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Environmental Pollution as a Cause of Papillary Fibroelastoma: Hints for a New Etiological Hypothesis?

Marisa De Feo,[1] Giovanni Dialetto,[1] Veronica D'Oria,[1] Paolo Pepino,[2] Salvatore Giordano,[2] Maurizio Cotrufo,[2] Luca Salvatore De Santo[3]

[1]Department of CardioThoracic Sciences, Second University of Naples, Naples, Italy; [2]Department of Cardiovascular Surgery, Pineta Grande Hospital, Caserta, Italy; [3]Department of Cardiac Surgery, University of Foggia, Foggia, Italy

ABSTRACT

Background: Environmental pollution has adverse human health effects, mostly on the respiratory tract but also on the cardiovascular system. Papillary fibroelastomas are exceedingly rare primary cardiac tumors; their pathologic origin remains still elusive.

Case Report: This is a brief report on 3 patients referred for surgical treatment of papillary fibroelastomas in the last 6 months. All patients were born, lived, and worked in a narrow region in Campania that is under active monitoring because of high rates of environmental pollution.

Conclusions: Known mechanisms of cardiovascular damage generated by environmental pollution are cross linked with described papillary fibroelastomas' etiological cascade. Evidence is suggested for a common origin. These results provide intriguing but inconclusive insights into pathophysiological pathways that may link exposure to environmental pollution and development of papillary fibroelastomas.

INTRODUCTION

Substantial epidemiological evidence suggests that environmental and air pollution have adverse human health effects. Although many studies have focused on respiratory health end points, there is growing evidence that particulate matter (PM) is a risk factor for cardiovascular disease [Chen 2008].

Primary cardiac tumors are rare. The reported prevalence in autopsy varies among authors. It ranges from 0.0017% to 0.28% and 0.02% to 0.33%, and in a clinical series where echocardiography was used to make the diagnosis, it was 0.019%. Papillary fibroelastomas (PFE) are exceedingly rare primary cardiac tumors and affect the aortic, mitral, tricuspid, and pulmonary valves, in this order of frequency. PFE are benign and slow growing, but their clinical manifestations may be fatal due to the potential for cerebral, pulmonary, and coronary circulation embolization, implying the need for surgical resection in symptomatic patients. PFE's pathologic origin remains elusive, and several etiological hypotheses have been postulated [Mariscalco 2010].

Here we describe a series of 3 patients referred for PFE to our institution in the last 6 months. All had been born and were living in a narrow geographical area that is under constant monitoring because of high levels of environmental pollution.

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Correspondence: Luca Salvatore De Santo, Viale Colli Aminei 491, 80131 Naples, Italy; 00390817062520; fax: 00390815464594 (luca.desanto@ospedalemonaldi.it).

CASE REPORT

Naples and Caserta are overpopulated neighboring provinces in the south of Italy. During the last 20 years, a definite border area, including 24 municipalities of the southern part of Caserta and the northern side of Naples, has been heavily polluted by illegal waste disposal practices, namely dumping of toxic waste and illegal burning of both toxic and solid urban waste. Available evidence, mainly deriving from criminal trials, suggests that these illegal practices have been operating in the area at least since the 1980s. The World Health Organization in conjunction with the Italian Ministry of Health and National Council for Research decided to monitor the state of health of the population and to investigate the all cause mortality rates along with the incidence of malignancies and congenital malformation (CM). Recently released data from the World Health Organization revealed that that this area has a high PM pollution and that there is an increase of all cause mortality as high as 43%. Such a mortality rate was partly due to an excess of malignancies affecting the lung, pleura, stomach, bladder, kidney, and liver. A significant increase of CM was also disclosed. Subsequent epidemiological analyses detected a tendency toward clustering of several cancer sites and CMs (namely, cardiovascular, urogenital, and limb malformations) in this fairly well defined area. These results were adjusted for the possible confounding effect of socioeconomic deprivation [Comba 2006; Fazzo 2008].

This series includes 3 patients (2 women, mean age 58 years), born, living, and working in the above described area, who were referred for symptomatic PFEs (2 aortic and 1 mitral valve involvement). All patients were hypertensive with mild coronary artery disease and showed chronic obstructive pulmonary disease (COPD) needing medical treatment.

One patient had already undergone total thyroidectomy for adenocarcinoma. They all experienced an uneventful postoperative course after shave resection. Diagnosis of PFE was confirmed by pathology examination.

DISCUSSION

This report of 3 cases suggests that long-term exposure to environmental pollution may be involved in the development of PFE.

Although it is challenging to make empirical observations relating to potential mechanistic pathways of disease from epidemiologic evidence, the results of this report are largely consistent with the pathophysiological pathways that link long-term PM exposure and cardiopulmonary mortality on one side and etiological hypotheses on PFE on the other.

Biological mechanisms linking cardiopulmonary mortality and particulate air pollution include pulmonary and systemic inflammation, accelerated atherosclerosis, and altered cardiac autonomic function. A growing body of knowledge supports the hypothesis that fine particulate air pollution may provoke alveolar inflammation, resulting in the release of potentially harmful cytokines and increased blood coagulability [Pope 2000]. Several other findings support the notion that particulate pollution may be associated with changes resulting from vasoconstriction and endothelial cells' toxic damage and activation [Pope 2004].

On the other side, 2 known PFE etiological hypotheses deserve a consideration in this context: organized thrombosis and posttraumatic-degenerative process. The organized thrombosis hypothesis is based on the histochemical presence of fibrin, hyaluronic acid, and laminated elastic fibers within the fronds of the tumor [Edwards 1991; Shapiro 2001]. The observation that these tumors are more frequently found in older patients with longstanding heart disease supports the theory that PFEs are secondary to wear and tear and may represent a posttraumatic tumor or a degenerative process. Reported correlation with previous cardiac surgery and radiation damage adds further support to this latter hypothesis [Mariscalco 2010].

These results provide intriguing but inconclusive insights into pathophysiological pathways that may link exposure to environmental pollution and PFE, but the uncommon cluster of origin and presentation and the coexistence of lung and cardiovascular chronic diseases make this series worthy of mention. Further epidemiological and molecular studies are needed to confirm this empirical observation.

REFERENCES

Pope CA 3rd. 2000. Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? Environ Health Perspect 108:713-23.

Pope CA 3rd, Burnett RT, Thurston GD, et al. 2004. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 109:71-7.

Chen H, Goldberg MS, Villeneuve PJ. 2008. A systematic review of the relation between long-term exposure to ambient air pollution and chronic diseases. Rev Environ Health 23:243-97.

Comba P, Bianchi F, Fazzo L, et al. 2006. Cancer mortality in an area of Campania (Italy) characterized by multiple toxic dumping sites. Ann N Y Acad Sci 1076:449-61.

Edwards FH, Hale D, Cohen A, Thompson L, Pezzella AT, Virmani R. 1991. Primary cardiac valve tumors. Ann Thorac Surg 52:1127-31.

Fazzo L, Belli S, Minichilli F, et al. 2008. Cluster analysis of mortality and malformations in the Provinces of Naples and Caserta (Campania Region). Ann Ist Super Sanita 44:99-111.

Mariscalco G, Bruno VD, Borsani P, Dominici C, Sala A. 2010. Papillary fibroelastoma: insight to a primary cardiac valve tumor. J Card Surg 25:198-205.

Shapiro LM. 2001. Cardiac tumours: diagnosis and management. Heart 85:218-22.