Epidemiologic investigation supports a positive relationship between exposure to air pollution and cardiovascular disease (Rich et al. 2006), with the number of deaths from such illness estimated to exceed that for respiratory disease after exposures to elevated levels of pollutants (Dockery 2001). Air pollutants have been associated with acute cardiac events, but population-level data are not directly relevant to the clinical presentation of individual cases. To our knowledge, this is the only case report of an individual suffering an episode of atrial fibrillation after exposure to an air pollutant. The resolution of the arrhythmia with termination of the particle exposure further supports a causal relationship between the two.

RELEVANCE TO CLINICAL PRACTICE: Exposure to air pollution, including particulate matter, may cause supraventricular arrhythmias.

KEY WORDS: air pollution, arrhythmias, atrial fibrillation, atrial flutter, heart diseases, particulate matter.

Case Report: Supraventricular Arrhythmia after Exposure to Concentrated Ambient Air Pollution Particles

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CONTEXT: Exposure to air pollution can result in the onset of arrhythmias.

CASE PRESENTATION: We present a case of a 58-year-old woman who volunteered to participate in a controlled exposure to concentrated ambient particles. Twenty minutes into the exposure, telemetry revealed new onset of atrial fibrillation. The exposure was discontinued, and she reverted to normal sinus rhythm approximately 2 hr later. No abnormality was evident on the volunteer’s laboratory examination or echocardiography that could explain an increased risk for supraventricular arrhythmia.

DISCUSSION: Epidemiologic evidence strongly supports a relationship between exposure to air pollutants and cardiovascular disease, but population-level data are not directly relevant to the clinical presentation of individual cases. To our knowledge, this is the only case report of an individual suffering an episode of atrial fibrillation after exposure to an air pollutant. The resolution of the arrhythmia with termination of the particle exposure further supports a causal relationship between the two.

RELEVANCE TO CLINICAL PRACTICE: Exposure to air pollution, including particulate matter, may cause supraventricular arrhythmias.

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The patient was admitted to the hospital overnight for observation and telemetry. The next morning, the ECG documented normal sinus rhythm. Her serum electrolytes, blood urea nitrogen, creatinine, glucose, creatine kinase, and the MB fraction were again normal, and her complete blood count was normal except for a hematocrit of 35.7% (the lower limit of normal is 36.0%). Resting transthoracic echocardiography demonstrated normal right ventricular contraction with an ejection fraction of 55–60%, aortic sclerosis, and diastolic left ventricular dysfunction. The left atrium was considered mildly dilated; all other chambers of the heart were normal in size. She was discharged and was not prescribed a new medication. Approximately 6 weeks later, she underwent electrophysiology study, which did not provoke atrial fibrillation or significant atrial ectopy. The study did indicate a reentrant circuit of the cavotricuspid isthmus, which was ablated to prevent potential future episodes of atrial flutter.

**Discussion**

The volunteer demonstrated evidence of increased supraventricular ectopy immediately preceding her exposure to CAPs, but there was no evidence of atrial arrhythmias. She then suffered the onset of atrial fibrillation a very short time after being exposed to CAPs. Within 2–3 hr after the exposure stopped, the arrhythmia resolved and she returned to normal sinus rhythm. Atrial fibrillation is the most common supraventricular arrhythmia, affecting 1–2% of the general population (Falk 2001). This arrhythmia is uncommon in people < 60 years of age, but it afflicts about 10% of the population by 80 years of age. Risk factors for atrial fibrillation include hypertension, (especially uncontrolled), coronary artery disease, heart failure, cerebrovascular disease, diabetes, thyroid conditions, sleep apnea, obesity, a past history of rheumatic heart disease and congenital heart defects, pericarditis, sick sinus syndrome, a family history of atrial fibrillation, and echocardiographic abnormalities (Kannel and Benjamin 2008, 2009). In addition, cigarette smoking, alcohol use, caffeine consumption, and stimulant drugs can help trigger atrial fibrillation. Of these defined risk factors, the volunteer had a history of well-controlled hypertension, and her body mass index was consistent with obesity. Her history of premature atrial contractions may also have increased her risk for atrial fibrillation (Binici et al. 2010). In a similar manner, preexisting cardiovascular disease, diabetes and impaired glucose tolerance, chronic obstructive pulmonary disease, and current cigarette smoking all increase susceptibility for cardiovascular disease associated with air pollution (Chen et al. 2006; Liao et al. 2009; Mills et al. 2007; Wheeler et al. 2006; Whitesel et al. 2009; Zareba et al. 2009). There was no obvious explanation for her onset of a supraventricular arrhythmia during the exposure. Although coincident atrial fibrillation cannot be excluded, the onset of her arrhythmia was associated with her exposure to ambient air pollution particles. The correlation between the resolution of the arrhythmia and the termination of the CAP exposure further supports a causal relationship between the two.

Systemic inflammation and underlying oxidative stress may increase the risk of atrial fibrillation (Kumagai et al. 2004). Patients with atrial fibrillation demonstrate evidence of inflammation, with elevated levels of inflammatory markers, including C-reactive protein, interleukin-6, and tumor necrosis factor-α (Chung et al. 2001; Gaudino et al. 2003). Some evidence suggests that statin treatment may potentially alter the risk for this arrhythmia by modifying oxidative stress (Siu et al. 2003). The specific association between increased arrhythmia induction and air pollution may reflect oxidant generation and inflammation after exposure, consistent with mechanisms involved in the initiation and maintenance of some other forms of atrial fibrillation (Mazzoli-Rocha et al. 2010). The oxidative stress and inflammation associated with the pollutant have been postulated to affect coronary perfusion and consequently enhance the propensity for such arrhythmias through tissue ischemia. However, the rapid onset of this volunteer’s atrial fibrillation after CAP exposure suggests that the basis for the arrhythmia may be a disruption of the normal cardiac autonomic control rather than a systemic inflammation, because the latter would require a longer period of time to develop (Routledge and Ayres 2005). In an animal model, diesel exhaust increased the sensitivity of the heart to triggered arrhythmias via an activation of airway sensory receptors [e.g., TRPA1 (transient receptor potential cation channel A1)] (Hazari et al. 2011). Several researchers have suggested that this leads to autonomic imbalance and a predisposition for arrhythmia development. A comparable mechanism has been proposed to explain the cardiac response to ozone and cigarette smoke (Joad et al. 1998; Mutoh et al. 2000).

![Figure 1. The volunteer’s ECG (12 lead and rhythm strip) before (A) and immediately after (B) exposure to concentrated ambient particles. The ECG before the exposure (A) reveals a regular sinus rhythm with defined P waves (arrows); the ECG after the exposure (B) is irregular, with “flutter” waves (arrows).](image-url)
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