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# Role of Asbestos burden in Lung Disease in north Indian population

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**Abstract:**

**Background:**Malignancies in pulmonary system has been a matter of concern but. Its etiology is hardly established pathologically since asbestos is known to be carcinogenic and highly persistent. So we had planned to study the burden of asbestos in patients of carcinoma lung and pleura.

**Material & method**:Transbronchial biopsy was taken via bronchoscopy from the suspected lung cancer patients to evaluate asbestos fiber burden. Biopsy sample were send to Department of Pathology, Career Institute of Medical Sciences, Lucknow, in formalin for histopathological examination and slides from pathology department and biopsy tissue were send to IITR for particle and fibre burden estimation.

**Results**: Mean number of asbestos fibre which came out in different biopsy tissues was $9.3x10^4$/gm of tissue. In which most common type was amphibole fibers. The mean of total, chrysotile and amphibole counts in age group <50 years is more than that of >50 years patients. Association between gender and fibre load reveals that mean of total, chrysotile and amphibole fibers counts in male patients are more than female patients. Risk prone occupationally exposed patients where having mean fibers (total, chrysotile and amphibole) more than that of no risk patients. Endobronchial type of biopsy tissues had mean fibre (total chrysotile and amphibole) more than other types of biopsies.

**Discussion:** Association between histopathological type and fibre load did not showed any statistically significant difference in different histopathology of lung cancer however maximum values were obtained in squamous cell carcinoma patients and minimum for small cell carcinoma.

**Conclusion:** As we see there is no significant result came out as far as asbestos burden and lung malignancy are concerned. However, the burden of the asbestos fibre has to be reviewed in larger studies so that any inference can be drawn for the source of this fibre in non occupationally exposed lung cancer patients.

**Keywords:** Asbestous, Lung pleura, Lung carcinoma, Association, Fibre load

**Introduction:**The major health hazards associated with asbestos are fibrogenicity and carcinogenicity, with lung cancer and mesothelioma as the main representatives. It has been shown that the risk of developing mesothelioma or lung cancer increases proportionally to the

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asbestos fibre burden of the lungs, and also the extent of pleural plaques has been found to be associated with fibre numbers in lung tissue. Thus fibre clearance and biopersistence are considered the most important factors for diagnosis and risk assessment of malignant and non-malignant diseases. Asbestos lung burden is the only indicator of cumulative lifetime exposure that can be measured reliably in a population-based study.

The first evidence of pulmonary fibrosis in association with asbestos use was reported at the turn of the century.Asbestos is a carcinogenic mineral fiber. Its occupational exposure and associated diseases (Lung cancer, ovarian cancer asbestosis) has been well documented globally as well as nationally. 17% of deaths in workers suffering from asbestosis were associated lung cancer and a figure which had risen to nearly 50% of deaths in 1961-3 [1]. In 1955 the first mortality study of asbestosis workers had shown 11 deaths from lung cancer associated with asbestosis, and none without [2].

The latency period from first asbestos exposure to the manifestation of an asbestos-induced disease may be 10–60 years with shorter times for asbestosis and plaques and longer times for lung cancer and mesothelioma. Thus an asbestos-related disease might occur a long time after exposure cessation. Therefore, the question of the traceability of asbestos fibres in lungs tissue after prolonged intervals is of significance, e.g. for the differentiation of asbestosis and further fibrotic lung diseases, such as idiopathic pulmonary fibrosis (IPF) [3, 4]. In this context lung dust analysis is considered a valuable tool [5, 6]. Asbestos fibre analyses in the lungs of asbestos-exposed workers have a long tradition in establishing a link between exposure and disease [7].

Malignancies in pulmonary system has been a matter of concern but. Its etiology is hardly established pathologically since asbestos is known to be carcinogenic and highly persistent. In biological system, efforts need to be made to establish any correlation of non occupational exposure of asbestos with its chronic effects in pulmonary and its surrounding systems. So we had planned to study the burden of asbestos in patients of carcinoma lung and pleura

**Material & methods:**

**Study design:** Single centre cross sectional study evaluated in August 2019 to July 2020**.** All suspected cases of lung and pleural malignancy were taken. Indoor and Outdoor patients of Pulmonary Medicine Department, of Career Institute of Medical Sciences, Lucknow. The study subjects (suspected cases of lung and pleural malignancy) were selected from OPD and Indoor patients of Career Institute of Medical Sciences, Lucknow. Their history personal as well as occupational was taken on prestructuredproforma.After detailed history, examination and investigation. Biopsy samples were taken via different procedures like:

- CT guided FNAC/Biopsy
- Bronchoscope biopsy/Bronchoalveolar lavage.
- Pleural biopsy closed/Open (Thoracoscopic)

Transbronchial biopsy was taken via bronchoscopy from the suspected lung cancer patients to evaluate asbestos fiber burden. Biopsy sample were send to Department of Pathology, Career Institute of Medical Sciences, Lucknow, in formalin for histopathological examination and

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slides from pathology department and biopsy tissue were send to IITR for particle and fibre burden estimation.

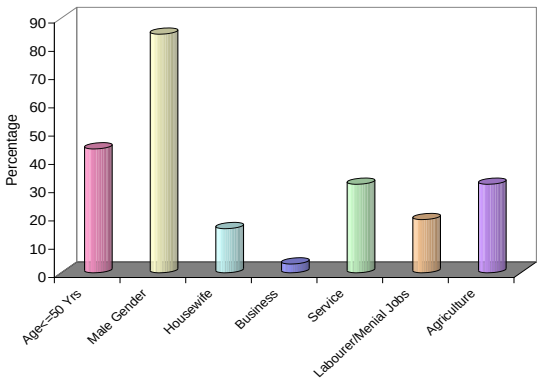
- Biopsy tissues pathology was done as per standard procedure and pathologists collaboration.
- Tissues (8-10 pg.) stained for pathological investigation was used for the localizations of particles and fibers in the tissue reasons. This was done through application of polarized and phase optical microscopy.
- Its fraction of tissue (-1 gm) was digested to remove organic content and isolation of particles and fibers following the method of (HAQ *et al.*) the data was expressed as number of particles/fibers per gram of tissue.

**Statistical Tools Employed**

The statistical analysis was done using SPSS (Statistical Package for Social Sciences) Version 15.0 statistical Analysis Software. The values were represented in Number (%) and Mean±SD. Mann Whitney U test was applied for the raw data from samples A and B must first be combined into a set of $N=n_a+n_b$ elements, which are then ranked from lowest to highest, including tied rank values where appropriate. These rankings are then re-sorted into the two separate samples.

**Results:**The present study was carried out at Department of Pulmonary Medicine and The Fiber Toxicology Division,CSIR – Indian Institute of Toxicology Research,Lucknowto evaluate the burden of asbestos on the lung and bronchial tissue among patients of different malignant and non-neoplasmic lesions of pulmonary system. Total 78 patients biopsy samples were send for fiber burden estimation.Due to smaller size of biopsy tissue fiber burden was analyzed only in 32 patients.

**Figure no. 1 shows the demographic characteristics of the patients.**




Age of patients ranged from 28 to 82 years. Mean age of patients was 54.09±13.42 years. Majority of patients were above 50 years of age (n=18; 56.3%).A total of 27 subjects (84.4%) were males and remaining 5 (15.6%) were females. The male to female ratio of the subjects was 5.4:1.Agriculture was the most common occupation (31.3%). An equal number (n=10, 31.3%) of subjects were in service *viz.* clerks, teachers, surveyors, *etc.* There were 6 (18.8%) subjects who were labourers/doing menial jobs such as labourers, drivers, construction work, hawker, *etc.* A total of 5 (15.6%) *i.e.* all the female subjects were housewives. One (3.1%) patient was doing business.

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
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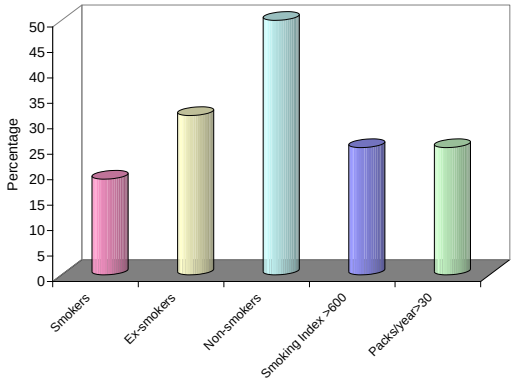
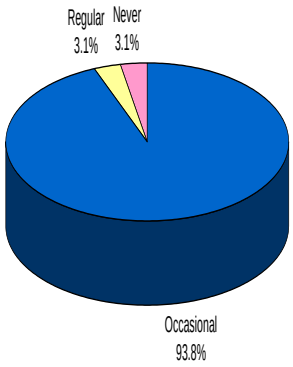
**Figure no. 2: Smoking Status of the Patients**

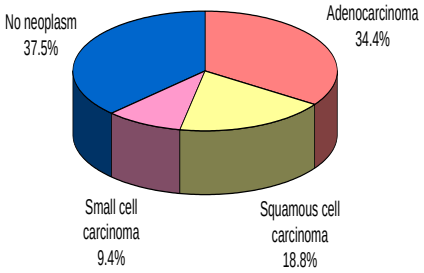



A total of 16 (50%) subjects were non-smokers, 6 (18.8%) were current smokers and 10 (31.3%) were ex-smokers. The smoking index ranged from 10 to 1200 with a mean value of 600.63±373.66. A total of 8 (25%) subjects had smoking index >600.Consumption of packs per year ranged from 1 to 60 with a mean value of 30.02±18.70. A total of 8 (25%) subjects used to consume >30 packs per year.

Distribution of subjects according to talcum powder use has been shown in Table 3 below:

**Figure no 3 : Talcum Powder Use (Occasional-< once in a week, regular-> once in a week), and figure no.4 Histopathological Findings of patients**




Most of the patients (n=30; 93.8%) were occasional users of talcum powder. There was 1 (3.1%) patient who was using it regularly for the last 30 years while 1 (3.1%) patient had never used it.

Maximum patients were non-neoplasmic (n=12; 37.5%) followed by those having Adenocarcinoma (34.4%), squamous cell carcinoma (18.8%) and small cell carcinoma (9.4%).Stagewise distribution of subjects has been shown in Table 5 below:

**Figure no 5 shows Histopathological Stage of the patients and figure no. 6 Distribution of subjects according to biopsy type has been shown**

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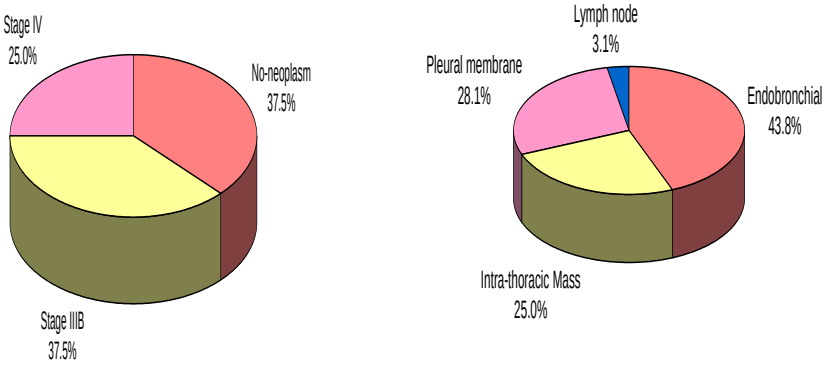
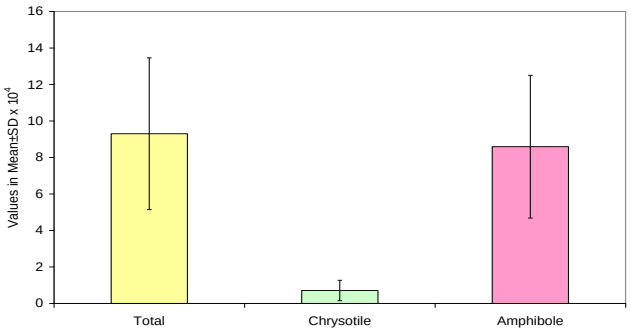



A total of 12 (37.5%) patients had non-malignant lesions and an equal number (n=12; 37.5%) had Stage IIIB of malignancy. One-quarter (25%) of patients had Stage IV of malignancy.

Maximum (n=14; 43.8%) specimen were taken by Endobronchial biopsy, followed by pleural biopsy in (n=28.1%) CT guided biopsy involving intrathoracic mass (n=8; 25%). In one (3.1%) case biopsy was done at lymph node.

**Table no. 7: shows calculation of Asbestos burden in the biopsy specimen**
$x10^4$/gm tissue




Total fibre load ranged from 2.29-22.30x$10^4$fibers per gm area of tissue with a mean value of 9.30±4.16x$10^4$.
In this fibre load the count of Chrysotile fibers ranged from 0.06-2.68x$10^4$ with a mean value of 0.71±0.56x$10^4$.
Number of amphibole fibers ranged from 2.09-19.63x$10^4$ with a mean value of 8.59±3.91x$10^4$.

**Table no 8: % Distribution of fibers with different sizes**
% Distribution

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
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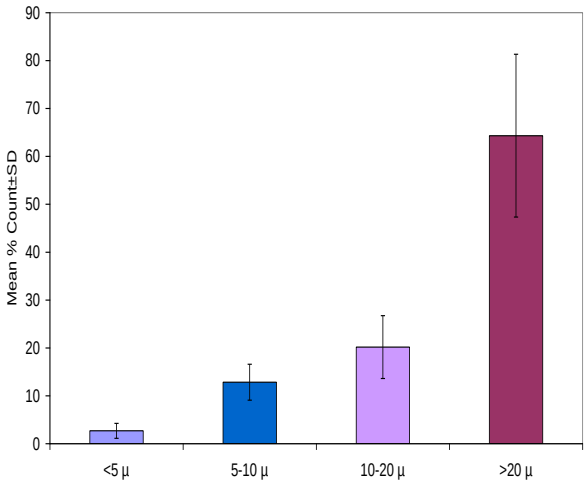


Maximum proportion of fibers was>20 µm in size (64.31±7.05%) while those having size <5 µm were minimum (2.68±1.55%).

## ASSOCIATIONS

Association of fibre load as well as fibre size was done for various demographic, histopathological type, stage and biopsy tissue types.

### I. Fibre Load

**Table 9 shows association between age and fibre load:**

*Values are x10$^4$ count*

| SN | Variable | Age ≤50 years (n=14) | | Age >50 years (n=18) | | Significance of difference (Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | z | p |
| 1. | Total | 9.50 | 4.21 | 9.14 | 4.24 | 0.323 | 0.750 |
| 2. | Chrysotile | 0.75 | 0.44 | 0.67 | 0.65 | 1.064 | 0.301 |
| 3. | Amphibole | 8.76 | 4.16 | 8.46 | 3.82 | 0.114 | 0.925 |

Comparison of total, chrysotile and amphibole counts in agegroups <50 years and >50 years revealed that mean count either for total or for chrysotile and amphibole did not differ significantly between two age groups (p>0.05).

**Table 10: Association between gender and fibre load**

*Values are x10$^4$ count*

| SN | Variable | Male (n=27) | | Female (n=5) | | Significance of difference(Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | z | P |
| 1. | Total | 9.35 | 4.23 | 9.00 | 4.25 | 0.363 | 0.725 |

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
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|---|---|---|---|---|---|---|---|
| 2. | Chrysotile | 0.75 | 0.59 | 0.46 | 0.22 | 1.116 | 0.285 |
| 3. | Amphibole | 8.60 | 3.90 | 8.53 | 4.42 | 0.234 | 0.841 |

Comparison of total, chrysotile and amphibole counts in males and females revealed that mean count either for total or for chrysotile and amphibole did not differ significantly between two genders ($p>0.05$).

**Table 11: Association between Occupation and Fibre Load**

*Values are x10$^4$ count*

| SN | Occupation | n | Total Fibre | | Chrysotile | | Amphibole | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD |
| 1. | Housewife | 5 | 9.00 | 4.25 | 0.46 | 0.22 | 8.53 | 4.42 |
| 2. | Business | 1 | 5.60 | . | 0.45 | . | 5.15 | . |
| 3. | Service | 10 | 9.94 | 5.26 | 0.81 | 0.73 | 9.13 | 4.76 |
| 4 | Labourers/Hawker | 6 | 9.76 | 4.77 | 0.54 | 0.31 | 9.23 | 4.63 |
| 5. | Agriculture | 10 | 8.89 | 3.00 | 0.85 | 0.60 | 8.04 | 2.67 |
| $\chi^2$ (Kruskall Wallis test) | | | 2.300 | | 2.025 | | 2.044 | |
| p | | | 0.681 | | 0.731 | | 0.728 | |

Although the mean total fibre as well as chrysotile and amphibole loads were minimum and much lower in subject doing business as compared to other occupational groups yet the difference among different occupational categories was not significant statistically.

**Table 11a: Association between Risk Category Occupations and Fibre Load**

*Values are x10$^4$ count*

| SN | Occupation | n | Total Fibre | | Chrysotile | | Amphibole | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD |
| 1. | No risk | 28 | 8.62 | 2.89 | 0.67 | 0.44 | 7.50 | 2.78 |
| 2. | Risk-prone | 4 | 14.1 | 8.42 | 1.00 | 1.15 | 13.1 | 7.61 |
| z (Mann Whitney U test) | | | 1.464 | | 0.293 | | 0.878 | |
| P | | | 0.333 | | 1.000 | | 0.667 | |

No significant difference between two occupation categories was observed though for all the three variables, the mean of those prone to risk was higher as compared to those who did not have a risk.

**Table 12: Association between Smoking Status and Fibre Load**

*Values are* x*10$^4$ count*

| SN | Smoking Status | n | Total Fibre | | Chrysotile | | Amphibole | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD |
| 1. | Non-smoker | 16 | 9.66 | 4.71 | 0.74 | 0.68 | 8.91 | 4.29 |
| 2. | Smoker | 6 | 11.62 | 3.94 | 0.69 | 0.26 | 10.92 | 4.08 |
| 3. | Ex-smoker | 10 | 7.33 | 2.46 | 0.66 | 0.50 | 6.67 | 2.19 |

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

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|---|---|---|---|
| $\chi^2$ (Kruskall Wallis test) | 4.015 | 0.467 | 3.606 |
| P | 0.134 | 0.792 | 0.165 |

Comparison of total fibre and amphibole levels in smokers were maximum and ex-smokers were minimum while chrysotile levels were minimum among ex-smokers and maximum among non-smokers yet the difference among different smoking strata was not significant statistically (p>0.05).

**Table 13: Association between Smoking Index and fibre load (n=16)**

*Values are x10$^4$ count*

| SN | Variable | Smoking Index <600 (n=8) | | Smoking Index >600 (n=8) | | Significance of difference (Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | z | P |
| 1. | Total | 10.45 | 4.63 | 7.43 | 1.44 | 1.785 | 0.083 |
| 2. | Chrysotile | 0.71 | 0.50 | 0.63 | 0.34 | 0 | 1 |
| 3. | Amphibole | 9.73 | 4.62 | 6.80 | 1.24 | 1.680 | 0.105 |

Comparison of total, chrysotile and amphibole counts in patients with smoking index <600 and those with smoking index >600 revealed that mean count either for total or for chrysotile and amphibole did not differ significantly between two groups (p>0.05).

**Table 14: Association between Packs per year and fibre load (n=16)**

*Values are x10$^4$ count*

| SN | Variable | Packs per year ≤25 (n=8) | | Packs per year ≥25 (n=8) | | Significance of difference (Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | z | P |
| 1. | Total | 10.45 | 4.63 | 7.43 | 1.44 | 1,785 | 0.083 |
| 2. | Chrysotile | 0.71 | 0.50 | 0.63 | 0.34 | 0 | 1 |
| 3. | Amphibole | 9.73 | 4.62 | 6.80 | 1.24 | 1.680 | 0.105 |

Comparison of total, chrysotile and amphibole counts in patients consuming ≤25 packs per year and those consuming >25 packs per year revealed that mean count either for total or for chrysotile and amphibole did not differ significantly between two groups (p>0.05).

**Table 15: Association between Biopsy type and Fibre Load**

*Values are x10$^4$ count*

| SN | Type | n | Total Fibre | | Chrysotile | | Amphibole | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD |
| 1. | Endobronchial | 14 | 10.01 | 4.82 | 0.73 | 0.75 | 9.28 | 4.37 |
| 2. | Intrathoracic | 8 | 8.61 | 4.24 | 0.70 | 0.39 | 7.91 | 4.03 |

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
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| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 3. | Lymph node | 1 | 4.46 | . | 0.18 | . | 4.28 | . |
| 4 | Pleural | 9 | 9.33 | 3.06 | 0.73 | 0.33 | 8.60 | 3.22 |
| $\chi^2$ (Kruskall Wallis test) | | | 4.243 | | 3.038 | | 4.515 | |
| P | | | 0.236 | | 0.386 | | 0.211 | |

Though, none of the differences were significant statistically (p>0.05). For all the three variables maximum value was obtained for Endobronchial biopsy and minimum for lymph node.

**Table 16: Association between Histopathological type and Fibre Load**
*Values are x10$^4$ count*

| SN | Type | N | Total Fibre | | Chrysotile | | Amphibole | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD |
| 1. | Non-neoplasmic | 12 | 9.38 | 5.45 | 0.70 | 0.74 | 8.68 | 4.89 |
| 2. | Adenocarcinoma | 11 | 9.18 | 3.69 | 0.70 | 0.52 | 8.49 | 3.57 |
| 3. | Sq cell carcinoma | 6 | 10.28 | 3.39 | 0.82 | 0.33 | 9.47 | 3.69 |
| 4 | Small cell carcinoma | 3 | 7.43 | 0.77 | 0.56 | 0.33 | 6.87 | 0.94 |
| $\chi^2$ (Kruskall Wallis test) | | | 1.054 | | 1.973 | | 0.813 | |
| P | | | 0.788 | | 0.578 | | 0.846 | |

Though, none of the differences were significant statistically (p>0.05). For all the three variables maximum value was obtained for squamous cell carcinoma and minimum for small cell carcinoma.

**Table 17: Association between Histopathological Stage and Fibre Load**
*Values are x10$^4$ count*

| SN | Type | N | Total Fibre | | Chrysotile | | Amphibole | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD |
| 1. | Non-neoplasmic | 12 | 9.38 | 5.45 | 0.70 | 0.74 | 8.68 | 4.89 |
| 2. | Stage IIIB | 12 | 9.76 | 3.37 | 0.80 | 0.44 | 8.96 | 3.41 |
| 3. | Stage IV | 8 | 8.49 | 3.36 | 0.58 | 0.43 | 7.91 | 3.32 |
| $\chi^2$ (Kruskall Wallis test) | | | 0.829 | | 1.877 | | 0.578 | |
| P | | | 0.661 | | 0.391 | | 0.749 | |

Though none of the differences were significant (p>0.05) for any of the thee variables yet the mean asbestos burden was minimum for all the three variables *viz.* total, chrysolite and amphibole count in Stage IV whereas the maximum value was observed for non-neoplasmic lesions (except Amphibole which was maximum in Stage IIIB).

**Table 18: Association between Malignancy Status and Fibre Load**

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

*Values are x10$^4$ count*

| SN | Type | N | Total Fibre | | Chrysotile | | Amphibole | |
|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD |
| 1. | Non-neoplasmic | 12 | 9.38 | 5.45 | 0.70 | 0.74 | 8.68 | 4.89 |
| 2. | Malignant | 20 | 9.25 | 3.33 | 0.71 | 0.44 | 8.54 | 3.33 |
| z (Mann-Whitney U test) | | | 0.019 | | 0.934 | | 0.039 | |
| P | | | 0.985 | | 0.366 | | 0.985 | |

No significant difference between two groups was observed (p>0.05) for any of the three variables.

**II. Proportional Size**

**Table 19: Association between age and fibre size**

*Values in % of total*

| SN | Size | Age ≤50 years (n=14) | | Age >50 years (n=18) | | Significance of difference (Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | z | P |
| 1. | <5 µm | 2.73 | 1.47 | 2.63 | 1.66 | 0.134 | 0.896 |
| 2. | 5-10 µm | 11.94 | 4.18 | 13.54 | 3.33 | 1.636 | 0.107 |
| 3. | 10-20 µm | 19.84 | 7.69 | 20.42 | 5.74 | 0.704 | 0.488 |
| 4. | >20 µm | 65.49 | 6.80 | 63.40 | 7.29 | 1.027 | 0.319 |

Comparison of mean proportion of fibers with different sizes in agegroups<50 years and >50 years revealed that mean proportion of fibers with different sizes either did not differ significantly between two age groups (p>0.05).

**Table 20: Association between gender and fibre size**

*Values in % of total*

| SN | Size | Male (n=27) | | Female (n=5) | | Significance of difference (Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | z | P |
| 1. | <5 µm | 2.61 | 1.61 | 3.04 | 1.31 | 0.939 | 0.361 |
| 2. | 5-10 µm | 13.11 | 3.83 | 11.40 | 3.29 | 0.779 | 0.448 |
| 3. | 10-20 µm | 19.64 | 6.26 | 23.00 | 8.15 | 0.961 | 0.361 |

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
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| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 4. | >20 µm | 64.64 | 7.04 | 62.56 | 7.59 | 0.416 | 0.687 |

No association between proportion of fibers of different sizes and gender was observed ($p>0.05$).

**Table 21: Association between Occupation and Fibre Size**

*Values in % of total*

| SN | Occupation | n | <5 µm | | 5-10 µm | | 10-20 µm | | >20 µm | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| 1. | Housewife | 5 | 3.04 | 1.31 | 11.40 | 3.29 | 23.00 | 8.15 | 62.56 | 7.59 |
| 2. | Business | 1 | 1.20 | . | 16.80 | . | 23.20 | . | 58.80 | . |
| 3. | Service | 10 | 2.18 | 1.40 | 13.92 | 3.69 | 18.44 | 4.37 | 65.46 | 6.31 |
| 4 | Labourers/Hawker | 6 | 2.67 | 0.88 | 12.57 | 3.83 | 15.97 | 4.69 | 68.80 | 4.55 |
| 5. | Agriculture | 10 | 3.14 | 2.08 | 12.26 | 4.16 | 22.70 | 7.72 | 61.90 | 8.22 |
| $\chi^2$ (Kruskall Wallis test) | | | 3.301 | | 3.034 | | 5.535 | | 4.837 | |
| p | | | 0.509 | | 0.552 | | 0.237 | | 0.304 | |

No significant association between occupation and fibre size was not observed ($p>0.05$).

**Table 22: Association between Smoking Status and Fibre Size**

*Values in % of total*

| SN | Smoking Status | n | <5 µm | | 5-10 µm | | 10-20 µm | | >20 µm | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| 1. | Non-smoker | 16 | 2.99 | 1.38 | 12.18 | 3.45 | 20.94 | 7.23 | 63.90 | 7.84 |
| 2. | Smoker | 6 | 2.77 | 1.61 | 10.70 | 1.67 | 16.93 | 3.92 | 69.60 | 3.36 |
| 3. | Ex-smoker | 10 | 2.12 | 1.79 | 15.20 | 4.13 | 20.88 | 6.62 | 61.80 | 6.02 |
| $\chi^2$ (Kruskall Wallis test) | | | 4.154 | | 6.569 | | 1.682 | | 6.686 | |
| P | | | 0.125 | | 0.037 | | 0.431 | | 0.035 | |

It was observed that mean proportion of fibre size between 5-10 µm was significantly higher in ex-smokers as compared to non-smokers and smokers while the mean fibre size >20 µm was significantly higher among smokers and minimum among ex-smokers. For fibre sizes <5 µm and 10-20 µm the differences among different smoking strata were not significant statistically.

**Table 23: Association between Smoking Index and fibre size (n=16)**

*Values are % of Total*

| SN | Variable | Smoking Index <600 (n=8) | | Smoking Index >600 (n=8) | | Significance of difference (Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | Z | P |

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 1. | <5 µm | 3.15 | 2.10 | 1.58 | 0.59 | 1.481 | 0.161 |
| 2. | 5-10 µm | 12.80 | 4.62 | 14.23 | 3.51 | 1.210 | 0.234 |
| 3. | 10-20 µm | 18.60 | 7.45 | 20.20 | 4.33 | 1.051 | 0.328 |
| 4. | >20 µm | 65.45 | 8.40 | 64.00 | 3.93 | 1.419 | 0.161 |

The association between smoking index and fibre size was not significant statistically ($p>0.05$).

**Table 24: Association between Packs per year and fibre size (n=16)**

*Values are % of Total*

| SN | Variable | Packs per year ≤25 (n=8) | | Packs per year ≥25 (n=8) | | Significance of difference (Mann-Whitney U test) | |
|---|---|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD | Z | P |
| 1. | <5 µm | 3.15 | 2.10 | 1.58 | 0.59 | 1.481 | 0.161 |
| 2. | 5-10 µm | 12.80 | 4.62 | 14.23 | 3.51 | 1.210 | 0.234 |
| 3. | 10-20 µm | 18.60 | 7.45 | 20.20 | 4.33 | 1.051 | 0.328 |
| 4. | >20 µm | 65.45 | 8.40 | 64.00 | 3.93 | 1.419 | 0.161 |

No significant association between fibre size and packs per year was observed.

**Table 25: Association between Biopsy type and Fibre Size**

*Values are % of Total*

| SN | Biopsy Type | n | <5 µm | | 5-10 µm | | 10-20 µm | | >20 µm | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| 1. | Endobronchial | 14 | 2.47 | 1.66 | 13.17 | 3.67 | 20.41 | 5.87 | 63.94 | 6.34 |
| 2. | Intrathoracic | 8 | 2.43 | 1.35 | 11.65 | 3.87 | 23.25 | 7.79 | 62.68 | 6.50 |
| 3. | Lymph node | 1 | 5.00 | . | 16.20 | . | 24.20 | . | 54.60 | . |
| 4. | Pleural | 9 | 2.96 | 1.55 | 13.02 | 4.06 | 16.60 | 5.62 | 67.42 | 8.02 |
| $\chi^2$ (Kruskall Wallis test) | | | 2.468 | | 1.867 | | 4.869 | | 4.805 | |
| P | | | 0.481 | | 0.601 | | 0.182 | | 0.187 | |

No significant association between fibre size and biopsy type was observed.

Table 26 shows association between histopathological type and fibre size:

**Table 26: Association between Histopathological type and Fibre Size**

*Values are % of Total*

| SN | HPE Type | n | <5 µm | | 5-10 µm | | 10-20 µm | | >20 µm | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| 1. | Non-neoplasmic | 12 | 2.75 | 1.91 | 13.27 | 3.32 | 21.28 | 5.78 | 62.70 | 7.05 |

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 2. | Adenocarcinoma | 11 | 2.36 | 1.16 | 12.24 | 4.21 | 21.87 | 7.42 | 63.53 | 5.94 |
| 3. | Sq cell carcinoma | 6 | 2.20 | 1.27 | 13.60 | 4.53 | 17.17 | 5.15 | 67.03 | 6.97 |
| 4. | Small cell carcinoma | 3 | 4.47 | 0.64 | 11.87 | 3.41 | 15.47 | 7.56 | 68.20 | 11.61 |
| $\chi^2$ (Kruskall Wallis test) | | | 0.235 | | 0.894 | | 2.737 | | 2.333 | |
| P | | | 0.889 | | 0.639 | | 0.255 | | 0.311 | |

None of the differences were significant statistically (p>0.05).

**Table 27: Association between Histopathological Stage and Fibre Size**
**% of Total**

| SN | HPE Type | n | <5 µm | | 5-10 µm | | 10-20 µm | | >20 µm | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| 1. | Non-neoplasmic | 12 | 2.75 | 1.91 | 13.27 | 3.32 | 21.28 | 5.78 | 62.70 | 7.05 |
| 2. | Stage IIIB | 12 | 2.60 | 1.15 | 11.55 | 4.02 | 19.72 | 8.63 | 66.13 | 7.78 |
| 3. | Stage IV | 8 | 2.68 | 1.69 | 14.15 | 3.82 | 19.18 | 4.15 | 64.00 | 6.04 |
| $\chi^2$ (Kruskall Wallis test) | | | 0.015 | | 3.072 | | 1.367 | | 2.005 | |
| P | | | 0.993 | | 0.215 | | 0.505 | | 0.367 | |

None of the associations were significant statistical

**Discussion:**

This study was done to evaluate the asbestos burden in pathological biopsy tissues obtained in suspected malignancy cases of lung and pleura and lymph nodes. Purpose of the study was done to evaluate the asbestos burden in lung cancer patients. In spite of few studies done on pleura and lymphnodes other studies which were done previously they were done on lung parenchyma burdenof patients. Most of the studies were done on occupational workers. Fiber burden studies done on lung parenchyma of necropsy or excised tissues from lung cancer patients.

This study is distinctive in this prospect that this is done on pathological tissues taken for diagnostic purposes in suspected lung and pleural malignancy patients. This study was done on 32 subjects of suspected malignancy cases of lung and pleura. Mean age of patients was 54.09+13.42(28-82years). In our study male subjects were more in number than the female M:F=27:5. Most of the subjects were above 50years of age.

In our study most common Occupation of the patients was agriculture, agriculture studies on non occupational exposure done by many investigators. Most of the studies were done on occupationally exposed subjects as JC Wagner et al. [8] did their study on lung of former employee of an asbestos textile factory at necropsy using transmission electron microscope.VL Roger et al.[9]done studies on non occupational IPF. Study done by V L Roger et al.reported a significant association between mesothelioma risk and lung concentration of short chrysolitefibers (<10mm). In our study we included patients not

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

occupationlly exposed and only 4 patient in labourers had risk of some asbestos exposure. As in Table l1 association between the risk category occupation and fibre load, Asbestos fibre burden.In risk prone occupation mean fibre was 14.1×104 which was more than no risk patients(mean 8.62×104f/gram). Still there was no significant difference between two occupations category was observed though for all the three variables, the mean of those prone to risk was higher as compared to those who did not have a risk.

Regarding the smoking status M.L. Warwok et al.[10] studied 35 to 82 yr patients and they divided them in three groups. The three groups did not differ significantly in mean age nor did they differ in the mean number of pack years. In our study total 50% subjects were non smokers 6 were current smokers and 31.3% were ex smokers. 30 Mean pack years (30.02+18.70), if we see in our study out of 32 patients 16 were current smokers (18.8%) Mean pack years 30.02+18.70 consumptions of pack years ranged from 1 to 60 total of 8(25%) subjects used to consume >30 pack per year. Two case control studies control studies on asbestos exposure and lung cancer had been carried out in Japan. Both studies showed that smoking had a higher risk of lung cancer than asbestos exposure [11], [12]. But it must be noted that no quantitative assessment of asbestos exposure was done and in heavily exposed cohort such as asbestosis, asbestos exposure had about 2 times of lung cancer than smoking.

There are two case series studies which demonstrated a significant content of asbestos fibers or bodies in the lungs of silicosis patients with or without lung cancer. A questionnaire survey on 885 (794 males 91 females). Asbestos workers (except textile worker) In Osaka conducted in 1983 showed that 65% was current smokers, 13% ex smokers for male and 23% current smokers and ex smoker for females.

In our study total fibers and amphibole maximum in smokers and minimum in ex smokers while chrysotile levels were minimum among ex smokers and maximum in non smokers.Yet difference among different smoking strata was not significant statistically. As Burten W.L. et al.[13] studied to examine the effects of cigarette smoking and asbestos exposure on location and histology of lung cancer they analyzed data from case control study that included 456 patients with stage I and II lung cancer. Patients with upper lobe tumor 14.6% had histology of significant asbestos exposure. The relationship between asbestos exposure and upper lobe location of tumor was also statistically significant. In their study the proportion of patients with significant exposure to asbestos was lower among those with adenocarcinoma but was not statistically significant.

In their study histology of asbestos did not predict tumor histology. In our study only 4 patients were on risk of occupational exposure. When we compare asbestos fiber load in no risk patients with risk prone patients the mean of those prone to risk was higher as compared to those who did not have a risk but if we assess statistical significance. This is not statistically significant. In our study as sample size was very less and association between risk prone and no risk patients and asbestos burden is not statistically significant we cannot comment on association between asbestos fiber burden and occupation of the patients.

In our study subjects included of age range 28 to 82 years and mean age of the patients was 15.09±13.42. In the study done by M.L. Warnok[10].they studied subjects ranged in age from 35 to 82 years they divided them in three groups. The three groups did not

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

differ significantly in mean age nor did they differ in mean number of pack years smoked. In their study the combined concentration of amosite and crocidolite fibers in 75 subjects ranged from undetectable to 69 million/gm.The range of chrysolite fiber concentration was somewhat smaller (7700 to 28 million/gm). Amosite fibers predominated over crocidolite fibers. In our study we divided patients in <50 years and >50 years mean fiber burden was more in <50 year age groups as compared with >56 years. Majority of fibers were of Amphibole type. Comparison of total, chrysolite and amphibole counts in age group <50 years and >50 years revealed that mean count either for total or for chrysolite and amphibole did not differ significantly between two age groups as YugandZin et al. [14].results shows that the medians of uncoated fibers count in different age groups increased in age dependent manner ($p<0.01$). The difference between our study & study done by YugandZin et al. is that their study was done on Lung paranchyma. In our study it was done on pathological tissue and as in our study difference was not significant between two groups. No conclusive statement can be given regarding association between age and asbestos burden in lung.

In our study subjects, males are more than females (27:5). Total asbestos burden was more in male subjects (mean 9.35±4.23) as compared to female (9.00±4.25), mean concentration of chrysolite in males 0.75 as compared to 8.53 for females. As we see study done by Yungang Lin et al. [15] to investigate the asbestos exposure level in non-occupational population and its relation to lung cancer. They included lung tissue from 65 random surgically treated lung cancer patients (42 males and 23 females) and 107 random autopsy cases. In each age group no difference in those counts between males and females was observed ($p>0.05$) moreover those counts in male lung cancer cases (median 62.5) and were greater than those in control, however, regarding the female group there was no statistically significant difference from control.

Their results suggest that environmental asbestos exposure in Hong Kong males may be one of the carcinogenic factor leading to lung cancer. In our study comparison of total, chrysolite and amphibole counts in males and females revealed that mean count either for total or for chrysolite and amphibole did not differ significantly between two groups ($p>0.05$) however mean asbestos burden is slightly higher in male patients (9.35×104 f/gm) as compared to female (9.00×104 f/gm). Chrysolite& amphiboles level was also more in male subjects than female but as difference between in these two is not statistically significant no association can be drawn between two groups.

As ML Wornick[10] studied 75 subjects and he divided them in three groups – Group 1 (white collar), Group 2 (Blue collar), Group 3 (construction worker). The median concentration of Asbestos bodies in Group 3 was 260 times that in Group 1. The median concentration of amosite was 40 times that in Group 1. Asbestos body concentration differed significantly among all three groups. For fibers classified as chrysotile the concentration in Group 3 were significantly greater than those in Group 1.

In our study total fiber burden in pathological tissue was more in risk group of occupation in which we included 4 patients who had significant history of asbestos exposure in construction work. Although there was no significant difference between two occupation categories was observed though for all the three variables the mean of those prone to risk was higher as compared to those who did not have risk.

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

If we study the association between biopsy type and fiber burden, we studied 32 subjects out of which 14 were from endobronchial growth, 8 from intrathoracic mass, 1 from lymph nodes and 9 from pleural biopsy. There are very few studies where fiber burden on pleural tissue was done and more rarely it was done in lymph nodes. YI Baris et al.[16] studied 575 inhabitants' fibers were found in the pleural tissue of two out of five cases examined. Yosunosuke Suzuki et al.[17] studied asbestos fiber in lung and mesothelial tissue taken from 151 human mesothelioma cases. The number of asbestos fibers in the lung was 45.64×106 f/gm in maximum and 0.08×106 f/gm weight in minimum. On an average 105×106 f/gmdw. In mesothelial tissue it was 240×106 f/gmdw in maximum, 0.03×106 f/g dw in minimum and 49.84×106 f/g dw on average. They told that these numbers were greater than those seen in general population.

As in our case, no asbestos burden study was done in lung paranchymal tissue. In mesothelial tissue (pleura), we studied 9 pleural biopsy samples. Mean fibre burden was 9.33×104 f/gmdw. Chrysotile mean 0.73×104 f/gmdw and Amphibole 8.60×104 f/gmdw. In our study, endobronchial biopsy tissue samples were also studied. In 14 subjects mean number of asbestos fibers were found – 10.01×104 f//g, mean chrysotile level was 0.70×104 f/g dw and amphibole was 9.28×104 f/g dw.

Though, in our study, none of the differences were significant statistically ($p>0.05$) for all three variables maximum value was obtained for endobronchial biopsy and minimum for lymph nodes. As in our study mean fibre burden data was very less as compare to their study. It might be due to the difference in biopsy sample which was studied. The difference in our study in that their study was done on mesothelioma patients which is known to be associated with asbestos. In our study it was done in non mesothelioma cases.

In study done by M.L. Warwock, 45% of tumors were adenocarcinomas, 42% were squamous cell carcinomas, and 13% were undifferentiated.

In our study, adenocarcinoma comprises of 34.4%, squamous cell carcinoma 18.8%, small cell carcinoma 9.4% and patients who were non-neoplasmic were 37.5% so as in ML Wornock study. Adenocarcinoma is the most common type in our study also.

In ML Wornick study neither the type nor the location of lung cancer was dependent on degree of parenchymal fibrosis or on residual AC burden.

In our study, maximum asbestos burden was found in squamous cell carcinoma (mean = 10.28±3.38) and Amphibole levels are (9.47±3.68)×104. In non-neoplasmic patients, adenocarcinoma patients and in small cell carcinoma patients, mean asbestosfibers was (9.38, 9.18 and 7.4x)×104 respectively but if we see the association none of the differences were significant statistically ($p>0.05$).

If we see the association between malignancy status and fiber load, a total of 12 patients were non-neoplastic and 20 were included in malignant. However, total fiber burden on non-neoplasmic patients was high as compared to malignant and amphibole fibers were high in nonneoplasmic category and mean number of chrysotile fibers were slightly more in malignant patients. Inspite of this there was no significant difference seen between two groups observed for any of the variables. When we compare fiber loads of malignant and non-malignant patients, though mean fiber load is more in non-malignant patients mean (9.38±5.45) than in malignant patient and Amphibole are also more in non malignant

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 3501 - 3519
Received 25 April 2021; Accepted 08 May 2021.

patients. Chrysotile fibers more in malignant than in non-malignant patients.Though all these differences in malignant and non-malignant patients is not significant statistically. In contrast with other studies as fiber body counts estimated by light microscopy revealed a significantly larger magnitude in lung cancer cases and adenocarcinoma cases (especially in males) than non-lung cancer cases.

Iqbal Ahmad et al.[18] studied that cosmetic powder are having contamination with asbestos and hypothesized that cosmetic powers can be a source of exposure of asbestos in non-occupationally exposed population. A study was also done by L. Paolatti et al.[19] In their study they took 11 samples of branded cosmetic and pharmaceutical powder. Surprisingly, all the samples were found contaminated with different levels of asbestos averaging 12.5%. Their fiber lengths were also micrometrically measured in the range of <10 µm, 10-20 µm and >20 µm which were 21.06%, 43.85% and 35.06% respectively. In our study Talcum powder use was also taken into account. We put patient in <once per week for occasional users and >once per week for regular users. Out of 32 patients, 30 patients were occasional users and 1 regular patient and one patient was categorical non-user. As in our study most of the patients were in occasional category so association was not studied in this regard.

In our study if we see the estimation of fiber burden was done in pathological tissues and most of the patients were of non-occupational exposure. The thing which has to be emphasised is that the mean fiberload estimated in our patient was in the multiplication of 104 f/g dw which is significantly less than the estimated burden of other studies. Overall, both environmental and occupational asbestos exposure and the characteristics of asbestos fibers (length, aspect ratio) were risk factors of lung cancer [20]. Therefore, not only occupational exposure, but also environmental exposure to asbestos should be managed toreduce the risk of lung cancer.

The difference is that in our study the burden was done of pathological tissue and in most other studies it was done on lung parenchymal tissue or pleural tissue. It has to be noted that environmental factors can lead to asbestos exposure. So much larger studies are needed to establish any association between environmental factors to be responsible for this asbestos fiber load in lung and pleural tissue of non-occupationally exposed patients of lung and pleural malignancies. Larger studies are also needed to search out the source of this asbestos burden in non-occupationally exposed population.

**Conclusion:**

As we know that asbestos is very well known to cause mesothelioma and other lung malignancies, its fibre burden was usually studied in lung paranchymal tissues and mesothelial tissues in different studies previously done. As this study was done to evaluate asbestos burden in lung and pleural malignancy. Most of the patients in this study were non occupationally exposed. Although no statistically significant data came out to make any inference still fibre burden which came out in different sites of biopsy raises a question that what is the source of it.

As we see there is no significant result came out as far as asbestos burden and lung malignancy are concerned. However, the burden of the asbestos fibre has to be reviewed in

larger studies so that any inference can be drawn for the source of this fibre in nonoccupationally exposed lung cancer patients.

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