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ORIGINAL

Influence of meteorological conditions on hospital admission in patients with acute coronary syndrome with and without ST-segment elevation: Results of the AIRACOS study☆

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KEYWORDS
Meteorological variables;
Air pollution;
Population exposure;
Acute myocardial infarction

Abstract
Objective: Evaluate whether the meterological parameters affecting revenues in patients with ST-segment and non-ST-segment elevation ACS.
Design: A prospective cohort study was carried out.
Setting: Coronary Care Unit of Hospital Universitario de Canarias.
Patients: We study a total of 307 consecutive patients with a diagnosis of ST-segment and non-ST-segment elevation ACS. We analyze the average concentrations of particulate smaller than 10 and 2.5 μm diameter, particulate black carbon, the concentrations of gaseous pollutants and meteorological parameters (wind speed, temperature, relative humidity and atmospheric pressure) that were exposed patients from one day up to 7 days prior to admission.
Interventions: None.
Variables of interest: Demographic, clinical, atmospheric particles, concentrations of gaseous pollutants and meterological parameters.
Results: A total of 138 (45%) patients were classified as ST-segment and 169 (55%) as non-ST-segment elevation ACS. No statistically significant differences in exposure to atmospheric particles in both groups. Regarding meteorological data, we did not find statistically significant

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differences, except for higher atmospheric pressure in ST-segment elevation ACS (999.6 ± 2.6 vs. 998.8 ± 2.5 mbar, *p*=.008). Multivariate analysis showed that atmospheric pressure was significant predictor of ST-segment elevation ACS presentation (OR: 1.14, 95% CI: 1.04–1.24, *p*=.004).
Conclusions: In the patients who suffer ACS, the presence of higher number of atmospheric pressure during the week before the event increase the risk that the ST-segment elevation ACS.

PALABRAS CLAVE
Variables meteorológicas;
Contaminación atmosférica;
Exposición poblacional;
Infarto agudo de miocardio

Influencia de las condiciones meteorológicas en el ingreso hospitalario en pacientes con síndrome coronario agudo con y sin elevación del segmento ST: resultados del estudio AIRACOS

Resumen
Objetivo: Evaluar si los parámetros meteorológicos influyen en los ingresos de pacientes con síndrome coronario agudo (SCA) con y sin elevación del ST.
Diseño: Cohorte prospectiva.
Ámbito: Unidad Coronaria del Hospital Universitario de Canarias.
Pacientes: Se estudió un total de 307 pacientes consecutivos con el diagnóstico de SCA con y sin elevación del ST. Analizamos las concentraciones medias de partículas con tamaño inferior a 10 y 2,5 μm de diámetro, partículas de carbono negro, concentraciones de gases contaminantes y los parámetros meteorológicos a los que estuvieron expuestos los pacientes desde el día anterior hasta 7 días previos al ingreso.
Intervenciones: Ninguna.
Variables de interés principales: Demográficas, clínicas, partículas atmosféricas, contaminantes en fase gas y parámetros meteorológicos.
Resultados: Del total, 138 (45%) pacientes fueron clasificados como SCA con elevación del ST y 169 (55%) sin elevación del ST. No encontramos diferencias estadísticamente significativas en la exposición a partículas atmosféricas entre ambos grupos. Respecto a los datos meteorológicos, no encontramos diferencias estadísticamente significativas, a excepción de una mayor presión atmosférica en el SCA con elevación del ST (999,6 ± 2,6 vs. 998,8 ± 2,5 mbar, p=0,008). El análisis multivariante mostró que la presión atmosférica fue predictor significativo de presentación del SCA con elevación del ST (OR: 1,14 IC 95%: 1,04 a 1,24; p=0,004).
Conclusiones: En los pacientes que sufren un SCA, la presencia de cifras más elevadas de presión atmosférica durante la semana previa al evento incrementa el riesgo de que dicho SCA sea con elevación del ST.

Introduction

Many epidemiological studies point to the existence of an association between exposure to air pollution and harmful health effects, resulting in a significant increase in morbidity-mortality.[1] Acute coronary syndrome (ACS) constitutes an important public health problem in Spain.[2,3] Although different studies have shown certain meteorological variables such as atmospheric pressure, temperature or wind to exert the greatest impact upon the incidence of acute myocardial infarction, the underlying cause–effect relationship and the reasons for the observed seasonal variations remain unclear.[4,5]

The present study explores the possible association between meteorological parameters and hospital admission in patients with (STE-ACS) and without ST-segment elevation ACS (NSTE-ACS).

Patients and methods

Study population

The AIRACOS (AIR and Acute COronary Syndrome) is an observational study that has been described in detail elsewhere.[6] In brief, the AIRCOS was carried out in the Canarias University Hospital Complex (Canary Islands, Spain), which provides medical care for the northern part of the island of Tenerife, with a recruitment population of 343,025 inhabitants. The study included patients with a diagnosis of STE-ACS and NSTE-ACS. Specifically, STE-ACS was defined by the presence of compatible symptoms, persistent (>20 min) ST-segment elevation ≥1 mm on at least two adjacent leads or in the presence of left bundle block, presumably of new onset, and cardiac troponin I elevation ≥0.5 ng/ml (cut-off point ≥0.5 ng/ml for the diagnosis of acute myocardial

infarction [AMI]; immune reagents of the Vitros 5100 system from Orthoclinical Diagnostics, USA). In turn, NSTE-ACS was defined by the presence of compatible symptoms, cardiac troponin I elevation $\geq$0.5 ng/ml or dynamic ST-segment changes (descent $\geq$1 mm or non-persistent elevation on at least two adjacent leads).[2]

All the patients included in the AIRACOS signed the corresponding informed consent form. The study protocol abided with the guidelines of the Declaration of Helsinki (1975), as reflected in the prior approval obtained from the Clinical Research Ethics Committee of the center. Likewise, the present study has been inscribed in and warranted by the clinical trials registry (www.clinicaltrials.gov: NCT01799148). The main objectives of the AIRACOS were to determine whether there is an association between air pollution levels and inflammatory and oxidative stress markers, and to explore their correlation to the prognosis of patients with ACS.

The data presented herein correspond to a substudy of the AIRACOS survey, in which the analysis of atmospheric pressure in relation to ACS was not a pre-specified objective. This is therefore a post hoc and hypothesis-generating analysis. We included those patients that met all the inclusion criteria and none of the exclusion criteria.[6] The population of this substudy comprised a total of 307 patients.

Methodology of the air pollution data

The air pollutant measurements were made in a single location representative of the urban background conditions of a metropolitan area (Santa Cruz de Tenerife).[7,8] Previous studies have shown that the pollutants measured simultaneously at different points in the northern part of the island of Tenerife are closely correlated, due to the dominant influence of the meteorological conditions.[9]

The pollutants whose environmental air concentrations are limited by current legislation were monitored using the European reference techniques[10] (European Directive on air quality 2008/50/EC, Spanish Royal Decree [RD] 102/2011): particles with an aerodynamic diameter of under 10 and 2.5 μm (gravimetry and beta-attenuation), nitrogen dioxide (IR chemiluminescence), sulfur dioxide (UV fluorescence) and ozone (UV absorption). In addition, a multiple-angle absorption photometer (VIS) was used to determine the concentrations of particulate black carbon–the environmental air concentrations of which have not yet been limited by current legislation but are of great scientific interest due to their potential effects upon health.[8,9] The meteorological variables were recorded by means of standard electronic sensors and techniques used in meteorology (temperature, relative humidity, air speed and atmospheric pressure determined with a thermometer, hygrometer, anemometer and barometer, respectively). All the equipment recorded data with a time resolution of one minute.

Study variables

A number of clinical variables were recorded in each patient, including coronary risk factors, age, gender, body mass index (BMI), previous vascular disease, and medication at hospital discharge. The following laboratory test parameters were also documented: blood count with leukocyte formula, renal function, total cholesterol, triglycerides, troponin I, C-reactive protein and malondialdehyde. The number of diseased coronary vessels were recorded from the coronariographic studies, and the left ventricular ejection fraction was registered.

With regard to the air pollutants and meteorological variables, each patient was associated to the mean daily exposure values (24 h) recorded from the day before (lag 1) to day 7 (lag 7) prior to admission.[6]

Statistical analysis

A time series analysis was made with interaction between contamination and the modifier (STE-ACS and NSTE-ACS). Continuous variables are reported as the mean $\pm$ standard deviation (SD), and as the median (interquartile range [IQR]) in the case of a non-normal distribution. Qualitative variables in turn are presented as absolute frequencies and percentages. The baseline characteristics in the two patient groups were compared by means of the chi-squared test for categorical variables. In the case of continuous quantitative variables with a normal distribution, we used the Student's *t*-test, or the equivalent nonparametric tests, as applicable (Mann–Whitney *U*-test or Kruskal–Wallis test). A multivariate analysis was performed based on a binary logistic regression model in which the dependent variables were STE-ACS (value 1) and NSTE-ACS (value 0), and the independent variables were those parameters yielding a p-value <0.05 in the univariate analysis. The results are expressed with the odds ratio (OR), with the 95% confidence interval (95%CI). Statistical significance was considered for $p < 0.05$ in all cases. The SPSS version 20.0 statistical package (Chicago, IL, USA) was used throughout.

Results

Of the 307 patients participating in the study, 138 (45%) belonged to the STE-ACS group and 169 (55%) to the NSTE-ACS group. In the STE-ACS group, 51 patients suffered acute thrombotic occlusion of the anterior descending artery, 55 of the right coronary artery, and 32 of the circumflex artery. The mean age was 63 $\pm$ 12 years, and 76.2% of the subjects were males (Table 1). The characteristics of both groups are summarized in Table 2. Significant differences were observed between the two groups in terms of age and the number of diseased coronary vessels–the patients in the NSTE-ACS group being older, with a larger proportion of two and three diseased vessels, and lower troponin I levels.

The meteorological data, gaseous pollutants and atmospheric particles are reported in Table 3. The meteorological variables showed the patients in the STE-ACS group to be exposed to higher atmospheric pressure than those in the NSTE-ACS group. On comparing the concentrations of the gaseous pollutants with exposure in both groups, the patients with STE-ACS were seen to be less exposed to sulfur dioxide. With regard to exposure to the different atmospheric particles, no statistically significant differences were observed between the two groups.

Table 1 Baseline characteristics of the study population.

	(n = 307)
Age, years	63 ± 12
Gender (males)	234 (76.2)
Body mass index (kg/m²)	28.5 ± 4
Previous cerebrovascular event	13 (4.2)
Clinical condition	
STE-ACS	138 (45)
NSTE-ACS	169 (55)
Cardiovascular risk factors	
Arterial hypertension	173 (64.5)
Smoking	129 (56.4)
Hypercholesterolemia	187 (60.9)
Diabetes mellitus	98 (31.9)
Medication at discharge	
Aspirin	307 (100)
Clopidogrel	283 (92.1)
Beta-blockers	273 (88.9)
ACEIs/ARA-II drugs	217 (70.7)
Statins	307 (100)
Antidiabetic medication	98 (31.9)
Coronary angiography	
Normal coronary arteries	15 (4.9)
Single-vessel lesion	150 (48.9)
Two-vessel lesion	88 (28.7)
Three-vessel lesion	54 (27.6)
LVEF (%)	57 ± 10
Blood test parameters upon admission	
Hemoglobin (mg/dl)	14.4 ± 7.4
Hematocrit (%)	41.6 ± 4.7
Leukocytes (10^9/l)	11.8 ± 3
Neutrophils (10^9/l)	7.1 ± 1.3
Creatinine (mg/dl)	0.9 ± 0.5
Troponin I (ng/ml)	31.1 ± 22.1
Total cholesterol (mg/dl)	175 (147–201)
Triglycerides (mg/dl)	135 (107–174)
C-reactive protein (mg/dl)	8.3 (5–17.3)
Malondialdehyde (nmol/l)	2.2 (1.7–2.9)

ARA-II, angiotensin-II receptor antagonists; LVEF, left ventricular ejection fraction; ACEIs, angiotensin-converting enzyme inhibitors; STE-ACS, acute coronary syndrome with ST-segment elevation; NSTE-ACS, acute coronary syndrome without ST-segment elevation.
Values expressed as n (%), mean ± standard deviation or median [interquartile range].

In a multivariate model (Table 4), atmospheric pressure (OR: 1.14; 95%CI: 1.04–1.24; $p = 0.004$) was found to be an independent predictor of the presentation of STE-ACS.

Discussion

To our knowledge, this is the first study in Spain to analyze the relationship between meteorological conditions and hospital admission due to STE-ACS and NSTE-ACS. The results of our study, which serve to generate hypotheses, indicate that patients with STE-ACS have been exposed to higher atmospheric pressure values during the previous days.

The impact of atmospheric pressure upon cardiovascular disease has been studied in different European countries, with contradictory results. In effect, while some studies have found no association between atmospheric pressure and the incidence of acute myocardial infarction,[5,11–13] other studies have indeed reported a correlation.[14,15]

A prospective study[15] involving a 10-year follow-up of 257,000 males between 25 and 64 years of age detected a V-form relationship between atmospheric pressure and the rate of coronary episodes – the incidence being lowest when the atmospheric pressure was 1016 mbar, i.e., approximately the separating point between high and low pressures. This study[15] was carried out in the city of Lille (France), where in the course of its 10-year duration the highest recorded atmospheric pressure was 1044 mbar, the minimum pressure 991 mbar, and the mean pressure 1017. According to the results of the study, an increase of 10 mbar above a pressure of 1016 mbar was associated to an 11% increase in total coronary episodes, an 18% increase in coronary deaths, a 7% increase in myocardial infarctions, and a 30% increase in relapse rate. In turn, with a 10 mbar decrease below 1016 mbar, these increments were found to be 12%, 13%, 8% and 30%, respectively. In our study, with each mbar increase in atmospheric pressure during the week before the event, the risk of admission due to STE-ACS versus NSTE-ACS was found to increase 14%.

Differences in climate, orography and data analyzing methodology must be taken into account on comparing our results with those of the study conducted in Lille. Firstly, the present study was carried out in Tenerife, corresponding to a subtropical region with practically permanent high pressures due to the Azores anti-cyclonic effect. In contrast, Lille is characterized by greater meteorological variability, with the alternation of winter and summer high pressure periods due to anti-cyclonic situations versus low pressure periods in spring and autumn due to the passing of showers typical of such middle latitudes.[15] Accordingly, the pressure variation range differs between the two studies: 28 mbar in our study versus 53 mbar in the study published by Danet et al.[15] Secondly, in our study the orography is abrupt, with a greater time variability in atmospheric pressure than in flatter regions, as is the case of the city of Lille. Lastly, in our study we used the mean pressures during the 7 days prior to patient admission, while Danet et al. employed data recorded during 10 years.[15]

The physiopathological mechanisms of STE-ACS and NSTE-ACS may be superimposable. Both conditions are characterized by atheroma plaque rupture, fissure or erosion, with the generation of an overlying thrombus–the latter being occlusive in the case of STE-ACS and non-occlusive or intermittently occlusive in the case of NSTE-ACS.[2]

The results of our study serve to generate hypotheses; as a result, such small differences in atmospheric pressure may at least in part have clinical consequences based on the concept of extra-arterial dynamic pressure.[16] This concept was introduced by Saul[16] as pressure proportional to atmospheric pressure and counter to transmural pressure. An increase in extra-arterial dynamic pressure[16] acts against arterial dilatation, with a decrease in vessel lumen,

Table 2 Comparison of the clinical and laboratory test data between the two groups.

	STE-ACS (n = 138)	NSTE-ACS (n = 169)	*p*-Value
Age, years	62 ± 12	65 ± 11	0.03
Gender (males)	106 (45.3)	128 (54.7)	0.82
Body mass index (kg/m²)	27.71 ± 4.25	28.19 ± 4.66	0.34
Previous cerebrovascular event	4 (2.9)	9 (5.3)	0.29
Cardiovascular risk factors			
Arterial hypertension	69 (50)	104 (61.5)	0.07
Smoking	60 (43.4)	69 (40.8)	0.45
Hypercholesterolemia	81 (58.7)	106 (62.7)	0.47
Diabetes mellitus	39 (28.3)	59 (34.9)	0.21
Medication at discharge			
Aspirin	138 (100)	169 (100)	1
Clopidogrel	133 (96.4)	150 (88.7)	0.09
Beta-blockers	123 (89.1)	150 (88.8)	0.91
ACEIs/ARA-II DRUGS	94 (68.1)	123 (72.8)	0.37
Statins	138 (100)	169 (100)	1
Antidiabetic medication	39 (28.3)	59 (34.9)	0.21
Coronary angiography			
Normal coronary arteries	0 (0)	15 (8.9)	<0.001
Single-vessel lesion	85 (61.6)	65 (38.5)	
Two-vessel lesion	31 (22.5)	57 (33.7)	
Three-vessel lesion	22 (15.9)	32 (18.9)	
LVEF (%)	57 ± 10	59 ± 9	0.09
Blood test parameters upon admission			
Hemoglobin (mg/dl)	14.03 ± 1.69	14.79 ± 9.86	0.37
Hematocrit (%)	41.63 ± 4.79	41.51 ± 4.60	0.82
Leukocytes (10^9/l)	10.7 ± 3.9	9.6 ± 3	0.85
Neutrophils (10^9/l)	7.0 ± 1.4	6.8 ± 1.1	0.90
Creatinine (mg/dl)	0.85 ± 0.28	0.91 ± 0.57	0.28
Troponin I (ng/ml)	54.17 ± 29.14	12.21 ± 9.58	<0.001
Total cholesterol (mg/dl)	178 (150–204)	171.5 (145–200.5)	0.86
Triglycerides (mg/dl)	134.5 (104.7–158.2)	136.5 (111–200.2)	0.90
C-reactive protein (mg/dl)	7.9 (5–16.07)	8.6 (5–17.7)	0.27
Malondialdehyde (nmol/l)	2.235 (1.742–2.815)	2.230 (1.607–2.875)	0.44

ARA-II, angiotensin-II receptor antagonists; LVEF, left ventricular ejection fraction; ACEIs, angiotensin-converting enzyme inhibitors. Values expressed as n (%), mean ± standard deviation or median [interquartile range].

and therefore facilitates occlusive atherogenic phenomena resulting in STE-ACS.

The originality of our study focuses on three aspects: (a) It is the first study in Spain to prospectively analyze the effect of atmospheric pressure in patients admitted due to acute ischemic coronary disease; (b) In the study published by Danet et al.,[15] the analysis was population based, in contrast to our own study, where the analysis focused on hospital admissions due to ACS; and (c) In the patients who suffered ACS, increased atmospheric pressure in the week prior to admission was found to be a predictor of the presentation of STE-ACS.

Recently in Spain, and specifically in the region of Galicia, a retrospective study showed the incidence of acute myocardial infarction to be associated to atmospheric pressure.[17] The difference with respect to our study is that we used a retrospective design and for the first time showed the presence of elevated atmospheric pressure values during the week prior to the event to increase the risk that ACS will manifest as STE-ACS (i.e., acute coronary syndrome with ST-segment elevation).

Determining the influence of the variations in atmospheric pressure upon the physiopathological aspects of ACS could improve our understanding of the underlying cause–effect relationship and help to identify patients at risk, as well as design individualized preventive strategies. In this regard, the best preventive measure is adequate education of patients referred to the meteorological conditions of the region in which they live.[18] Accordingly, patients at risk should be made aware that sudden changes in atmospheric pressure are fundamentally caused by changes in altitude.

Meteorological variables are not independent but interrelate with each other; it therefore seems reasonable to assume that atmospheric pressure, humidity and other factors might contribute to variations in the incidence of ACS and explain the heterogeneity of the results found in the literature.[19]

Table 3 Data referred to gaseous pollutants, atmospheric particles and meteorological variables between the day before and the 7 days before admission, in the two study groups. All values are expressed with the mean concentration.

	STE-ACS (n = 138)	NSTE-ACS (n = 169)	*p*-Value
Meteorological variables			
Wind speed (m/s)	3.1 ± 0.4	3.1 ± 0.5	0.5
Temperature (°C)	21.2 ± 2.6	21.7 ± 2.7	0.06
Relative humidity (%)	60.2 ± 4	59.6 ± 5	0.3
Atmospheric pressure (mbar)	999.6 ± 2.6	998.8 ± 2.7	0.008
Gaseous pollutants (μg/m^3)			
Sulfur dioxide	7.1 ± 2.1	7.6 ± 2.3	0.03
Nitrogen dioxide	4.7 ± 1.3	4.7 ± 1.2	0.8
Ozone	66.9 ± 7.5	66.5 ± 8	0.7
Atmospheric particles (μg/m^3)			
PM-10	17.6 ± 5.4	17 ± 5.6	0.3
PM-2.5	8.6 ± 2	8.7 ± 2	0.9
Black carbon	855.8 ± 205.2	872.3 ± 196.7	0.4

PM, particle material with aerodynamic diameter <10 μm (PM-10) and <2.5 μm (PM-2.5).
Values expressed as mean ± standard deviation.

Table 4 Independent predictors of the presentation of STE-ACS.

	OR	95%CI	*p*-Value
Age	0.97	0.95–0.99	0.023
Atmospheric pressure[a,b]	1.14	1.04–1.24	0.004

CI, confidence interval; OR, odds ratio.
[a] Mean of the last 7 days before admission.
[b] Adjusted for coronary disease ($p = 0.6$), troponin I ($p = 0.06$) and sulfur dioxide ($p = 0.5$).

Limitations

Our study has a number of limitations that should be commented:

(a) A problem inherent to studies of this kind on the effects of air pollution is error in the measurement of exposure, mainly as a result of the differences between what is measured by the recording stations and the actual exposure of each individual in the population (inter-individual variability).[20,21]
(b) Different population-based studies[5,12–17] have analyzed the influence of meteorological parameters upon the incidence of ACS. In all these studies the analysis has been made at population level, al contrast to our study, where analysis focuses on the hospital admissions due to ACS.
(c) The climate of a given region depends on a series of geographical factors: latitude, orography and the geographical setting. Our region is located between 28 and 29° N of the equator, and is therefore close to the tropic of cancer, which as a result of the prevailing winds is characterized by a uniform environmental temperature. Consequently, the results of our study should be confirmed by establishing comparisons with the data obtained in other geographical areas presenting climatic conditions different from our own.
(d) The present study has conducted a post hoc analysis of atmospheric pressure in relation to the type of ACS, with the inherent possibility of findings attributable to chance.

In conclusion, the present study shows that in patients with ACS, the presence of increased atmospheric pressure during the week prior to the event increases the risk that such ACS will manifest as STE-ACS (i.e., acute coronary syndrome with ST-segment elevation).

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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