and clean air days.

In the literature, it has been reported that the monocyte/HDL cholesterol ratio may be a prognostic biomarker for oxidative stress and inflammation in cardiovascular diseases [1,13–15]. Stadler and Marsche stated that atherogenic dyslipidemia is associated with an increased risk of cardiovascular disease, and atherogenic dyslipidemia is associated with HDL [16]. Genceli et al. stated that a high monocyte/HDL cholesterol ratio can be used as a marker for the development of atherosclerosis and the prognosis of cardiovascular diseases [15]. In another study, Dolapoglu et al. reported that the monocyte/HDL cholesterol ratio

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increased in patients after coronary artery bypass graft (CABG) with a history of myocardial infarction (MI), but the predictive value of the monocyte/HDL cholesterol ratio was not significant in the preoperative and postoperative period of cardiovascular surgery [13]. In our study, the relationship between ASCVD and the monocyte/HDL cholesterol ratio was statistically positive in both the low-density PM10 group and the high-density PM10 group. Although this finding is consistent with previous studies emphasizing the relationship between monocyte/HDL cholesterol ratio and coronary artery diseases, this suggests that air pollution may not have an independent effect on monocyte/HDL cholesterol levels in these patients.

Kosovar highlighted that the monocyte/HDL cholesterol ratio can be used as an indicator of chronic inflammation in acute ischemic stroke patients [17]. In our study, a statistically significant difference in MHR was found between patients with and without cerebrovascular accident (CVA) on days when PM10 levels were below 50 μg/m³. However, no statistically significant association was observed on high pollution days. These findings suggest a potential link between air quality and MHR in CVA patients, particularly under low pollution conditions, but further studies are needed to confirm this relationship.

In a study, Chen et al. demonstrated that HDL underwent carbamylation modification and C-HDL increased endothelial cell adhesion of monocytes in individuals with cardiovascular disease and Type 2 Diabetes Mellitus (DM) comorbidity [18], indicating the potential pro-atherogenic role of C-HDL in atherosclerosis among Type 2 DM patients [18]. Even though there was no link between the monocyte/HDL cholesterol ratio and cardiovascular risk in people with diabetic neuropathy, Pence and Aktas found a significant connection between this ratio and cardiovascular risk in all patients, including those without diabetes [19]. Kan and Karaibrahimoglu found that the monocyte/HDL cholesterol ratio could help predict cardiovascular risk in patients with diabetic retinopathy [20]. In our study, a statistically significant difference in the monocyte/HDL cholesterol ratio was observed between patients with and without diabetes mellitus (DM) on both days with air pollution (PM10≥50 μg/m³) and days without air pollution (PM10<50 μg/m³).

Even though there was a negative relationship between monocyte/HDL cholesterol and PM10 levels below and above 50 μg/m³ in patients with diabetes, when these patients were compared based on their rising HbA1c levels, the monocyte/HDL cholesterol ratio actually increased as their HbA1c levels went up. More studies are needed on the effect of air pollution on the monocyte/HDL cholesterol ratio in patients diagnosed with DM.

Cakmak Karaaslan et al. reported that the monocyte/HDL cholesterol ratio could be used to predict negative outcomes and prognosis in patients with acute myocarditis [21]. Kalayci states that the monocyte/HDL cholesterol ratio has a similar diagnostic value to neutrophil/lymphocyte ratios (NLR) and platelet/lymphocyte ratios (PLR) in dipper and non-dipper hypertension cases in terms of patients with cardiovascular diseases and does not display a significant difference between the two groups [22]. In our study, it was determined that the monocyte/HDL cholesterol ratio in patients with and without HT did not show a statistically significant difference in both groups.

In our study, the monocyte/HDL cholesterol ratio showed a statistically significant association with ASCVD in both PM10<50 μg/m³ and PM10≥50 μg/m³ groups. Additionally, a statistically significant increase in the ratio was observed in patients with CVA on days with PM10<50 μg/m³. These findings support the potential role of the monocyte/HDL ratio as a biomarker for endothelial dysfunction and inflammation in ASCVD and CVA. In patients with diabetes mellitus, the monocyte/HDL cholesterol ratio was significantly lower compared to non-diabetic individuals on both clean-air and polluted days. This inverse relationship was statistically significant in both PM10 groups. Additionally, MHR levels tended to increase in line with rising HbA1c values, although further studies are needed to better understand this association.

Although this research includes a large number of participants, its main limitation is its single-center, retrospective, and cross-sectional design and limited budget. However, our results support the studies in the literature suggesting that there is a relationship between monocyte/HDL cholesterol and ASCVD. In addition, since there are not many studies on air pollution and its effect on inflammation markers and monocyte/HDL cholesterol ratio, it is important in terms of pioneering studies in this field. In this respect, our research has made an important contribution to the literature.

Conclusion

According to the study's findings, the monocyte/HDL ratio is a straightforward, inexpensive, and easy-to-apply indicator of cardiovascular risk for primary care physicians and those living in rural areas with inadequate laboratory resources. However, large-scale, multicenter studies are needed to explain the full role of air pollution in monocyte/HDL cholesterol ratio and inflammation.

While the study supports the association between MHR and cardiovascular risk, as well as its variation with air pollution, the results should be interpreted with caution due to potential confounding variables related to patient selection and clinical context.

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Conflict of Interests

The authors declare that there is no conflict of interest in the study.

Financial Disclosure

The authors declare that they have received no financial support for the study.

Ethical Approval

This study was approved by the clinical research ethics committee of the Erzincan Binali Yıldırım University. (Approvel Date: 13.10.2022), (Approvel number: E-26447783-050.01.04-207585).

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