Published in final edited form as:
Int J Cardiovasc Imaging. 2013 April ; 29(4): 827–833. doi:10.1007/s10554-012-0154-x.

Relationship between particulate matter exposure and atherogenic profile in "Ground Zero" workers as shown by dynamic contrast enhanced MR imaging

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Abstract

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Conflict of interest None.

In this pilot study, we hypothesize that dynamic contrast enhanced magnetic resonance imaging (DCE-MRI) has the potential to evaluate differences in atherosclerosis profiles in patients subjected to high (initial dust cloud) and low (after 13 September 2001) particulate matter (PM) exposure. Exposure to PM may be associated with adverse health effects leading to increased morbidity. Law enforcement workers were exposed to high levels of particulate pollution after working at "Ground Zero" and may exhibit accelerated atherosclerosis. 31 subjects (28 male) with high (n = 19) or low (n = 12) exposure to PM underwent DCE-MRI. Demographics (age, gender, family history, hypertension, diabetes, BMI, and smoking status), biomarkers (lipid profiles, hs-CRP, BP) and ankle-brachial index (ABI) measures (left and right) were obtained from all subjects. Differences between the high and low exposures were compared using independent samples *t* test. Using linear forward stepwise regression with information criteria model, independent predictors of increased area under curve (AUC) from DCE-MRI were determined using all variables as input. Confidence interval of 95 % was used and variables with $p > 0.1$ were eliminated. $p < 0.05$ was considered significant. Subjects with high exposure (HE) had significantly higher DCE-MRI AUC uptake (increased neovascularization) compared to subjects with lower exposure (LE). (AUC: 2.65 ± 0.63 HE vs. 1.88 ± 0.69 LE, $p = 0.016$). Except for right leg ABI, none of the other parameters were significantly different between the two groups. Regression model indicated that only HE to PM, CRP > 3.0 and total cholesterol were independently associated with increased neovascularization (in decreasing order of importance, all $p < 0.026$). HE to PM may increase plaque neovascularization, and thereby potentially indicate worsening atherogenic profile of "Ground Zero" workers.

Keywords

DCE-MRI; Particulate matter; Atherosclerosis; Plaque neovascularization

Introduction

Research has demonstrated a relationship between exposure to ambient air pollutants and cardiovascular disease (CVD) [1]. Studies in cohorts suggest links between pollution exposure and cardiovascular mortality, coronary heart disease events [2], and stroke [3]. Many studies have shown associations between short-term exposures to increased levels of air pollutants and CVD events [4]. This association is further increased in populations with risk factors for cardiovascular disease such as high cholesterol levels, diabetes mellitus, hypertension, and heart failure [5]. The biologic mechanisms for these associations are not entirely clear but are thought to be related to systemic inflammation, autonomic nervous system imbalance, changes in vascular compliance, altered cardiac structure, and development of atherosclerosis [6]. As CVD remains one of the leading causes of mortality and morbidity in the industrialized world, it is important to examine its relationship with exposure to air pollution.

Airborne particulate matter consists of particles that are of different sizes. Particles of sizes down to 0.5 μM demonstrate that they can penetrate the most distal airway units and potentially the systemic circulation and cause a variety of pulmonary effects [7]. Particles less than 2.5 μm ($PM_{2.5}$) have been more strongly linked with cardiovascular disease [8]. Different components of PM may affect the risk for cardiovascular disease by different mechanisms including electrophysiologic changes, inflammation, coagulation, endothelial cell function effects and increased atherogenesis. It is speculated that changes in atherosclerotic burden are due to changes in endothelial function and consequent inflammatory response brought about by exposure to PM. The endothelium is one of the major regulators of vascular homeostasis, and exerts a number of vasoprotective effects. Thus, measuring endothelial dysfunction and its subsequent consequences such as

inflammation and plaque angiogenesis/neovascularization would be of critical clinical importance.

Plaque angiogenesis may therefore be an attractive target to identify asymptomatic high-risk lesions or evaluate propensity for atherosclerotic disease progression. Dynamic contrast enhanced (DCE) MRI has been successfully used as a non-invasive tool to study the extent of plaque neovascularization in animals and patients with atherosclerosis [9–12]. The reproducibility and reliability of DCE-MRI has also been previously shown in several studies [10]. It has also been recently used as an evaluation metric in multi-center clinical trials with imaging that use as an endpoint [13].

An estimated 60,000 men and women worked at “Ground Zero” and Staten Island Landfill (wreckage depository site) after 11 September 2001 and were exposed to thousands of tons of fine particulate matter, cement dust, glass fibers, asbestos, lead, polychlorinated biphenyls (PCBs), and other pollutants [14, 15]. This cohort presents a unique opportunity to examine the effect of an acute particulate matter exposure event on the atherosclerosis cascade, which in turn may offer some insight into cardiovascular prognosis of individuals exposed to PM.

The non-invasive in vivo evaluation of atherosclerotic disease therefore presents an ideal mechanism to evaluate the effect of particulate matter exposure on the atherosclerosis cascade that is primarily responsible for cardiovascular events. It also presents itself as a well-validated and accurate tool for examining subclinical effects of cardiovascular disease. In this small pilot study, we plan to use DCE-MRI to evaluate the relationship between levels of particulate matter exposure after 11 September 2001 and atherosclerotic disease progression as evaluated by changes in plaque neovascularization.

Methods

The institutional review board approved this study and all subjects provided informed consent prior to participating in this study.

Study population

Subjects were recruited from the Law Enforcement Cardiovascular Screening Program (LECS) subset of the World Trade Center Medical Monitoring and Treatment Program. 31 law enforcement personnel (28 male) with high (n = 19) or low (n = 12) exposure were recruited. Exposure was classified as early, high-intensity exposure if the individual arrived during the morning of 11 September 2001 (Day 1) and was present during the collapse of North or South tower and the initial dust cloud; and as low-intensity exposure if they arrived on or after 13 September 2001 (Day 3) when PM was present but reduced.

Demographics (age, gender, family history, hypertension, diabetes, BMI, and smoking status), biomarkers (lipid profiles, hs-CRP, blood pressure) and ankle-brachial index (ABI) measures (left and right) were obtained from all subjects.

Multi contrast MRI

The bilateral carotid arteries of subjects were imaged on a 3T whole body MRI systems (Philips Achieva, The Netherlands). An 8-channel phased array coil (Philips Medical Systems, Shanghai, China) was used for carotid imaging.

Acquisition—After localization with fast gradient echo sequences, multi-contrast MR images were obtained using double inversion recovery (DIR) turbo spin echo (TSE) technique. Carotid images were acquired without cardiac triggering and free breathing. Proton density (PDW), T1 and T2 weighted images were acquired [16, 17]. A total of 6

transverse images starting and extending below the carotid bifurcation were obtained. Imaging parameters were as follows: repetition time, 2,000/2,000/1,000 ms (PDW/T2/T1 images); echo time 10/40/10 ms (PDW/T2/T1 images); field of view 14 cm; slice thickness 3 mm; 10 % inter-slice gap; Pixel size 0.5 mm × 0.5 mm; no phase wrap; number of signal averages 2/4/3 (PDW/T2/T1 images); turbo factor (echo train length), 11/11/7 (PDW/T2/T1 images); no zero filling. Inversion time, 157 ms. SPAIR was used for fat suppression [16, 17]. Images were obtained without cardiac or respiratory gating.

Image analysis—Inner and outer wall boundaries were manually traced by a trained observer (S.K.W.) on T2W images. A software program (Vessel Mass, Leiden, The Netherlands) was then used to determine the lumen area, wall area, total vessel area, normalized wall index and wall thickness [13, 18–21]. Plaque characterization was also performed based on multi-contrast images [22].

Dynamic contrast enhanced (DCE) MRI

Acquisition—DCE-MRI of the vessel wall was performed using a multislice T1 W black blood turbo spin echo (TSE) sequence (dose: 0.1 mmol/kg followed by 20 ml saline chase injected at 4 ml/s via an antecubital vein, acquisition time approximately ~12 min) [9].

Image analysis—Dynamic images were processed by applying de-noising and motion correction algorithms [10, 12]. DCE-MRI data were analyzed using non model-based approaches [23]. The area under the signal intensity versus time curves for 2 and 7 min after contrast agent injection were calculated [10]. All DCE-MRI analysiswas done using a custom-built software program in MATLAB (Math Works, Inc, Natick, MA, USA) [9, 10].

Statistical analysis

Statistical analysis was performed using IBM SPSS Version 19 (IBM SPSS Inc., Armonk, NY, USA). Continuous variables were expressed as mean ± SD. Levene's test was used to test for equality of variance. Differences between the high and low exposures in continuous variables were then compared using an independent samples *t* test. Using linear forward stepwise multivariate regression with Akaike information criteria corrected (AICC) [24], independent predictors of increased area under curve (AUC7) from DCE-MRI were determined using all variables as input. Confidence interval of 95 % was used and variables with $p > 0.1$ were eliminated. $p < 0.05$ was considered significant.

Results

Table 1 shows the results of demographics, biomarkers, ankle brachial indices, and MRI measures. Average across all patients, as well as distribution across the high and low exposure groups are presented. In terms of demographics and biomarkers, the 2 groups were well matched with no significant differences observed between the two groups.

Morphometric MRI did not show any significant differences between the exposure groups. None of the individuals in either exposure group had complex carotid artery plaques that could be classified [22] by multi contrast MRI. Figure 1 shows MR images from sample patients with high exposure (HE) and low exposure. Subjects with HE had significantly higher DCE-MRI AUC uptake (increased neovascularization) compared to subjects with lower exposure (LE). (AUC7: 2.65 ± 0.63 HE vs. 1.88 ± 0.69 LE, $p = 0.016$). Figure 2 shows sample DCE-MRI AUC maps from sample patients with high and low exposures.

The ABI of the right side was significantly higher in the low exposure group compared to the HE group. (ABI-r: 1.18 ± 0.08 HE vs. 1.26 ± 0.08 LE, $p = 0.017$).

Multivariate AICC regression model indicated that only HE to PM, CRP >3.0 and total cholesterol were independently associated with increased neovascularization as defined by DCE-MRI AUC uptake (in decreasing order of importance, all $p < 0.026$; Adjusted R^2 of model = 0.455).

Discussion

This preliminary pilot study hinted that law enforcement personnel exposed to high levels of PM after 11 September 2001 may have increased neovascularization as measured by DCE-MRI compared to those with low exposure. These results also seem to indicate that exposure to PM after 9/11 may potentially be associated with increased progression of atherosclerosis.

Previous studies have shown that high levels of exposure in non-rescue and non-recovery individuals, when compared with low exposed non-rescue and non-recovery individuals, were associated with heart-disease-related mortality [25]. This association was not found in rescue and recovery personnel. To our knowledge, the current study is among the first to use MR imaging to look at cardiovascular complications in law enforcement personnel with WTC exposure. This study seems to indicate that long-term cardiovascular effects may indeed be associated with WTC PM Exposure and that such effects warrant further investigation.

Studies have also shown that acute mental stress can lead to changes in myocardial wall motion or changes to perfusion [26] as shown by imaging. It is speculated that this effect may also be due to arterial vasoconstriction during mental stress, occurring at sites that manifest similar vasoconstriction during acetylcholine infusion. Other studies demonstrate that mental stress also effects endothelial function in the short term [27]. If this endothelial dysfunction caused by stress in the short term is coupled with deleterious effects caused by acute PM exposure over a prolonged period, it could provide a mechanism for advancing the rate of atherosclerosis progression in the exposed population.

Limitations

While the results of this study show some promise, several limitations need to be addressed. Firstly, this study was performed in a very small population and these findings need to be validated in a larger population. The current population could also be affected by selection bias, as the subjects were volunteers that were interested in participating in the study. This study also was limited to law enforcement personnel, who have higher rates of cardiovascular disease, in general, than the general population. The subject exposure groups in this study were also not fully similar in other aspects such as smoking status, gender and cholesterol which may potentially bias the results. Framingham risk scores were also not calculated for these subjects. The number of samples required for performing a robust multivariate regression is also limited. Despite these significant caveats, the multivariate regression identified HE to PM as an independent predictor of DCE uptake lending credence to the possibility that the effect observed in this study merits further investigation.

Inflammation is posited as the mechanism for the increased progression of atherosclerosis. 18-FDG-PET imaging [28, 29] may provide more direct evidence of increased inflammation in vasculature. Because of radiation exposure and cost issues, this was not done as part of this pilot study, but is being considered as a possible avenue for future work.

The use of DCE-MRI for evaluating atherosclerosis is a relatively new field and the optimal models for analysis are still under development. Previous studies have established a relationship between neovascularization on histology and DCE-MRI measures, and have used DCE-MRI to evaluate treatment effects in both humans and animal models [9–11, 30].

The reproducibility and robustness of the DCE-MRI approaches used in this study have also been previously evaluated [10]. Nevertheless, there was no histological verification possible in this study as none of the patients fit the criteria for a carotid endarterectomy.

Finally, the Akaike information criterion model has limitations as it only measures a relative goodness of fit and does not reveal how well a model fits the data in an absolute sense. The model also assumes a linear model with normally-distributed errors (conditional upon regressors).

Conclusions

High exposure to PM may be associated with plaque neovascularization, and thereby potentially indicates worsening atherogenic profile of “Ground Zero” workers. The results of this study also indicate that more future work is warranted to examine the long-term cardiovascular effects of particulate matter exposure in WTC exposure patients as well as in other groups who might have excessive particulate exposure from occupational or environmental sources.

Acknowledgments

Partial support was provided by: NIH/NHLBI R01 HL071021 (ZAF).

Abbreviations

AICC	Akaike information criteria corrected model
AUC	Area under the signal intensity versus time curve
CVD	Cardiovascular disease
DCE-MRI	Dynamic contrast enhanced MRI
DIR	Double inversion recovery
IMT	Intima-media thickness
LDL	Low-density lipoprotein
MRI	Magnetic resonance imaging
NO	Nitric oxide
PCB	Polychlorinated biphenyls
PDW	Proton density weighted
PM	Particulate matter
T1W	T1-weighted
T2W	T2-weighted
TSE	Turbo spin echo

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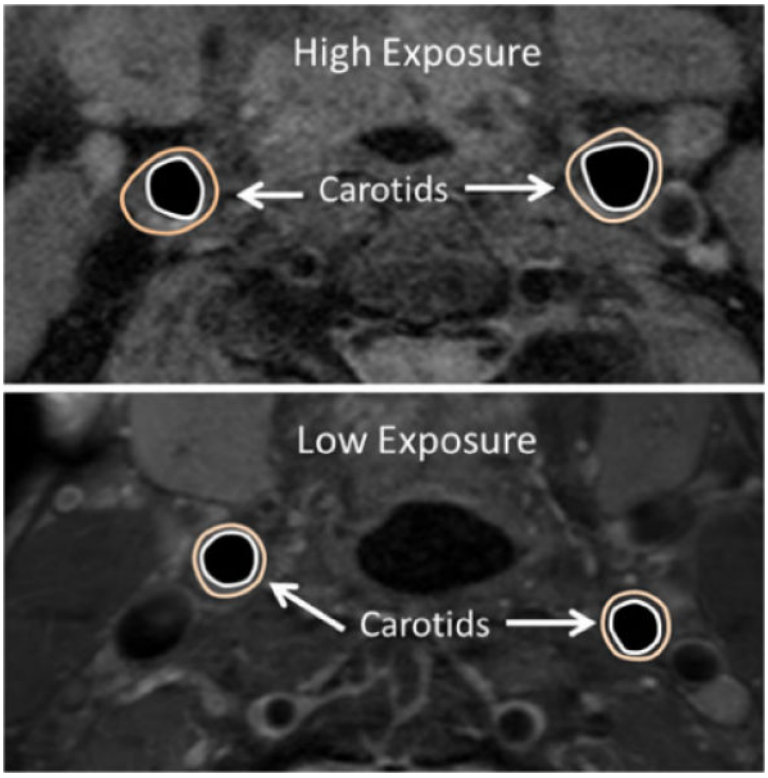

Fig. 1.
Sample carotid images from patients with high (*top*) and low (*bottom*) PM exposure

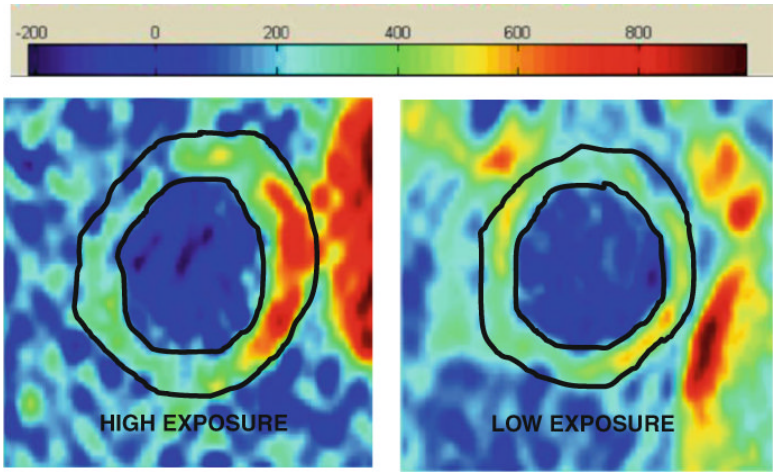

Fig. 2.
Sample DCE-MRI AUC (7 min) images of a left common carotid artery from high and low PM exposure patients

Table 1

Characteristics of study population in the two exposure groups

Variable	Total, n (%) N = 31	High exposure, n (%) N = 19	Low exposure, n (%) N = 12	*p* value
Demographics				
Gender-male	28 (90.3)	18 (94.7)	10 (83.3)	
Gender-female	3 (9.7)	1 (5.3)	2 (16.7)	
Smoker (current)	1 (3.2)	1 (5.3)	0 (0.0)	
Smoker (past)	4 (12.9)	3 (15.8)	1 (8.3)	
Diabetes	0 (0.0)	0 (0.0)	0 (0.0)	
Hypertension	5 (16.1)	3 (15.8)	2 (16.7)	
Family history of CVD	15 (48.4)	8 (42.1)	7 (58.3)	
Calcium score >75th percentile	7 (22.6)	4 (57.1)	3 (42.9)	

	N = 31	N = 19	N = 12	
Measured variables				
Age (years)	45.1 ± 4.4	44.6 ± 4.1	45.8 ± 4.8	0.497
Total cholesterol (mg/dl)	200 ± 43	205 ± 43	190 ± 43	0.338
HDL (mg/dl)	52 ± 19	55 ± 20	47 ± 17	0.298
LDL (mg/dl)	121 ± 34	123 ± 36	117 ± 30	0.657
Fasting blood glucose	88 ± 24	89 ± 30	85 ± 8	0.662
Heart rate (bpm)	68 ± 11	70 ± 12	64 ± 7	0.134
SBP (mm Hg)	121 ± 9	120 ± 9	121 ± 10	0.930
DBP (mm Hg)	76 ± 7	76 ± 8	77 ± 7	0.667
BMI	27 ± 2.7	27 ± 2.6	28 ± 2.7	0.206
Waist: hip ratio	0.91 ± 0.05	0.92 ± 0.05	0.90 ± 0.04	0.202
ABI-left	1.23 ± 0.11	1.21 ± 0.10	1.26 ± 0.12	0.215
ABI-right	1.21 ± 0.09	1.18 ± 0.08	1.26 ± 0.08	0.017

	N = 31	N = 19	N = 12	
MRI morphometrics				
Wall area (cm^2)	0.26 ± 0.08	0.26 ± 0.07	0.27 ± 0.10	0.882
Total vessel area (cm^2)	0.61 ± 0.12	0.61 ± 0.11	0.61 ± 0.14	0.999
Lumen area (cm^2)	0.35 ± 0.05	0.35 ± 0.05	0.35 ± 0.05	0.801
Normalized wall index	0.42 ± 0.05	0.41 ± 0.04	0.42 ± 0.06	0.702
Wall thickness (mm)	1.06 ± 0.24	1.05 ± 0.20	1.07 ± 0.30	0.869

	N = 23	N = 16	N = 7	
DCE-MRI measures				
Norm. AUC-2 min	2.76 ± 1.61	3.14 ± 1.6	1.88 ± 1.37	0.084
Norm. AUC-7 min	2.42 ± 0.73	2.66 ± 0.63	1.88 ± 0.69	0.016

Values are indicated as mean ± SD and categorical data is indicated as absolute numbers and percentages