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Exposure to Brake Dust and Malignant Mesothelioma: A Study of 10 Cases with Mineral Fiber Analyses

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Objectives: A large number of workers in the USA are exposed to chrysotile asbestos through brake repair, yet only a few cases of malignant mesothelioma (MM) have been described in this population. Epidemiologic and industrial hygiene studies have failed to demonstrate an increased risk of MM in brake workers. We present our experience of MM in individuals whose only known asbestos exposure was to brake dust and correlate these findings with lung asbestos fiber burdens.

Methods: Consultation files of one of the authors were reviewed for cases of MM in which brake dust was the only known asbestos exposure. Lung fiber analyses were performed using scanning electron microscopy (SEM) in all cases for which formalin-fixed or paraffin-embedded lung tissue was available.

Results: Ten cases of MM in brake dust-exposed individuals were males aged 51–73 yr. Nine cases arose in the pleura and one in the peritoneum. Although the median lung asbestos body count (19 AB/g) is at our upper limit of normal (range 0–20 AB/g), half of the cases had levels within our normal range. In every case with elevated asbestos fiber levels by SEM, excess commercial amphibole fibers were also detected. Elevated levels of chrysotile and non-commercial amphibole fibers were detected only in cases that also had increased commercial amphibole fibers.

Conclusions: Brake dust contains exceedingly low levels of respirable chrysotile, much of which consists of short fibers subject to rapid pulmonary clearance. Elevated lung levels of commercial amphiboles in some brake workers suggest that unrecognized exposure to these fibers plays a critical role in the development of MM.

Keywords: asbestos; auto repair; brake dust; chrysotile; fiber analysis; mechanic; mesothelioma; occupation; peritoneum; pleura

INTRODUCTION

It is estimated that nearly 1000000 workers are involved in installing and repairing clutch facings and brake shoes and linings (Huncharek, 1990). Because these automotive friction materials contain asbestos, there is concern that brake repair workers are at increased risk for developing malignant mesothelioma (MM). Numerous studies have examined airborne asbestos fiber concentrations during brake maintenance operations, demonstrating measurable levels for considerable periods of time and at distances extending several meters from the actual operation (Hickish and Knight, 1970; Lorimer *et al.*, 1976; Rohl *et al.*, 1976; Levine, 1978; Rodelsperger *et al.*, 1986; Kauppinen and Korhonen, 1987; Moore, 1988; Roggli and Pratt, 1988). Despite the large number of such workers, only a few cases of MM in individuals exposed to brake dust have been described, mostly in the form of case reports (Newhouse and Thompson, 1965; Langer and McCaughey, 1982; Jarvholm and Brisman, 1988; Huncharek *et al.*, 1989).

Analyses of brake dust have shown that it contains <1% asbestos, most of which is short chrysotile fibers <1 μm in length, with much of the asbestos having been converted to forsterite due to the heat generated during the braking process (Williams and Muhlbaier, 1982). Conclusions on the relationship between brake dust exposure and MM have been based predominantly on epidemiological studies, which have

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shown no increased risk of MM resulting from exposure to automotive friction products (McDonald and McDonald, 1980; Teta *et al.*, 1983; Spirtas *et al.*, 1985, 1994; Woitowitz and Rodelsperger, 1994; Teschke *et al.*, 1997; Agudo *et al.*, 2000). A meta-analysis of epidemiologic data concluded that the relative risk of MM in brake workers was 0.9, indicating no excess risk above that expected in the general population (Wong, 2001).

Studies correlating exposure with the actual concentration of asbestos fibers in the lungs of brake repair workers with MM are sparse (Langer and McCaughey, 1982; Woitowitz and Rodelsperger, 1994; Roggli *et al.*, 2002). Most of these reports have included cases with substantial exposure to asbestos through other types of employment. We describe our findings, including data from lung fiber analyses, in individuals with MM whose only known exposure to asbestos was to automotive friction materials.

MATERIALS AND METHODS

The 10 cases selected for the current study were obtained from one of the authors (V.L.R.) consultation files, which contain data on more than 1900 cases of MM. These cases were all medico-legal consultations, which included five referred by plaintiffs and five by defendants of brake manufacturers. The diagnosis of MM was based on the gross distribution of tumor, histologic appearance and the results of histochemical and immunohistochemical studies using previously described criteria (Roggli *et al.*, 1992a). In all cases, the diagnosis of MM was made independent of asbestos exposure history and tissue mineral fiber content. Information regarding age, sex, primary tumor site, occupation, smoking history and duration of exposure was also obtained. Occupational exposure information was obtained by direct patient interview and thorough review of the medical records. Only cases in which occupational contact with brake dust was the sole recognized source of asbestos exposure were included in our study.

Fiber analyses were performed on formalin-fixed or paraffin-embedded lung parenchyma using the sodium hypochlorite digestion procedure, as previously described (Roggli *et al.*, 1992b). Digested lung tissue was collected on 0.4 μm pore size Nuclepore filters. For light microscopic analysis, the filter was mounted on a glass slide. Asbestos bodies were quantified using a magnification of 400×. Only ferruginous bodies exhibiting typical morphology with thin, translucent cores were counted as asbestos bodies (Roggli, 1992a). Results were reported as asbestos bodies per gram wet lung tissue (AB/g), with a detection limit of ~3 AB/g for a 0.3 g sample (Roggli, 1992b). For scanning electron microscopic (SEM) analysis, the filter was mounted on a carbon disc with colloidal graphite and then sputter-coated with gold. A JEOL JSM-6400 scanning electron microscope (JEOL, Peabody, MA) with a screen size of 22.7 × 17.3 cm was used to quantify uncoated fibers and AB at a screening magnification of 1000×. Only fibers ≥5 μm in length with a length to width ratio of at least 3:1 and approximately parallel sides were counted. Fibers meeting these criteria were quantified by examining 100 consecutive fields, with a total area of ~2.53 mm^2, or until a 200 fiber count was reached. The thinnest fibers we have observed at this screening magnification are ~0.15 μm in diameter. The limit of detection is ~400 fibers/g for a 0.3 g sample (Roggli, 1992b). For cases in which no asbestos fibers were detected, the value was reported as less than the detection limit for that case.

The chemical composition of fibers was determined by energy dispersive X-ray analysis. Asbestos fibers were classified as commercial amphiboles, specifically amosite + crocidolite (AC), non-commercial amphiboles, including tremolite, anthophyllite and actinolite (TAA), or chrysotile (Roggli *et al.*, 1992b). Tissue concentration of AC, TAA and chrysotile was calculated in each case using the proportion of each type of fiber and the total asbestos fiber concentration. Non-asbestos mineral fibers (NAMF) were classified according to their morphology and X-ray spectra (Roggli *et al.*, 1992b). For cases in which no fibers of a particular category were detected, the value was reported as less than the detection limit for that case. The results of fiber analysis for MM cases were compared with 20 reference cases which, as previously described, had normal lungs, no history of asbestos exposure and no evidence of asbestos-related disease at autopsy (Srebro *et al.*, 1995).

RESULTS

Ten cases of histologically confirmed MM in which the only identified asbestos exposure was to brake dust were retrieved from the files of one of the authors (V.L.R.). Salient clinical features of the cases are summarized in Table 1. All were men, nine of whom had tumors arising in the pleura and one in the peritoneum. The age ranged from 51 to 73 (median 60) yr. Information regarding smoking was available in seven cases. All seven smoked or were ex-smokers. The length of exposure to brake dust ranged from 7 to 40 (median 24) yr. All patients had direct exposure to brake dust as automotive/brake mechanics.

The predominant histologic pattern was epithelial in five cases, while three were biphasic and one showed desmoplastic features. Information regarding tumor type was not available in one case. Pleural plaques were present in four of seven informative cases. Asbestosis was not evident histologically in any case.

Table 1. Demographic, pathologic and occupational information

Case	Age (yr)/sex	Tumor type/site	Occupation	Smoking (pack-yr)	Exposure duration (yr)	Pleural plaque
1	61/M	E/PL	Auto machinist	2; XS	37	Yes
2	58/M	E/PL	Brake mechanic	43	27	Yes
3	55/M	E/PL	Brake mechanic	60	24	ND
4	73/M	B/PL	Auto mechanic	20+	40	No
5	51/M	E/PE	Brake repair	XS	11	ND
6	53/M	D/PL	Auto mechanic	ND	7	No
7	ND/M	ND/PL	Brake repair	ND	15	ND
8	66/M	B/PL	Brake repair	XS	40	Yes
9	71/M	B/PL	Auto repair	21; XS	17	Yes
10	ND/M	E/PL	Brakeline repair	ND	24	No

B, biphasic; D, desmoplastic; E, epithelial; M, male; ND, not determined; PE, peritoneal; PL, pleural; XS, ex-smoker.

Table 2. Results of lung tissue analysis

Case	AB/g[a]	AC[b]	TAA[b]	Chrysotile[b]	NAMF[b]
1	1490	3270	2180	2180	9800
2	25	4810	440	<440	1750
3	50	380	4630	<660	660
4	<5	<720	720	<720	2880
5	24	<580	1160	<580	5780
6	13.4	490	490	<490	980
7	9.1	340	<340	<340	340
8	2.6	120	240	<120	850
9	560	6000	3280	2180	6550
10	14	1440	2170	720	2890
Median	19	440	940	<620	2320
Reference cases[c]	3 (<0.2–22)	<600 (<100–<2540)	<600 (<170–2540)	<600 (<100–1000)	<600 (210–10 160)

AB, asbestos bodies; AC, commercial amphiboles (amosite + crocidolite); NAMF, non-asbestos mineral fiber; TAA, non-commercial amphiboles (tremolite + anthophyllite + actinolite).

[a]AB/g, asbestos bodies/g wet lung by light microscopy.

[b]Total coated (AB) and uncoated fibers ≥5 μm (length)/g wet lung as determined by scanning electron microscopy and energy dispersive X-ray analysis.

[c]Median values and range (in parentheses) for 20 cases with normal lungs at autopsy, no history of asbestos exposure or evidence of asbestos-related disease (Srebro *et al.*, 1995).

The results of the fiber analyses are summarized in Table 2. The median asbestos body count in brake dust-exposed individuals (19 AB/g) is at our upper limit of normal (range 0–20 AB/g) (Roggli *et al.*, 1992b). In five of the 10 cases, the asbestos body content was within our normal range (cases 4, 6–8 and 10). Non-commercial amphiboles, principally tremolite, with some actinolite and anthophyllite, predominated over commercial amphibole fibers in half of the cases and were elevated in two cases (cases 3 and 9). Excess commercial amphibole fibers were detected in five of the six cases with elevated tissue asbestos content (cases 1–3, 9 and 10). Amosite was the principal commercial amphibole in four of these cases and crocidolite predominated in the fifth (case 9). In one additional case (case 5), asbestos body counts by light microscopy were marginally elevated, but neither commercial amphibole fibers nor chrysotile were detected and non-commercial amphibole fibers were within our normal range by SEM. Chrysotile was detected in three cases and exceeded our normal range in two of these cases (cases 1 and 9). All cases with excess chrysotile or non-commercial amphibole fibers also showed increased levels of commercial amphibole fibers. Pleural plaques were present in four (cases 1, 2, 8 and 9). Commercial amphibole fibers were detected in all four of these cases and elevated in three. Non-asbestos mineral fibers included among others talc, silica, miscellaneous silicates and rutile. These were within our normal range in all cases.

DISCUSSION AND CONCLUSIONS

By virtue of its heat resistance and tensile strength, asbestos has found an application in the manufacture

of brake and clutch products (Greenberg and Darcy, 1992). Since the 1940s, the only form of asbestos used in automotive friction materials is chrysotile (Langer and McCaughey, 1982). Chrysotile is a major component of friction materials, accounting for 30–80% of the finished product (Greenberg and Darcy, 1992). However, according to most studies, the concentration of respirable chrysotile liberated by various brake maintenance operations, such as 'blowing out' brake surfaces and grinding brake shoes, is low (Lynch, 1968; Hickish and Knight, 1970; Lorimer *et al.*, 1976; Rodelsperger *et al.*, 1986). In a number of industrial hygiene reports detailing asbestos exposures among automotive mechanics, the concentration of airborne asbestos has been below the Occupational Safety and Health Administration's permissible exposure limit (PEL) of 0.1 fiber/cm^3 (National Institute for Occupational Safety and Health, 1980a,b; Cheng and O'Kelly, 1986; Rodelsperger *et al.*, 1986; Kauppinen and Korhonen, 1987).

The low level of airborne chrysotile liberated during brake repair is consistent with the results of analyses of brake dust samples (Williams and Muhlbaier, 1982). Chrysotile fibers in unworn brake linings are entirely in a bonded or encapsulated state. During braking, temperatures exceeding 700–800°C are reached. Much of the chrysotile is broken down into forsterite, a non-asbestos anhydrous magnesium silicate (Williams and Muhlbaier, 1982; Wong, 2001). Of the small number of residual intact chrysotile fibers, most remain embedded in the plastic binding material. Less than 0.1–1% of the fibers in brake dust are free chrysotile, the majority of which are short fibers, <1 μm in length (Lynch, 1968; Hatch, 1970; Davis and Coniam, 1973; Williams and Muhlbaier, 1982; Wong, 2001). Of note, a considerably higher concentration (2–15%) of free chrysotile has been detected in used brake linings (Rohl *et al.*, 1976). However, this material may not be representative of brake dust particles that become airborne (Williams and Muhlbaier, 1982).

There has been considerable debate regarding the relative pathogenicity of chrysotile. Evidence from a number of studies on workers exposed to chrysotile suggests that chrysotile asbestos poses a lower risk of MM than does amphibole asbestos (McDonald and McDonald, 1978; Wagner *et al.*, 1982; Churg *et al.*, 1984; McDonald *et al.*, 1984; Dunnigan, 1988; McDonald, 1988; Roggli and Pratt, 1988; Newhouse and Sullivan, 1989). The relative low potency for inducing MM has been ascribed to the low pulmonary retention of chrysotile (Churg *et al.*, 1989; Mossman and Gee, 1989; Churg and Vedal, 1994). Fiber size also appears to play an important role in the development of MM. Experimental animal studies have shown a very low fibrogenic and carcinogenic potential for short asbestos fibers (Wagner *et al.*, 1974; Stanton *et al.*, 1981; Davis and Jones, 1988). This finding is of particular relevance in the case of brake workers, as the majority of chrysotile fibers in brake dust are <1 μm in length (Williams and Muhlbaier, 1982).

A number of studies have examined workers with exposure to chrysotile through the mining and milling of asbestos. Non-commercial amphibole fibers are natural contaminants of Canadian chrysotile. Lung fiber burden analyses performed many years after exposure show that non-commercial amphibole levels often exceed chrysotile concentrations (Churg and Warnock, 1980; Churg and Wiggs, 1986). Among the few lung fiber burden analyses performed in workers exposed solely to chrysotile in brake dust, Langer found only chrysotile fibers (1 μg/5 g wet lung), the overwhelming majority of which were <10 μm in length (Langer and McCaughey, 1982). However, in other cases, only amphibole fibers have been identified (Woitowitz and Rodelsperger, 1994). In the present series, the lung fiber burden of workers whose only identified asbestos exposure was to chrysotile in brake dust was within our normal range in half of the analyzed cases. The other analyzed cases showed increased levels of commercial amphiboles in all but one. The few cases with elevated chrysotile or non-commercial amphibole fibers also had elevated commercial amphiboles.

There are a number of limitations of the present study. First of all, the study is a case series of medicolegal cases, and may not be representative of all individuals exposed to brake dust occupationally. Secondly, historical information obtained by patient interview is subject to the limitations of recall bias for events that occurred decades previously. This may explain the absence of reported exposures to commercial amphiboles in our study. Thirdly, there is the possibility that long chrysotile fibers with diameters <0.15 μm were missed by the use of SEM at a screening magnification of 1000×. However, we think that this is unlikely, since a number of investigators using the somewhat more sensitive transmission electron microscopy method have found no correlation between the concentration of chrysotile asbestos and the risk of mesothelioma in individuals exposed to both chrysotile and commercial amphibole fibers (McDonald *et al.*, 1989; Churg and Vedal, 1994; Rodelsperger *et al.*, 1999).

A more troublesome issue is the possibility that chrysotile fibers might be able to interact with mesothelial cells, induce neoplastic transformation and then be removed from lung, leaving no trace of the initial excess fiber burden. In this regard, it should be noted that there is no disagreement regarding the greater persistence of the amphibole fibers compared to chrysotile, nor is there disagreement regarding the greater potency of amphiboles for producing mesothelioma. Furthermore, experimental studies with

man-made mineral fibers indicate that it is the fibers that persist within the lung that cause fibrosis and neoplasia (Hesterberg *et al.*, 1994, 1995, 1996). There are no studies in experimental animals indicating that chrysotile can cause disease and subsequently be completely cleared from the lungs. Thus the 'hit-and-run' hypothesis for chrysotile is a flimsy one without any solid scientific support.

Lung burden analyses in automotive brake repair workers with MM in our series reflect tissue asbestos content within the normal range or elevated commercial amphiboles. These findings, combined with data from prior lung fiber analyses (Langer and McCaughey, 1982; Woitowitz and Rodelsperger, 1994), industrial hygiene studies (Hickish and Knight, 1970; Lorimer *et al.*, 1976; Rohl *et al.*, 1976; Levine, 1978; Rodelsperger *et al.*, 1986; Kauppinen and Korhonen, 1987; Moore, 1988; Roggli and Pratt, 1988) and epidemiological reports (Wong, 2001), strongly suggest that friction product exposure, such as that encountered by automotive mechanics, is unlikely to contribute to the development of MM. The presence of elevated commercial amphiboles in the lungs of some brake workers indicates that unrecognized asbestos exposure through other forms of employment plays a confounding role in the development of MM in this population. Other cases have tissue asbestos contents indistinguishable from background controls and may be considered to be spontaneous or idiopathic.

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