

Farina (1963)



265

Lavoro Umano, volume 15, pages 276-281, 1963

ASBESTOSIS AND LUNG NEOPLASMS
FURTHER CONTRIBUTION TO A CLINICAL CASE

Farina, S. and G. Mazzanti

Institute of Special Medical Pathology and Clinical Methodology,
University of Bologna (Director: Prof. D. Campanacci)
Department of Occupational Medicine (Director: Prof. G. D. D'Antuono)

FA-115

ASBESTOS INFORMATION ASSOCIATION/
Public Service
1835 K Street, N.W. Suite 402
Washington, D. C. 20005

For some time now, researchers have tried to evaluate the effect of a series of agents found in the air of the working environment on the generation of heteroplastic growths in the respiratory tract, with particular emphasis on the lung.

As a matter of fact, recent statistics indicate a significant increase of neoplasias of this nature.

Lately, improved information regarding the toxic inhalants present in the working environment has brought into focus the possible effect of a great number of agents that could contribute to, or actually induce, such conditions.

It is quite obvious that the attention of researchers has been directed primarily to agents whose physical and chemical properties are responsible for their marked irritative action at the level of the various mucosae. Accordingly, microtraumatic phenomena occurring at this level have been studied in regard to their involvement in the development of metaplastic conditions affecting various organs and tissues.

One such agent, which is well capable of acting via a chronic irritative effect at the level of the respiratory tract mucosa by producing irreversible sclerogenous lesions, is asbestos.

Examination of the pertinent literature fails to yield much information on the relationship between asbestosis and lung neoplasias, although there is much discussion regarding their occurrence which, according to some is a mere coincidence, while others claim lung fibrosis originating from an occupational condition might play a role.

The first observation of such an association has been reported by Ly and Smith, in 1935. Subsequently, many other investigators have reported

simultaneous presence of lung neoplasia and asbestosis-induced lung fibrosis in both live and autopsy material (Egbert; Gaiger; Wedler; Desmeules; Roux; Giroux and Sirois; Lynch and Connan; Rombola; Chauvet).

Anatomicopathological statistics compiled by Gloyne, Wedler, and Doll revealed that the incidence of this particular association varied from 10-15% a finding which would seem to definitely confirm the effect of asbestos fiber inhalation on the induction of lung neoplasia.

Furthermore, experimental studies performed by Nordmann and Sorge have shown the presence of lung carcinomas in 20% of the mice exposed to the inhalation of asbestos dust. This finding also supports the claims regarding the carcinogenicity of asbestos.

On the contrary, Vigliani (1940) failed to find lung cancer in any of 76 patients with asbestosis that he studied. Furthermore, systematic investigations carried out by Braun and Truan (1958) in a large number of asbestos workers, in Canada, showed that the incidence of lung neoplasia in this particular group was no higher than that recorded for individuals not exposed to asbestos dusts.

A consistent finding in studies demonstrating an association between lung neoplasia and the occupational condition mentioned has been a history of exposure to asbestos risk by the affected individuals.

The presence of proliferative processes primarily in the lower lung which is also the site of the most advanced degree of lung fibrosis, is in sharp contrast to the normal site of neoplasia, which is encountered more frequently in the central and upper lung lobes. According to the statistics mentioned above, it emerges that the incidence of the disease among female

is high, as opposed to the well-established finding that lung neoplasia affects almost exclusively the male sex.

All the findings could be indicative of more than a simple causal relationship between asbestosis and lung neoplasia.

The patient described in the present report represents a relatively interesting contribution to other similar cases described earlier, although not representing any decisive etiopathogenetic feature.

CLINICAL REPORT

The patient, a 54 year old worker, was employed as an insulator since 1926.

He was in good health until the month of September 1960 when he started having persistent cough and exertional dyspnea. Radiological examination performed at that time revealed darkening of the lower lung field. Wide streaks emerging from the enlarged hili and advancing towards the base of the lungs which, in turn, showed sharp reticulation.

Based on these findings, a radiological diagnosis of asbestosis was established and the case was forwarded to the insurance authorities for disability.

By June 1962, the patient presented gradual exacerbation of the dyspneic symptoms, together with a cough with little expectoration; furthermore, the patient complained of an oppressive pain in the right hemithorax.

Objective examination demonstrated a definite basal hypophonesis, rough and diffuse breathing over the entire pulmonary field where there was also evidence of a noise known as "fresh snow pounding" (D'Antuono et al.). There was no evidence of any other significant objective alterations.

Electrocardiographic recording was negative.

Urine examination was also negative. Examination of the sputum demonstrated the presence of bacterial flora consisting of cocci, gram-positive and gram-negative diplococci; the search for Koch's bacillus was also negative.

Erythrocyte sedimentation rate was high (I.K. = 37). Respiratory function tests revealed the presence of a constrictive type of ventilatory insufficiency of average severity.



Fig. 1

Hematological examination: red blood cells: $4,800,000/\text{mm}^3$; white blood cells: $8,000/\text{mm}^3$; Hb: 72%; hematocrit: 0.95; N: 67%; E: 2%; M: 6%; L: 25%.

R. W. and Mainicke: negative.

Standard chest x-ray, in addition to the previous findings recorded at the level of the lower pulmonary fields, also demonstrated the presence of parenchymal darkening at the level of the right cardiophrenic angle, roundish, with well-defined edges and homogenous density (Fig. 1). Repeat radiographic examination performed 20 days later confirmed the initial picture, without any appreciable changes.

On account of the inability to define the nature of this opacity, and

due to the negative results yielded by the laboratory tests (including cytological examinations and histological study of the expectoration), except for the increased sedimentation rate, the patient was placed on antituberculous treatment (chloramines) in order to determine the eventual changes in the lung findings, about 1 month later.

Radiographic examination performed at this time (Fig. 2) confirmed the presence of a mass located to the right of the heart, well evident stratigraphically in the anterior planes (Fig. 3), whose size was actually greater than that recorded during the previous radiographies.



Fig. 2

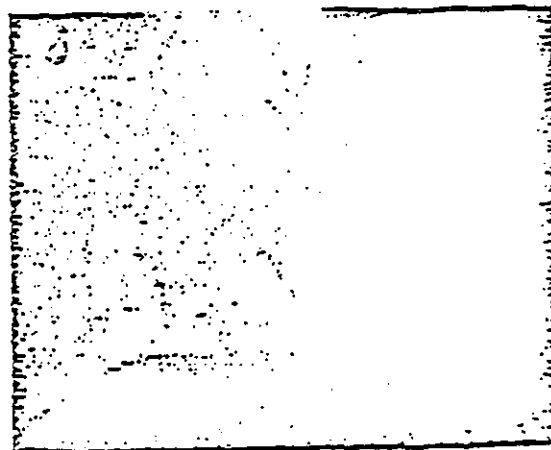


Fig. 3

Soon after the last examination, the patient presented blood in the expectorate, while pain and dyspnea increased; the general condition of the patient deteriorated gradually. He had persistent fever, and died in cachexia early in December 1962.

As a result of the rapid decline of his general condition, and especially based on the radiographic and stratigraphic picture which, as described above, demonstrated the presence of a definitely progressing parenchymal opacity, diagnosis of asbestosis associated with lung neoplasia was established.

The features described above lead to a series of interesting observations.

Thus, first of all, it appears that the association mentioned is relatively rare. As a matter of fact, we encountered only a single case of neoplasia in a total of more than 30 individuals exposed to asbestos.

The group observed by us is rather small and does not allow any definite conclusions, but it confirms the discrepancy of the data reported on this point in the German and English literature (which support the hypothesis of a relatively high incidence of neoplasia), on the one hand, and the findings of Italian investigators (Vigliani, Rombola) who found that the rates of neoplasia are much less significant, on the other.

It appears that the discrepancies and the subsequent differences in the evaluation of the oncogenic activity of asbestos fibers could be attributed to intrinsic geographical differences in the composition of asbestos itself, a fact reflected in the statistics obtained in various countries, rather than to errors performed in the diagnostic investigation of the workers at risk.

Actually, as is well-known, the variety of asbestos used in Italy is most exclusively white asbestos, a material with a low iron content as compared

to the blue asbestos used by the workers studied in the German and English statistics.

Nevertheless, the differences in the chemical composition of asbestos could be relevant if the role played by the predisposition of the individual at risk, and the severity of the risk itself, are well-known.

The role of asbestos per se, a material whose activity is explained by a great number of fine needles present in the asbestos fiber, should be an important factor only for the induction of trauma at the level of bronchial mucosa sequent to long-term exposure.

In conclusion, it seems that asbestos alone has only a predisposing role similar to that exerted by other agents having similar mechanisms, at different levels.

SUMMARY

The case history of a patient where asbestosis was associated with emphysema of the lung is described briefly. The discrepancies recorded in the literature on this particular subject are pointed out, and the statistical, experimental, and possible oncogenic properties of asbestos are discussed. Although not regarded as definitely carcinogenic, the chemical composition of the agent is considered to be a significant factor.

BIBLIOGRAPHY

1. Braun D. C.; Truan, T. D.: An epidemiological study of lung cancer in asbestos miners. Arch. Industr. Health 17: 634, 1958.
2. Cartier, P.: cited by Rombola.
3. Chauver, M.: Asbestosis and bronchial cancer. Presse Med. 66: 908,
4. D'Antuono, G.; Mazzanti, G.; Farina, S.: Clinical and radiographic ob-

- variations on lung asbestosis. Giorn. Pneumol. 5: 1, 1961.
5. Doll, R.: Mortality from lung cancer in asbestos workers. Brit. J. Indust. Med. 12: 81, 1955.
 6. Garnez-Rieux, C.; Marchand, M.; Mounier-Kuhn, P.; Policard, A.; Roche, Occupational respiratory diseases. Masson, Paris, 1961.
 7. Isselbacher, K. J.; Klaus, H.; Hardy, H. L.: Asbestosis and bronchogenic carcinoma. Report of one autopsied case and review of the available literature. Amer. J. Med. 15: 721, 1953.
 8. Linquette, M.; Voisin, C.: Silicosis and other pneumoconioses. Flammarion, Paris, 1960.
 9. Lynch, K.; Connan, M.: Asbestosis analysis of 40 necropsied cases. I Chest 14: 874, 1960.
 10. Lynch, K.; Smith, W. A.: Pulmonary asbestosis. Carcinoma of lung in asbesto-silicosis. Amer. J. Cancer 24: 56, 1935.
 11. Nordmann, M.; Sorge, A.: cited by Rombola.
 12. Rombola, G.: Asbestosis and lung cancer in an asbestos textile plant. Med. Lavoro 46: 242, 1955.
 13. Wedler, H. W.: Asbestosis and lung cancer. Dtsch. Med. Wschr. 69: 571, 1943.