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Original Article

Lung Function Profiles among Individuals with Nonmalignant Asbestos-related Disorders

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ABSTRACT

Background: Inhalation of asbestos fibers can lead to adverse health effects on the lungs. This study describes lung function profiles among individuals with nonmalignant asbestos-related disorders (ARDs).
Methods: The study population was from the Workers' Compensation (Dust Diseases) Board of New South Wales, Sydney, Australia. Lung function measurements were conducted in males with asbestosis ($n = 26$), diffuse pleural thickening (DPT; $n = 129$), asbestosis and DPT ($n = 14$), pleural plaques only ($n = 160$) and also apparently healthy individuals with a history of asbestos exposure ($n = 248$). Standardized spirometric and single-breath diffusing capacity for carbon monoxide (DL_{CO}) measurements were used.
Results: Mean age [standard deviation (SD)] was 66.7 (10.3) years for all participants. Current and ex-smokers among all participants comprised about 9.0% and 54.8%, respectively. Median pack-years (SD) of smoking for ex- and current-smokers were 22.7 (19.9). Overall 222 participants (38.6%) and 139 participants (24.2%) had forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) measurements < 80% predicted, and 217 participants (37.7%) had FEV_1/FVC results < 70%. A total of 249 individuals (43.8%) had DLco values < 80% predicted and only 75 (13.2%) had DLco/VA results < 80% predicted. A total of 147 participants (25.6%) had peak expiratory flow (PEF) measurements < 80% predicted. The presence of ARDs lowered the lung function measurements compared to those of healthy individuals exposed to asbestos.
Conclusion: Lung function measurement differs in individuals with different ARDs. Monitoring of lung function among asbestos-exposed populations is a simple means of facilitating earlier interventions.

1. Introduction

Asbestos is a naturally occurring mineral fiber which is known as a human carcinogen [1]. More than 100,000 people die annually in occupational settings due to asbestos-related diseases caused by inhalation of asbestos fibers [2]. Diseases related to asbestos exposure mostly affect the respiratory system, but in rare cases they affect other locations such as the ovaries and larynx [1]. Reduced lung function among individuals with a history of asbestos exposure is common and has been well recognized [3–6]. Although substantial evidence for impairment of lung function is already described in populations exposed to asbestos, many studies have included cohorts of asbestos-exposed workers, with fewer comparisons between asbestos-exposed individuals without disease and those with asbestos-related disorders (ARDs). The use of lung function measurements to screen ARDs is still widely employed as a primary screening tool.

Several recent reports have warned that ARDs in Asian countries could increase in the near future [7–9] because asbestos consumption has been high since the late 1980s and into the 1990s [10].

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Caution is necessary in ensuring that an accurate diagnosis of ARD is made prior to monitoring of lung function. Nevertheless, adverse health impacts from asbestos exposure are not well recognized in this region. The purpose of this study was to determine the lung function profiles among individuals with nonmalignant ARDs.

2. Materials and methods

2.1. Study population

We obtained data from a study population that is well-described elsewhere [11]. In brief, the study was conducted at the Workers' Compensation (Dust Diseases) Board of New South Wales, Sydney, Australia. This study had approval from the Human Research Ethics Committee of St. Vincent's Hospital, Sydney, Australia. All participants gave their written informed consent and were not compensated for their participation. Participants were diagnosed with their respective ARDs according to the American Thoracic Society (ATS) criteria [12]. Criteria for asbestosis included a history 25 fiber/mL-years exposure to asbestos, the presence of bilateral fine end-inspiratory crackles on auscultation, and the presence of subpleural interstitial opacities on chest radiology (usually high resolution computer tomography scan), in the absence of other causes of interstitial pulmonary fibrosis. Criteria for the diagnosis of diffuse pleural thickening (DPT) were involvement of >25% of the chest wall on plain chest radiology, 8 cm × 5 cm × 3 cm in total on chest computer tomography, and/or the presence of Blesovsky's syndrome [13]. Pleural plaques were diagnosed according to their presence on chest radiograph or computer tomography scan, and included calcified and non-calcified circumscribed pleural thickening.

In New South Wales, compensation for ARDs is provided by the Dust Diseases Board (DDB) according to the Workers' Compensation (Dust Diseases) Act 1942. Individuals with known or suspected "dust diseases" are referred to the DDB and undergo a standardized occupational history documenting asbestos exposure, clinical examination, lung function testing, and medical imaging as required. Information is then reviewed by a Medical Authority consisting of at least three appropriately qualified thoracic physicians. A diagnosis is reached or declined, and an assessment of disablement made according to American Medical Association IV criteria [14].

2.2. Pulmonary function assessment

Forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), percent ratio between FEV_1 and FVC (FEV_1/FVC), single-breath carbon monoxide diffusing capacity (DLco) and DLco/alveolar volume (DLco/VA), and peak expiratory flow (PEF) were measured by spirometry using the Sensormedics Vmax 22D-Spectra (Viasys Healthcare, Conshohocken, PA, USA). During the study period, the same equipment was used by the same trained technician. At least three FVC manoeuvres and DLco trials were obtained for each participant according to ATS guidelines [15,16]. The results were expressed as absolute values and as percent of the predicted values for FVC, FEV_1, and DLco calculated on the basis of age, height, and gender for Caucasians, using the predictive equations provided by the European Respiratory Society 1993 update [17].

2.3. Data analysis

Descriptive statistics were performed for outcome and predictor variables and covariates. Analysis of variance test and generalized liner models (GLM) were used to compare means, while adjusting for confounding factors, namely age, smoking pack-years, and body mass index (BMI). The GLM procedure uses the method of least squares and analyzes data within the framework of general linear models, and in this case, we use the models not only to compare two or more means, but also as linear regressions, to predict the effect of a factor on an outcome variable. All comparisons were two-sided and $p < 0.05$ was treated as significant. Analyses were performed in SAS, version 10 (SAS Institute Inc., Cary, NC, USA).

3. Results

3.1. Study population

Baseline characteristics of the participants in this study are shown in Table 1. Data analyzed from the 577 males comprised the following categories: asbestosis ($n = 26$), DPT ($n = 129$), asbestosis and DPT ($n = 14$), pleural plaques only ($n = 160$, PPs) and apparently healthy individuals formerly exposed to asbestos ($n = 248$). Mean age (SD) was 66.7 (10.3) years for all participants. Current and ex-smokers among all participants comprised about 9.0% and 54.8%, respectively. Median pack-years (SD) of smoking for current and ex-smokers were 22.7 (19.9). A total of 469 participants (81.3%) were overweight and obese (BMI ≥ 25).

3.2. Pulmonary function measures

Pulmonary function outcome measures are described in Table 2. Out of the sample population, 222 participants (38.6%) and 139 participants (24.2%) had FEV_1 and FVC measures that were <80% predicted, respectively, and 217 participants (37.7%) had FEV_1/FVC

Table 1
Characteristics of participants by asbestos-related disorder category

Characteristic	All	Asbestos-related disorder category					
		Healthy*	Asbestosis	DPT†	Asbestosis/DPT	Pleural plaques	p‡
Subjects included in final analysis [n (%)]	577 (100)	248 (43.0)	26 (4.5)	129 (22.4)	14 (2.4)	160 (27.7)	
Age [Mean (SD)] (y)	66.7 (10.3)	60.9 (10.5)	72.7 (6.7)	71.8 (6.8)	72.9 (6.7)	69.3 (8.9)	<0.0001
Smoking status (%)							
Ex-smoker	54.8	42.3	61.5	73.6	85.7	55.0	<0.0001
Current smoker	9.0	11.7	11.5	5.4	0	8.1	
Never smoker	36.2	46.0	26.9	20.9	14.3	36.9	
Pack years [mean (SD)]	22.7 (19.9)	21.0 (18.2)	24.8 (14.4)	23.5 (18.8)	15.9 (18.0)	24.5 (23.7)	0.0076
Body mass index, kg/m^2 [n (%)]							
18.5–24.9	105 (18.2)	46 (18.6)	3 (11.5)	17 (13.2)	2 (14.3)	37 (23.1)	0.2027
25.0–29.9	294 (51.0)	117 (47.2)	17 (65.4)	69 (53.5)	10 (71.4)	81 (50.6)	
≥30	175 (30.3)	83 (33.5)	6 (23.1)	43 (33.3)	2 (14.3)	41 (25.6)	

ANOVA, analysis of variance.
* Apparently healthy with a history of asbestos exposure.
† Diffuse pleural thickening.
‡ For significance testing: Chi-square tests were performed for proportions and ANOVA analyses for arithmetic means.

Table 2
Unadjusted lung function profiles among participants with asbestos-related disorders

Lung function measures	All	Asbestos-related disorder category					
		Healthy*	Asbestosis	DPT†	Asbestosis/DPT	Pleural plaques	p‡
FEV_1	84.3 (19.4)	92.5 (17.2)	78.4 (14.0)	69.5 (16.4)	68.6 (11.7)	85.9 (18.4)	<0.0001
FVC	92.5 (17.5)	100.2 (14.6)	81.9 (15.8)	79.4 (16.0)	71.4 (13.2)	94.7 (14.9)	<0.0001
FEV_1/FVC	70.7 (10.0)	72.6 (8.7)	73.6 (9.3)	67.2 (10.8)	73.9 (10.1)	69.8 (10.5)	<0.0001
DLco	82.6 (20.0)	90.6 (18.9)	59.6 (14.1)	72.7 (16.6)	62.2 (17.8)	83.6 (17.7)	<0.0001
DLco/VA	102.5 (20.5)	103.2 (18.6)	90.4 (23.6)	104.2 (22.5)	98.0 (25.2)	102.2 (20.4)	0.031
PEF	93.6 (22.0)	100.5 (20.3)	94.1 (19.9)	80.8 (21.7)	80.4 (19.3)	94.4 (20.4)	<0.0001

ANOVA, analysis of variance; DLco, diffusing capacity for carbon monoxide; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; PEF, peak expiratory flow; SD, standard deviation.
Data are presented as mean (SD).
* Apparently healthy with a history of asbestos exposure.
† Diffuse pleural thickening.
‡ For significance testing: ANOVA analyses for arithmetic means.

results that were <70% predicted. A total of 249 participants (43.8%) had DLco values < 80% predicted and only 75 participants (13.2%) had DLco/VA results < 80% predicted. A total of 147 participants (25.6%) had PEF measures of <80% predicted (Table 2). Mean (SD) FEV_1% predicted levels in individuals with asbestosis [78.4 (14.0)], DPT [69.5 (16.4)], asbestosis/DPT [68.6 (11.7)] and PPs [85.9 (18.4)] were significantly lower than the apparently healthy individuals formerly exposed to asbestos [92.5 (17.2)] ($p < 0.0001$). Mean (SD) FVC% predicted levels in individuals with asbestosis [81.9 (15.8)], DPT [79.4 (16.0)], asbestosis/DPT [71.4 (13.2)] and PPs [94.7 (14.9)] were significantly lower than the apparently healthy individuals with a history of asbestos exposure [100.2 (14.6)] ($p < 0.0001$). Mean (SD) DL_{CO}% predicted levels in participants with asbestosis [59.6 (14.1)], DPT [72.7 (16.6)], asbestosis/DPT [62.2 (17.8)] and PPs [83.6 (17.7)] were significantly lower than the apparently healthy participants formerly exposed to asbestos [90.6 (18.9)] ($p < 0.0001$). Comparison of spirometry and diffusion capacity outcomes between the healthy individuals and individuals with ARDs revealed statistically significant differences in mean percent predicted for all of these parameters (Table 2).

Age, smoking pack-years, and BMI were included in GLM models to adjust for their effects on the lung function profiles of the healthy and ARD groups (Table 3) which was similar to the unadjusted profiles reported in Table 2. The apparently healthy participants with a history of asbestos exposure had better lung function than those with ARDs, and differences across all groups were statistically significant ($p < 0.0001$, Table 3). Interestingly, when compared to the unadjusted lung function profiles, controlling for the effects of these three independent variables results in an overall reduction in the lung function only among the healthy participants. The unadjusted mean (SD) of FEV_1, FVC, FEV_1/FVC, DLco, DLco/VA, and PEF predicted values in the healthy group were 92.5 (17.2), 100.2 (14.6), 72.6 (8.7), 90.6 (18.9), 103.2 (18.6), and 100.5 (20.3), respectively, whereas the adjusted mean (SD) of FEV_1, FVC, FEV_1/FVC, DLco, DLco/VA, and PEF predicted values in the healthy group were 90.4 (15.6), 98.8 (12.1), 70.8 (10.2), 87.6 (16.2), 101.9 (16.8), and 98.2 (18.4), respectively. Of the six lung function measures, the lowest mean values recorded were evenly distributed among the participants with asbestosis, DPT, and asbestosis/DPT.

4. Discussion

Although the best way of elimination of ARDs is simply to ban asbestos usage, new cases of mesothelioma, the worst health effect of asbestos exposure, continue to rise worldwide, due to the long latency period between exposure and disease development [18,19]. Effective screening of ARDs is essential for the better management of ARDs. Asbestos exposure is mostly associated with lung function impairments [3–6]. Lung function has also been proven to be the best predictor of survival of asbestos exposure workers [20].

The current study found significant differences between ARDs across all lung function measures, in agreement with previous studies. The presence of pleural plaques has been significantly associated with respiratory impairment in some studies [21], but not in others. Male asbestos-exposed individuals with DPT showed lower DL_{CO} and FVC than individuals without a history of asbestos exposure alone, but the FEV_1/FVC values were not different [22]. In a study from India, workers exposed to asbestos had a mean FVC <80% of the predicted value [23]. Abejie and colleagues [24] found chrysotile exposed workers in China had significantly reduced FVC, FEV_1, FEV_1/FVC, and DL_{CO}, and workers with asbestosis had lower FVC and DL_{CO} values but not FEV_1/FVC values, compared to workers

Table 3
Adjusted lung function profiles among participants with asbestos-related disorders

Lung function measures	All	Asbestos-related disorder category					
		Healthy*	Asbestosis	DPT†	Asbestosis/DPT	Pleural plaques	p‡
FEV_1	85.6 (20.2)	90.4 (15.6)	80.8 (15.4)	72.1 (17.9)	69.5 (13.0)	86.6 (19.1)	<0.0001
FVC	94.2 (18.7)	98.8 (12.1)	83.5 (16.9)	81.3 (17.4)	72.5 (14.3)	95.0 (15.2)	<0.0001
FEV_1/FVC	71.4 (11.6)	70.8 (10.2)	75.6 (9.8)	68.9 (11.4)	75.1 (12.3)	70.7 (11.2)	<0.0001
DLco	84.8 (22.3)	87.6 (16.2)	63.1 (16.5)	75.3 (19.1)	64.7 (18.9)	85.5 (18.4)	<0.0001
DLco/VA	104.6 (22.5)	101.9 (16.8)	92.1 (25.2)	105.0 (23.2)	98.4 (25.8)	103.4 (21.6)	<0.0001
PEF	95.2 (23.9)	98.2 (18.4)	96.8 (21.3)	83.3 (24.2)	81.2 (21.4)	95.4 (21.3)	<0.0001

ANOVA, analysis of variance; DLco, diffusing capacity for carbon monoxide; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; PEF, peak expiratory flow; SD, standard deviation.
Data are presented as mean (SD); adjusted by age, smoking status, BMI.
* Apparently healthy with a history of asbestos exposure.
† Diffuse pleural thickening.
‡ For significance testing: ANOVA analyses for arithmetic means.

with asbestos exposure but without asbestosis. A similar finding in China was also reported by Wang et al. [25], who reported a significant drop in FVC and DL_{CO} in 248 workers exposed to mainly chrysotile.

Our findings support previous data that lung function in individuals with ARDs is significantly lower than currently healthy asbestos-exposed individuals. In our study, important factors which affect lung function such as smoking history, age, and BMI were adjusted for [26,27], but interestingly, controlling for the effects of these three independent variables did not have a large effect on lung function in those with ARDs, only on those who were apparently healthy, possibly due to confounding by severity. It has been known that cumulative exposure of asbestos is strongly associated with lung function decline [28]. The lack of exposure assessment was a limitation in the study, but unfortunately the data was not collected.

In conclusion, occupational and/or environmental exposure to asbestos is strongly related to an increased decline in lung function. Our study shows significant differences in lung function in individuals with ARDs compared to currently healthy individuals with a history of previous exposure to asbestos. Regular monitoring of lung function among asbestos-exposed populations is a simple tool to facilitate earlier interventions.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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