

Case report

Open Access

Suspected association of ventricular arrhythmia with air pollution in a motorbike rider: a case report

Kent Emilsson

Address: Department of Clinical Physiology, Örebro University Hospital, SE-701 85 Örebro, Sweden

Email: Kent Emilsson - kent.emilsson@orebroll.se

Published: 3 June 2008

Received: 14 November 2007

Journal of Medical Case Reports 2008, **2**:192 doi:10.1186/1752-1947-2-192

Accepted: 3 June 2008

This article is available from: <http://www.jmedicalcasereports.com/content/2/1/192>

© 2008 Emilsson; licensee BioMed Central Ltd.

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/2.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Introduction: Premature ventricular complexes are to some extent a normal finding in healthy individuals and the prevalence increases with age and is more common in men. Premature ventricular complexes can occur in association with a variety of stimuli, and a lesser known cause is the association between air pollution and ventricular arrhythmias.

Case presentation: A previously healthy man started to ride a lightweight motorbike in heavy traffic. A few weeks later he was admitted to hospital with premature ventricular complexes in bigeminy, which decreased after a few days when he was not exposed to exhaust fumes. A few weeks later he started using the motorbike again and the same symptoms developed once more, only to subside when he stopped riding in heavy traffic.

Conclusion: Studies have shown an association between air pollution and premature ventricular complexes and other kinds of arrhythmias. The mechanism may be changes in cardiac autonomic function, including heart rate and heart rate variability. Air pollution should be considered when patients present with arrhythmias and no other causes are found.

Introduction

To some extent premature ventricular complexes (PVCs) are a normal finding in healthy individuals and the prevalence increases with age and is more common in men. However, PVCs can occur in association with a variety of stimuli and can be produced by direct mechanical, electrical and chemical stimulation of the myocardium. Often they are noted in patients with false tendons, ischaemic or inflamed myocardium and during infection, hypoxia, anaesthesia or surgery. They can be provoked by a variety of medications, by electrolyte imbalance, by tension states, by myocardial stretch and by excessive use of tobacco, caffeine or alcohol [1]. A cause of PVCs that is not so well known among physicians is the association between air pollution and ventricular arrhythmias.

Case presentation

A 43-year-old previously healthy man, a physician, living in a city of about 150,000 inhabitants, began to use a lightweight motorbike to commute to work, a distance of about 10 km, in heavy traffic in the middle of August 2006. He had previously been travelling in a car fitted with an air pollution filter, and had experienced no previous heart symptoms. He also walked or jogged about 6 km four to five times a week with no problems and was not on any medication. He felt relaxed and did not experience stress while riding the motorbike in heavy traffic. There were numerous traffic lights on his journey to work, which meant that he was forced to stop behind buses and trucks on several occasions where he experienced a strong smell of exhaust fumes.

After commuting to and from work by motorbike for about 2 weeks he began experiencing cardiac extrasystoles, something not previously experienced; on one occasion he was unable to sleep due to palpitations. He sought help and had an electrocardiogram (ECG) the following morning, which showed PVCs in bigeminy. The patient had also sinus tachycardia with a heart rate of about 110 beats per minute.

The patient was admitted to the cardiac intensive care unit and was examined using echocardiography, which was found to be normal, and there were no signs of false tendons. No ischaemia was seen on ECG and there were no signs of infarction. The frequency of PVCs began to decrease about 8 hours after admission. Blood tests showed no indications of infarction or infection, his blood glucose was normal and his lipid status and thyroid status were within normal limits. The patient had no fever. During the night and the next morning only a few PVCs and some premature atrial complexes were observed and the patient was discharged home. The diagnosis was given as myocarditis, although this diagnosis was uncertain.

The patient rested for 2 weeks with no further symptoms before returning to work. For the first few weeks he drove his car to work, but then began to use his motorbike again. Having used it for a few weeks on the same route he again began to experience extrasystoles and therefore contacted his physician, who recommended an exercise test and Holter ECG.

In the few days before the Holter ECG was applied the patient refrained from using his motorbike and began to feel better. Only a few PVCs and premature atrial complexes were found during 24 hours of Holter monitoring. The heart rate variability (HRV) showed a pattern with a somewhat high low-frequency to high-frequency ratio. An exercise test was carried out and the patient performed well, with no chest pain, arrhythmias or signs of ischaemia.

The patient began to believe that there was an association between using his motorbike and his symptoms and decided to stop using it. Since then no symptoms, apart from an occasional single extrasystole, have been noted by the patient.

Discussion

In the present case there was no obvious explanation for the PVCs. Myocarditis was thought to be the underlying cause, even if this diagnosis was uncertain. This is a condition with various clinical presentations, from non-specific symptoms (fever, myalgias, palpitations or exertional dyspnoea) to fulminant haemodynamic collapse and sudden

death. Myocarditis is difficult to diagnose and endomyocardial biopsy remains unequivocally the gold standard for establishing the diagnosis [2], although this cannot always be used in clinical practice. Cardiac biomarkers, especially troponin T or I, are now routinely measured by most clinicians when a clinical diagnosis of myocarditis is considered [2]. In the present case, however, the cardiac biomarkers creatine kinase and troponin T were not elevated.

Echocardiography can also help in the diagnosis of myocarditis, often showing left ventricular dysfunction and segment wall abnormalities in patients with biopsy-proven myocarditis [2]; here, however, the echocardiography was also normal. The patient had no obvious signs of ongoing infection and he had normal laboratory findings. Thus, myocarditis was an uncertain diagnosis in this case.

There was no electrolyte imbalance and the thyroid status was normal. There were no signs of ischaemia during the exercise test. The patient was a non-smoker and did not drink coffee or use alcohol. Thus, there was no obvious cause for the PVCs in this case.

A less well-known cause of PVCs is exposure to exhaust fumes from vehicles. Some studies have shown increased premature atrial complexes and PVCs in patients with implanted cardioverter defibrillators when exposed to high concentrations of air pollutants [3,4]. In addition to reported associations between PVCs and air pollution, there are also studies showing the association between air pollution and atrial fibrillation [5] and supraventricular extrasystoles and supraventricular tachycardia [6].

Most of the studies so far concerning arrhythmia and air pollution are on subjects with known heart disease; however, one study of healthy young non-smoking male highway patrol troopers in the United States has shown an increased number of premature supraventricular beats and changes in HRV [7].

The mechanism by which air pollution leads to cardiac morbidity and mortality remains unknown. Hypoxia has been suggested as a possible cause of air pollution-induced cardiac damage or vulnerability, but this has been shown not to be a culprit mechanism [8]. Pope et al. [9] suggest that it is due to changes in cardiac autonomic function, reflected by changes in mean heart rate and HRV. The inhaled environmental particles may promote a systemic sympathetic stress response, causing the heart rate to increase, the HRV to decrease and the ratio of low-frequency to high-frequency to be higher (as in the present case), causing ventricular tachyarrhythmias and ventricular fibrillation [8]. In a similar manner the inhaled particles also stimulate irritant receptors in the

lung parenchyma and respiratory airways, which can lead to increased bronchoconstriction, increased vagal responses of the heart, increased HRV and increased high-frequency domain, possibly leading to bradyarrhythmias in appropriate settings [8].

A link has been demonstrated between carbon monoxide from vehicles and ventricular arrhythmias [3,10], but there are also studies that have shown that carbon monoxide has no significant effect on ventricular electrical stability or the frequency of ventricular ectopic activity [11,12].

In the present case it would have been of interest to obtain an ambulatory ECG during a motorbike ride in order to report changes in autonomic factors or changes in cardiac electrical instability in the myocardial substrate (as measured by T-wave alternans). This could not be done in the present case but could perhaps be performed in the future in a similar case. Moreover, while the subject did not report feeling stressed one cannot exclude the possibility that noise and stress contributed to the findings, and measures of cortisol, for instance using salivary cortisol, would have settled this issue. This was not possible in this case but may also be performed in the future if a similar case is noted.

Conclusion

Studies have shown an association between air pollution and PVCs and other kinds of arrhythmias. The mechanism may be changes in cardiac autonomic function, including heart rate and HRV. Air pollution should be considered when patients present with arrhythmias and no other causes are found.

Abbreviations

ECG; electrocardiogram; HRV: heart rate variability; PVC: premature ventricular complex.

Competing interests

The author declares that they have no competing interests.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Acknowledgements

The author would like to thank Dr Derek Filbey for his linguistic revision of the manuscript.

References

1. Olgin JE, Zipes DP: **Specific arrhythmias: diagnosis and treatment.** In *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine* 7th edition. Edited by: Zipes DP, Libby P, Bonow RO, Braunwald E. Philadelphia, PA: Elsevier Saunders; 2005:840-841.

2. Magnani JW, Dec GW: **Myocarditis: current trends in diagnosis and treatment.** *Circulation* 2006, **113**:876-890.
3. Dockery DW, Luttmann-Gibson H, Rich DQ, Link MS, Schwartz JD, Gold DR, Koutrakis P, Verrier RL, Mittleman MA: **Particulate air pollution and nonfatal cardiac events. Part II. Association of air pollution with confirmed arrhythmias recorded by implanted defibrillators.** *Res Rep Health Eff Inst* 2005, **124**:83-126.
4. Rich DQ, Schwartz J, Mittleman MA, Link M, Luttmann-Gibson H, Catalano PJ, Speizer FE, Dockery DW: **Association of short-term ambient air pollution concentrations and ventricular arrhythmias.** *Am J Epidemiol* 2005, **161**:1123-1132.
5. Rich DQ, Mittleman MA, Link MS, Schwartz J, Luttmann-Gibson H, Catalano PJ, Speizer FE, Gold DR, Dockery DW: **Increased risk of paroxysmal atrial fibrillation episodes associated with acute increases in ambient air pollution.** *Environ Health Perspect* 2006, **114**:120-123.
6. Berger A, Zareba W, Schneider A, Rückerl R, Ibalid-Mulli A, Cyrus J, Wichmann HE, Peters A: **Runs of ventricular and supraventricular tachycardia triggered by air pollution in patients with coronary heart disease.** *J Occup Environ Med* 2006, **48**:1149-1158.
7. Riediker M, Devlin RB, Griggs TR, Herbst MC, Bromberg PA, Williams RW, Cascio WE: **Cardiovascular effects in patrol officers are associated with fine particulate matter from brake wear and engine emissions.** *Part Fibre Toxicol* 2004, **1**:2.
8. Stoen PH, Godleski JJ: **First steps toward understanding the pathophysiologic link between air pollution and cardiac mortality.** *Am Heart J* 1999, **138**:804-807.
9. Pope CA III, Verrier RL, Lovett EG, Larson AC, Raizenne ME, Kanner RE, Schwartz J, Villegas M, Gold DR, Dockery DW: **Heart rate variability associated with particulate air pollution.** *Am Heart J* 1999, **138**:890-899.
10. Dockery DW, Luttmann-Gibson H, Rich DQ, Link MS, Mittleman MA, Gold DR, Koutrakis P, Schwartz JD, Verrier RL: **Association of air pollution with increased incidence of ventricular tachyarrhythmias recorded by implanted cardioverter defibrillators.** *Environ Health Perspect* 2005, **113**:670-674.
11. Verrier RL, Mills AK, Skornik WA: **Acute effects of carbon monoxide on cardiac electrical stability.** *Res Rep Health Eff Inst* 1990, **35**:1-14.
12. Dahms TE, Younis LT, Wiens RD, Zarnegar S, Byers SL, Chaitman BR: **Effects of carbon monoxide exposure in patients with documented cardiac arrhythmias.** *J Am Coll Cardiol* 1993, **21**:442-450.

Publish with **BioMed Central** and every scientist can read your work free of charge

"BioMed Central will be the most significant development for disseminating the results of biomedical research in our lifetime."

Sir Paul Nurse, Cancer Research UK

Your research papers will be:

- available free of charge to the entire biomedical community
- peer reviewed and published immediately upon acceptance
- cited in PubMed and archived on PubMed Central
- yours — you keep the copyright

Submit your manuscript here:

http://www.biomedcentral.com/info/publishing_adv.asp

