Research Article

Study of effect of traffic air pollutants on blood coagulation parameters among Saudi traffic policemen in Eastern Province

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Received May 17, 2015. Accepted August 18, 2015

Abstract

Background: Traffic policemen who work in the busy traffic signal areas for years together are exposed to the risk of air traffic pollution. In the long run, the pollutants may produce disease such as blood coagulation disorders in the exposed individuals with changes in blood coagulation mechanism.

Objective: To study the effect of traffic air pollution on blood coagulation parameters in Saudi policemen in Eastern Province.

Materials and Methods: Blood coagulation profile (prothrombin time (PT), PTT, antithrombin III, protein S, protein C, and fibrinogen) were measured in 50 male traffic policemen and in 25 matched control subjects. However, particulate and gaseous air pollutants were analyzed in different signals in Dammam and Khobar in Eastern Province (NO_2, VOCs, O_3, SO_2, CO, CO_2, and PM_{10} levels).

Result: Data of this study revealed that traffic policemen are exposed to significant higher levels of gaseous and particulate air pollutants that might modify or change blood coagulation profile. Others blood coagulation profile is not affected to some extent by higher levels of air pollutants.

Conclusion: Our study found minor changes in some coagulation parameters with higher concentrations of ambient air pollutants. However, air pollution levels showed statistically no consistent association with the activated partial thromboplastin time, protein S, protein C, and fibrinogen.

KEY WORDS: Bloods coagulation, air pollution, particulate, traffic policemen, Dammam, Eastern Province

Introduction

Over the past two decades, a growing body of evidence has led to a heightened concern about the potential deleterious health effects of ambient air pollution and its relation to coagulation disease.[1] Several air pollutants, including particulate matter (PM), carbon monoxide (CO), nitrogen oxides (NO_x), sulfur dioxide (SO_2), and ozone (O_3) have been associated with increased hospitalization and mortality as a result of coagulation disease and stroke. The underlying mechanisms linking air pollutants and increased cardiovascular risk remain unclear, although prior studies have associated exposure to air pollution with activation of inflammatory pathways, production of reactive oxygen species, endothelial injury and dysfunction, arterial vasoconstriction, and alterations in blood coagulation factors. Seaton et al. proposed that inhaled pollutants induce alveolar inflammation with release of mediators capable of increasing blood coagulability.[2,3]

Dammam is the capital of the Eastern Province of Saudi Arabia. It is about 400 km away from Riyadh. It is the major seaport of the region. Khobar is another large city in the Eastern Province. Hence, air pollution abatement will remain a challenge

Access this article online	
Website: http://www.ijmsph.com	Quick Response Code:
DOI: 10.5455/ijmsph.2016.1705201587	

and increasing traffic flow on Dammam and Khobar roads leads to traffic congestion and increase the impact of air pollution on the public and mainly on the traffic policemen at different traffic signals distributed along the main roads in the two major cities.

The contribution of inflammation to blood coagulation is de novo synthesis of tissue factor on leukocytes and endothelial cells stimulated by inflammatory cytokines, acute phase C-reactive protein, and reactive oxygen species. Exposure of the blood to tissue factor triggers the extrinsic coagulation pathway whose function is monitored by the prothrombin time (PT) through interaction of tissue factor with activated coagulation factor.[4,5] The plasma levels of several coagulation factors have been investigated in epidemiological studies as potential mediators of air pollution-related hypercoagulability. Coagulation factors such as FVII and fibrinogen, which are part of the acute-phase responses mediated by cytokines released during inflammatory reactions, increase after short-term exposure to particles. However, whether air pollution exposure is associated with hypercoagulability as measured with global coagulation tests has never been determined.[6,7]

Traffic policemen who work in the busy traffic signal areas for years together are exposed to the risk of air traffic pollution. In the long run, the pollutants may produce diseases such as asthma and bronchitis in the exposed individuals with changes in normal blood functions. The United Nations estimated that more than 600 million people in urban areas worldwide were exposed to dangerous levels of traffic-generated air pollutants.[8]

Baccarelli et al.[9] have performed several studies regarding air pollution exposure and changes in blood homeostasis. To investigate the association between pollution levels (PM10, CO, NO2, SO2, and O3) and changes in global coagulation tests, such as PT and activated partial thromboplastin time (APTT), it was shown that air pollution is associated with changes in global coagulation function, suggesting a tendency toward hypercoagulability after short-term exposure to air pollution.[9] Both gaseous air pollutants (i.e., O_3, SO_2 and NO_x, CO) and PM cause adverse effects on health. However, the most serious effects are related to PM, because particles contain a broad range of toxic substances and are considered reliable indicators of other pollutants (such as nitric oxides) and hence of the global adverse impact of air pollution.[10]

In humans, increased exposure to air pollution is associated with the increase in plasma of several hemostasis components. Plasma viscosity increased markedly during air pollution. Healthy volunteers experimentally exposed to concentrated PM had increased plasma levels of fibrinogen, an acute-phase inflammatory protein, an important determinant of increased viscosity and an established risk factor for venous and arterial thrombosis.[11] Another marker of thrombophilia, such as plasma homocysteine, is positively associated with exposure to traffic-related pollutants, especially PM and black carbon. A study conducted in 1218 healthy individuals found that the degree of chronic exposure to air pollution was associated with the shortening of such a global coagulation test as the prothrombin time. The same study confirmed that homocysteine increases in proportion to the degree of exposure to gaseous and particulate air pollutants, albeit only in smokers.[12,13]

PM has been associated with transient increases in plasma viscosity and thrombus formation, indicating the effects of circulating particles on changes in hemostasis. In recent study that includes men and women of the age group 25–64 years, high concentrations of SO_2, CO, and TSP were associated with increased plasma viscosity.[14] Bonzini et al.[15] demonstrated heightened thrombin formation in workers from a steel-production plant exposed to high concentrations of inhalable particles. In these workers, the link between inflammation and hypercoagulability was emphasized by the concomitant increase in C-reactive protein. The fibrinolytic system is an important regulator of thrombus formation, propagation, and vascular occlusion. The main aim of the study was to assess traffic air pollution and its impact on blood coagulation among Saudi traffic policemen in Eastern Province in Kingdom of Saudi Arabia.

Materials and Methods

Collection of different types of air pollutants gaseous (VOCS, CO2, O3, CO, NO2, and [SO2 and PM10]) samples by the use of calibrated instrumentation from different traffic signals sites which selected randomlly in Eastern Province, Saudi Arabia.

Four air pollutants were measured during this study including: PM_{10} which was measured in micrograms per cubic meter (μg/m3); SO2, CO, VOCs, O_3, nitrogen dioxide (NO2), O3, and carbon dioxide (CO2) which were measured in parts per million (ppm). At each sampling traffic signal, 25 readings (over 2-h period) were directly recorded on the basis of 5 min averages for each gaseous pollutant at morning, noon, and evening time. Therefore, in this study, there were 4000 records for each of the NO2, VOCs, O_3, SO2, CO, CO2, and PM_{10} levels.

Collected samples were then analyzed by standard methods for computation of concentrations of SO_2, NO_2, O_3, VOCs, CO, and CO_2. The EntryRAE (PGM-3000) Multi-Gas Monitor was used for measurement of CO_2 and the VRAE Hand Held 5 Gas Surveyor (Model 7800 Monitor) was used for measurement of NO_2, SO_2, O_3, and CO. For quality assurance purposes, data of the two gas monitors were calibrated against known concentrations of these gases.

PM_{10} were sampled on 60-mm-diameter glass fiber filters by the precalibrated hand-held battery portable air sampler on the basis of 2-h samples. Levels of PM were determined gravimetrically in $\mu g/m^3$. Eight hours gravimetric measurements of PM_{10} concentrations took place at three sites of the traffic signal in Dammam and Khobar.

Blood samples were collected from traffic policemen randomly selected exposed to air pollutants (n = 50) in Dammam and Khobar in eastren province for monitoring the blood coagulation paramerters (PT, PTT, protien C, protien S, and fibronogen) and matched control group (n = 25) were

analyzed using the STA-R automated coagulation analyzer (Diagnostica Stago, Asnières-sur-Seine, France).

Data Analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS) and Excel. Descriptive statistics and independent *t*-test were used to analyze significance of the tested variables.

Result

In Table 1, significantly lower levels of CO, NO2, and O3 are observed. However, significantly higher levels of SO2, PM10, CO2, and VOCs are found in different traffic signals in Dammam and Khobar at morning time and these might be due to high traffic density.

In Table 2, significantly lower levels of CO, NO2, and O3 are observed. However, significantly higher levels of SO2, PM10, CO2, and VOCs are found in different traffic signals in Dammam and Khobar at afternoon time and these might be due to high traffic density.

In Table 3, significantly lower levels of CO, NO2, PM10, and O3 are observed. However, significantly higher levels of SO2, CO2, and VOCs are found in different traffic signals in Dammam and Khobar at evening time.

Changing meteorological conditions on short and long time scales may affect the atmospheric pollutants' concentrations.

In Table 4, there is no significance differences in age, BMI, PT, PTT, antithrombin III, and protein C levels where there is slight significance in the levels of fibrinogen and protein S levels.

Discussion

Traffic congestion contributes to traffic pollution, and hence affects public health, and may cause annoyance particularly for those live, or work in heavy traffic roads. Traffic emission levels are higher in congested, stop-and-go and idling traffic than they are when traffic is moving at a steady speed. Traffic pollutants, of which PM, total volatile organic compounds (VOCs) SO_2, and CO, have significant effects on the policemen controlling the traffic congestion.

Traffic air pollution releases significant amounts of gas emissions and compounds including carbon monoxide (CO), CO_2, PM, SO_2, NO_2, particles, and volatile organic compounds (VOCs). Industry and transport are the two primary sources of air pollution in Saudi Arabia.

Particulates and gaseous emission of pollutants from industries and auto-exhaust are responsible for rising discomfort, increasing airway diseases, decreasing productivity, and deteriorating air quality in surrounding environment.

Monitoring the impact of primary industrial particulate emissions on air quality, major ambient air particles derived from these emissions are found to be mainly in the range of 2.5–10 m.[16–18]

Air pollutants that originate in the outdoor atmosphere from automobile and factory emissions and other combustion processes are likely to be present indoors. In the absence of indoor sources of these pollutants, concentrations indoors will be close to or lower than those outdoors. Over the past two decades, a growing body of evidence has led to a heightened concern about the potential deleterious health effects of ambient air pollution and its relation to cardiovascular disease. Several air pollutants, including PM, CO, NOx, SO_2 and O_3 have been associated with increased hospitalization and mortality as a result of cardiovascular disease and stroke.[19–25]

The underlying mechanisms linking air pollutants and increased cardiovascular risk remain unclear, although prior studies have associated exposure to air pollution with activation of inflammatory pathways, production of reactive oxygen species, endothelial injury and dysfunction, arterial vasoconstriction, and alterations in blood coagulation factors. Seaton et al. proposed that inhaled pollutants induce alveolar inflammation with release of mediators capable of increasing blood coagulability. The contribution of inflammation to blood coagulation is de novo synthesis of tissue factor on leukocytes and endothelial cells stimulated by inflammatory cytokines, acute phase C-reactive protein, and reactive oxygen species. Exposure of the blood to tissue factor triggers the extrinsic coagulation pathway whose function is monitored by the PT through interaction of tissue factor with activated coagulation factor VII (FVII).[16–18]

Overall, however, most of these studies indicate that air pollutants can induce oxidative stress and inflammation, as well as prothrombotic responses by vascular endothelial cells and platelets with expression of inflammatory cytokines, cellular adhesion molecules, and coagulation factors. The plasma levels of several coagulation factors have been investigated in epidemiological studies as potential mediators of air pollution-related hypercoagulability. Coagulation factors such as FVII and fibrinogen, which are part of the acute-phase responses mediated by cytokines released during inflammatory reactions, increase after short-term exposure to particles.[26,27]

All the participants had normal coagulation times, with the PT INR between 11.5 and 11.7 and APTT ratios (R) between 29.0 and 29.2 among Dammam and Khobar policemen, respectively. PT measures the formation of the fibrin clot through the activity of the extrinsic and common coagulation pathways, which involve the interaction of tissue factor and activated FVII, in addition to FX and FV, prothrombin, and fibrinogen. The test is based on plasma recalcification in the presence of tissue factor. In another study not in accordance with our findings, a mildly shortened PT in association with high concentrations of PM10, CO, and NO2 apparently reflects air pollution-related changes in blood coagulation. The association of PT with PM10, CO, and NO2, but not with SO2 or O3, may indicate that traffic pollution is responsible for the changes in blood coagulability. Ambient CO and NO2 have been shown to be correlated with particles from traffic, whereas ambient O3 is considered a surrogate for secondary particle exposures. Previous reports have shown that particles originating from traffic are more associated than other pollutants with mortality effects.[28–30]

Table 1: Comparison of the mean morning levels of air pollutants in Dammam and Khobar traffic signals

		CO2 ppm	PM10 ($\mu g/m^3$)	CO ppm	NO2 ppm	SO2 ppm	VOCs ppm	O3 (ppm)
Dammam	Signal street 18	770	357	1.6	0.15	0.56	1.30	0.02
	Signal King Khaled	880	429	2.0	0.13	0.50	0.81	0.01
	Signal King Saoud	1030	559	3.0	0.22	0.50	0.99	0.01
	Signal Shateaa Mall	1000	464	2.6	0.14	0.60	0.65	0.01
	Signal Alhyate Plaza	1290	692	3.6	0.25	0.60	2.51	0.04
	Signal Hospital of Dammam	1250	560	1.8	0.19	0.40	1.95	0.02
	Signal Almazaraa street	1430	452	2.1	0.15	0.50	1.68	0.01
	Signal Almoutanbea library	1560	420	2.0	0.21	0.60	1.39	0.20
Khobar	Signal Dhahran Mall	990	581	3.2	0.08	0.09	0.91	0.07
	Signal Rashid Mall	960	550	2.1	0.06	0.15	1.26	0.02
	Signal beside Ebn Sinaa	1180	316	1.8	0.17	0.40	2.04	0.02
	Signal Beside Egypt airlines	1370	455	1.8	0.13	0.40	1.89	0.03
	Signal Al-Merdian Hotel	1290	464	2.9	0.08	0.60	1.57	0.01

Table 2: Comparison of the mean noon levels of air pollutants in Dammam and Khobar traffic signals

		CO2 ppm	PM10 ($\mu g/m^3$)	CO ppm	NO2 ppm	SO2 ppm	VOCs ppm	O3 ppm
Dammam	Signal street 18	950	391	1	0.22	0.60	1.96	0.03
	Signal King Khaled	1100	251	1	0.18	0.70	1.99	0.01
	Signal King Saoud	1400	209	3	0.23	0.80	1.2	0.07
	Signal Shateaa Mall	1100	397	2	0.22	0.40	0.72	0.01
	Signal Alhyate Plaza	1730	495	5	0.24	0.60	2.11	0.04
	Signal Hospital of Dammam	1440	251	2	0.22	0.50	1.75	0.03
	Signal Almazaraa street	1250	391	1	0.19	0.40	1.62	0.01
	Signal Almoutanbea library	1090	455	1	0.20	0.37	1.49	0.02
Khobar	Signal Dhahran Mall	2030	527	1	0.12	0.29	1.73	0.05
	Signal Rashid Mall	1650	439	2	0.11	0.42	1.39	0.03
	Signal beside Ebn Sinaa	1470	332	1	0.12	0.54	1.91	0.01
	Signal Beside Egypt airlines	2130	100	1	0.10	0.44	1.92	0.01
	Signal Al-Merdian Hotel	2220	115	2	0.10	0.37	1.88	0.01

Table 3: Comparison of the mean evening levels of air pollutants in Dammam and Khobar traffic signals

		CO2 ppm	PM10 ($\mu g/m^3$)	CO ppm	NO2 ppm	SO2 ppm	VOCs ppm	O3 (ppm)
Dammam	Signal street 18	2100	289	1	0.19	0.4	1.53	0.01
	Signal King Khaled	2790	311	4	0.24	0.4	1.67	0.03
	Signal King Saoud	2880	213	7	0.23	0.5	1.84	0.01
	Signal Shateaa Mall	3420	397	5	0.15	0.7	1.69	0.02
	Signal Alhyate Plaza	4340	381	3	0.30	0.4	1.73	0.03
	Signal Hospital of Dammam	3950	413	4	0.33	0.5	1.91	0.02
	Signal Almazaraa street	3330	345	2	0.25	0.4	1.76	0.03
	Signal Almoutanbea library	2330	213	2	0.18	0.6	1.54	0.03
Khobar	Signal Dhahran Mall	1890	432	2	0.12	0.6	1.22	0.01
	Signal Rashid Mall	2000	442	2	0.13	0.3	1.85	0.03
	Signal beside Ebn Sinaa	2050	128	1	1.50	0.4	1.54	0.02
	Signal Beside Egypt airlines	1900	337	2	0.10	0.5	1.75	0.01
	Signal Al-Merdian Hotel	2030	439	2	2.10	0.5	1.70	0.02

Table 4: Characteristics of the study subjects and mean levels of the coagulation measurement investigated

	Dammam Traffic Policemen *n* = 25	Khobar Traffic Policemen *n* = 30	Control Subjects *n* = 25	*P*-value
Age, years	31.25 ± 11	37.10 ± 15	33.4 ± 11.2	0.81
Body mass index	28.25 ± 6.9	29.25 ± 7.1	24.1 ± 3.2	0.39
Prothrombin time (11–13.4)	11.5 ± 0.7	11.7 ± 0.5	11.1 ± 0.3	0.26
Activated partial thromboplastin time (24.1–34.7)	29.0 ± 3.2	29.2 ± 3.1	26.3 ± 2.3	0.3
Fibrinogen, mg/dL (200–400)	300.25 ± 27.7	323 ± 36.9	318 ± 25.1	0.02*
Antithrombin (80–120%)	105.1 ± 13.8	103.2 ± 10.1	99.5 ± 9.5	0.29
Protein C (%) (70–130%)	97.9 ± 35.1	106.2 ± 12.3	98.1 ± 9.4	1.3
Protein S (%) (65–140%)	107.5 ± 19.7	98.1 ± 12.6	110.2 ± 14.1	0.04*

Similarly, levels of the other coagulation measurements were within the normal limits. No association between air pollutant concentrations at the time of the study and the APTT was found. In particular, increasing concentrations of PM10, CO, and NO2 were associated with shorter PT. No important relations with air pollution were found for the APTT, fibrinogen, and the natural anticoagulant proteins. Air pollution levels showed no consistent association with the APTT, natural anticoagulants, and fibrinogen. The observation of shortened PT may indicate that air pollution determines hypercoagulability, potentially contributing to the increase in cardiovascular events observed after exposure to air pollution.[31–33]

However, similar studies are not in accordance with our study where, increasing concentrations of PM10, CO, and NO2 were associated with shorter PT. In addition, a similar degree of shortening of the PT was related with 30-day average PM10 and NO2 levels. No important relations with air pollution were found for the APTT, fibrinogen, and the natural anticoagulant proteins. In contrast, the APTT was not associated with air pollution exposure in this study. Our finding of a mildly shortened PT in association with high concentrations of PM10, CO, and NO2 apparently reflects air pollution-related changes in blood coagulation.[34]

Similar study results are in accordance with the findings of this study in which air pollution exposure during commuting was not associated with consistent changes in inflammation markers, blood cell counts, or coagulation markers.[35]

Recent study revealed that association of PT with PM10, CO, and NO2, but not with SO2 or O3, may indicate that traffic pollution is responsible for the changes in blood coagulability. Ambient CO and NO2 have been shown to be correlated with particles from traffic, whereas ambient O3 is considered a surrogate for secondary particle exposures.[36,37]

A limitation of this study is that ambient air pollution was used as a surrogate for personal exposure, which may have resulted in a measurement error. Such a measurement error would generally tend to bias estimates toward the null and may have contributed to the lack of association of air pollution found for fibrinogen and natural anticoagulants.

Conclusion

The study adds to the evidence that elevated levels of ambient air pollution may cause coagulation responses. These changes in blood markers could represent additional risk factors especially in category of policemen continuously exposed to particulate and gaseous air pollutants.

Blood coagulation disorders have caused significant human and public health burden, with sustained reductions in air pollution exposure associated with increased life expectancy. We still have a long way to go in reducing air pollution levels and associated diseases. Future investigations of air pollution-induced blood coagulation disorders must not only include more studies to determine the mechanisms of action but also examine the role of each specific component of air pollution to determine what combination of particulate and gaseous air pollutants is to blame for this sudden increase in environment-induced health concerns. This information is paramount for policy makers to determine acceptable levels of air pollution and to design ways to minimize the harmful effects of air pollutants on the body.

Acknowledgment

The authors acknowledge the financial support (Grant No. 2014058) provided by Deanship of Scientific Research, University of Dammam, for conducting this study. They express their gratitude to the traffic policemen administration in Eastern Province for their valuable cooperation and massive support for completion of this work.

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How to cite this article: Bashawri LAM, Salama KF. Study of effect of traffic air pollutants on blood coagulation parameters among Saudi traffic policemen in Eastern Province. Int J Med Sci Public Health 2016;5:477-483

Source of Support: Nil, **Conflict of Interest:** None declared.