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Passive smoking and aortic arch calcification in older Chinese never smokers: The Guangzhou Biobank Cohort Study

Lin Xu [a,b], Chao Qiang Jiang [a], Tai Hing Lam [b,*], G Neil Thomas [c], Wei Sen Zhang [a], Kar Keung Cheng [c]

[a] Guangzhou No.12 Hospital, Guangzhou 510620, China
[b] Department of Community Medicine, School of Public Health, University of Hong Kong, Hong Kong, China
[c] Department of Public Health and Epidemiology, University of Birmingham, Birmingham, UK

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ABSTRACT

Objective: To study whether passive smoking is a risk factor for aortic arch calcification (AAC) among never smokers.
Background: We have previously reported that active smoking increases the risk of AAC, but the effect of passive smoking has not been reported.
Methods: We used baseline data of the Phase 1 Guangzhou Biobank Cohort Study (GBCS). 7702 older Chinese never smokers from the Phase 1 GBCS were included. Information on passive smoking and potential confounders were collected by standardized interviews and laboratory assays. AAC was diagnosed from chest X-ray by two experienced radiologists. Unconditional logistic regression was used to estimate odds ratios of AAC for passive smoking with adjustment for potential confounders.
Results: In women, the risk for aortic arch calcification (AAC) increased significantly with increasing duration of adulthood passive smoking exposure at home, at work and total duration of adulthood home and work exposure [adjusted odds ratio 1.24 (95% confidence interval 1.09–1.41) for high level of total exposure] (*P* for trend from 0.012 to 0.001). For passive smoking at home, at work and total exposure, significant trends of increasing severity of AAC with increasing duration of exposure were observed in men and women combined (*P* for trend from 0.05 to 0.002).
Conclusion: Passive smoking is a risk factor for aortic arch calcification. Studies of passive smoking and AAC, especially in developing countries can generate important local evidence to raise awareness and to support public health measures to protect non-smokers from second-hand smoke.
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1. Introduction

Based on numerous studies, mostly from Western populations in developed countries, many reviews, such as the 2006 US Surgeon General's have concluded that passive smoking causes respiratory ill health, lung cancer, coronary heart disease and many other health problems [1]. However, studies on the serious health hazards from passive smoking from developing countries are scanty, resulting in low public awareness. Likewise authoritative reviews from governments are rare and thus tobacco control measures protecting the public from second-hand smoke are weak. A 2007 report from the Chinese Ministry of Health has estimated that about 100,000 people are killed by passive smoking in China each year, and 540 million Chinese are exposed to second-hand smoke [2]. This great number of deaths attributable to second-hand smoke, which is likely to be an under-estimate, has not aroused much concern in the people and the press. More recent studies on Chinese have shown that passive smoking is associated with stroke,[3] chronic obstructive pulmonary disease (COPD)[4] and peripheral arterial disease [5]. From the Guangzhou Biobank Cohort Study, we have reported that passive smoking is associated with COPD with a dose–response relationship, and the association, if causal, suggests that 1.9 million excess deaths from COPD among never smokers could be attributable to passive smoking in the current population in China [4]. We have also found that active smoking is associated with aortic arc calcification (AAC) and also exhibits a dose–response relationship [6]. Our literature search found no report on passive smoking and AAC.

AAC is independently associated with coronary heart disease [7] and has a high specificity for the detection of severe coronary atherosclerosis in patients with coronary artery disease [8]. Most of the calcification within the aorta is detected in the aortic arch [9]. The appearance of aortic arch calcification (AAC) seen on chest radiograph usually presents in a strip, crust or pitch arc form of high radiographic density changes. We have found a high prevalence of AAC in older Southern Chinese (40.6%) in the Guangzhou Biobank Cohort Study, and 73.1% of the subjects with AAC were never smokers [9]. We

* Corresponding author. Tel.: +852 2819 9287; fax: +852 2855 9528.
E-mail address: hrmrlth@hkucc.hku.hk (T.H. Lam).

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hereby present the first report on the association between passive smoking and aortic arch calcification among never smokers, taking into account both exposures at home and indoor workplaces.

2. Methods

2.1. Study population

The Guangzhou Biobank Cohort Study has been described previously [10] and details of the methods on the analysis of passive smoking have been described in our paper on passive smoking and COPD [4]. Briefly, the Guangzhou Biobank Cohort Study is a population-based study that recruited older people aged from 50 to 85 years from Guangzhou, China. All of the subjects are permanent residents of Guangzhou. Subjects were randomly selected in a sampling frame constituted by the "Guangzhou Zunlao Xiehui" ("Guangzhou Health and Happiness Association for the Respectable Elders"). The present study used the baseline data from Phase 1 of the Guangzhou Biobank Cohort Study, which took place during November 2003 to November 2004 when 10413 subjects (3064 men and 7349 women) were recruited. The Guangzhou Biobank Cohort Study was approved by the medical ethics committee of Guangzhou Medical Association and written informed consent was collected from all participants.

2.2. Demographic characteristics and radiographic evaluation

Trained interviewers collected information using a structured standardized questionnaire and face-to-face interview. All subjects received a medical check-up including measurement of fasting plasma vascular risk factors. Most of the subjects (99.0%) had a plain chest radiograph from a Toshiba KSO-15R machine. Based on these radiographs the presence of aortic arch calcification was diagnosed by two radiologists who were blinded to the subjects' exposure. Details of the methods have been reported elsewhere [6,9]. The length and width of calcification plaque was used as an indicator of aortic arch calcification severity. In subjects with plaques at more than one location, the total length of calcification was calculated by adding the lengths of the differently located calcification plaques. The severity of aortic arch calcification was classified into 3 calcification grades (Grade 0 indicated no aortic arch calcification; grade I was defined as length of calcification plaque <10.0 mm and width of calcification plaque <4.0 mm; grade II was defined as length of calcification plaque ≥ 10.0 mm or width of calcification plaque ≥ 4.0 mm) [11]. 300 radiographs were randomly selected for re-diagnosis by the two specialist radiologists. The inter-observer consistency was satisfactory ($\kappa = 0.68$, $P<0.001$).

Participants were classified as smokers (including current smokers and former smokers) and never smokers. Among subjects who had chest X-ray, 7702 who had never smoked cigarette were included in the present study.

2.3. Passive smoking exposure assessment

We followed the methods previously described in our paper investigating the effects of passive smoking on COPD to assess exposure to passive smoking at home and at indoor workplaces in terms of intensity and duration of each exposure [4]. The intensity of exposure was defined as the number of smokers (none, one, two or more) during exposure. Subjects who had passive smoking exposure were then asked about how many hours per week and for how many years they received the exposure. In the analysis on indoor workplace exposure, 867 subjects who had never worked indoors were thus excluded. We calculated the number of hours of adulthood exposure at home and in indoor workplace, and the cumulative exposure by combining adulthood home and work exposure. The unit of exposure was set as the number of exposure years, assuming that 40 hours (h) of exposure per week for 52 weeks constituted one exposure year. Different cut-off points such as 30 h per week or 50 h per week were used in sensitivity analysis. No evidence was found to suggest that the results would be affected by such different cut-off points, so the medium level of 40 h per week was chosen to categorise levels of passive smoking exposure. The level of exposure was coded into low (none or less than 2 exposure years), medium (2–5 exposure years) and high (more than 5 exposure years).

2.4. Statistical analysis

We adjusted for potential confounders as shown in Table 1. These included age, sex, education (primary or below; middle school; and college or above), physical activity as assessed by the International Physical Activity Questionnaire [12] (IPAQ, physically active was defined as having vigorous activity at least 3 days a week, achieving at least 1500 metabolic equivalent (MET) minutes per week or activity on 7 days of the week achieving at least 3000 MET minutes per week), alcohol drinking (never and ever), body mass index (BMI, kg/m^2) and waist circumference (WC, cm), blood pressure (mm Hg), fasting total cholesterol (mmol/l), high density lipoprotein cholesterol (HDL-C, mmol/l), triglyceride (TG, mmol/l) and plasma glucose (FPG, mmol/l).

The χ^2 test was used for the comparison of categorical variables, and ANOVA for continuous variables. The independent relationship between the presence of AAC and passive smoking in never smokers was analysed using unconditional logistic regression, giving odds ratios (ORs), and adjusted for potential confounders. Linear trends for increasing odds ratios of AAC, including presence of AAC and severity of AAC, by levels of passive smoking exposure were tested. All analysis was performed using SPSS 15.0.

3. Results

3.1. Subject characteristics

Table 1 shows that consistent with prior studies, AAC in our sample was more common in women (40.28%) than in men (35.22%) [7,13,14]. The prevalence of AAC increased with age and diastolic blood pressure both in men and in women ($P<0.001$). The prevalence of adulthood exposure to passive smoking at home was more common in women (men: 15.4%, women: 61.2%) and exposure in the workplace was more common in men (71.3% versus 45.5%), whereas childhood home exposure was similar (49.8% versus 52.6%, Table 2).

3.2. Association of passive smoking with the presence and severity of AAC

No association was found between AAC and passive smoking as assessed by the number of smokers during exposure in both men and women (table not shown). Table 3 shows that in women, after adjusting for multiple potential confounders, the risk of AAC was significantly

Table 1
Characteristics of 7702 never smokers by aortic arch calcification (AAC) and sex.

Presence of AAC (number of subjects)	Men			Women		
	No (642)	Yes (349)	P[a]	No (4008)	Yes (2703)	P[a]
Age (SD), years	65.5 (5.3)	68.8 (5.9)	<0.001	62.2 (5.6)	65.9 (5.9)	<0.001
Education (>primary school, %)	494 (76.95)	254 (74.78)	0.33	1861 (46.43)	1137 (41.70)	0.001
Occupation (manual, %)	264 (41.12)	136 (38.97)	0.72	2771 (69.14)	1905 (70.48)	0.18
Drinking (%)						
Never	283 (44.08)	167 (47.85)	0.16	2544 (63.49)	1802 (66.69)	0.011
Former	256 (39.88)	118 (33.81)		1154 (28.80)	685 (25.50)	
Current	103 (16.04)	64 (18.34)		309 (7.71)	211 (7.81)	
Physical activity (IPAQ)						
Regular activity (%)	382 (59.50)	213 (61.03)	0.52	2674 (66.72)	1728 (63.93)	0.060
Waist circumference						
Female >80 cm; Male >90 cm, %	136 (21.38)	78 (22.61)	0.66	2014 (50.46)	1373 (51.21)	0.54
Body mass index (>25.0 kg/m^2, %)	211 (32.92)	114 (32.76)	0.96	1445 (36.28)	918 (34.24)	0.088
Total cholesterol (SD), mmol/l	5.51 (1.10)	5.55 (1.08)	0.37	5.91 (1.14)	6.21 (1.16)	<0.001
Triglyceride (SD), mmol/l	1.54 (1.02)	1.55 (1.23)	0.88	1.66 (1.11)	1.67 (1.15)	0.99
High density lipoprotein (SD), mmol/l	1.56 (0.36)	1.59 (0.36)	0.016	1.72 (0.34)	1.74 (0.35)	0.015
Diastolic blood pressure (SD), mm Hg	77.41 (11.4)	77.42 (11.8)	0.99	74.37 (11.1)	74.20 (11.1)	0.52
Systolic blood pressure (SD), mm Hg	134.3 (21.1)	138.7 (23.0)	<0.001	132.1 (22.1)	136.7 (22.7)	<0.001

[a] P for the difference between presence of AAC (Yes/No).

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