Pak. J. Chest Med. 2023;29(01)

Short Report

journal homepage: https:/www.pjcm.net/

Pakistan Journal of Chest Medicine

Official journal of Pakistan Chest Society

Asbestos, Mesothelioma and Lung Cancer: An opinion

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Article History:

Received:	Jan 08, 2022
Accepted:	Feb 20, 2022
Available Online:	Mar 02, 2022

How to cite this Short Report:
Jargin SV. Asbestos, mesothelioma and lung cancer: An opinion. Pak J Chest Med 2022; 28(01):125-130.

A B S T R A C T

Health risks from asbestos exposures have been evaluated taking account of data from the past, when exposures at workplaces were higher than today. The linear no-threshold model has been applied although its relevance is unproven. Presumably, releases from natural emission sources dwarf man-made contributions to the atmospheric dispersion of asbestos. Fibers are frequently found in the lungs and pleura of deceased people from the general population. Finding of fibers does not prove that a disease is caused by asbestos. It can be reasonably assumed that a targeted search for mesothelioma and other asbestos-related conditions in exposed populations resulted in an increased detection rate. Histological and immunohistochemical characteristics of malignant mesothelioma partly overlap with other cancers, which may contribute to overdiagnosis in exposed populations. The etiology and differential diagnosis of malignant pleural mesothelioma as well as differences in carcinogenicity between chrysotile and amphibole asbestos are briefly discussed here. There are motives to strangulate the use of amphiboles fuelled by interests of chrysotile producers. In the author's opinion, current regulations applied in some countries are exceedingly restrictive and should be revised on the basis of independent research. The promising way to reliable information would be largescale animal experiments. It can be reasonably assumed that non-application of asbestos-containing brakes, fireproofing, insulation lagging etc. would augment the damage and numbers of civil victims of conflicts, fires and traffic accidents.

Key words: Asbestos; Fiber; Pleura; Dust Diseases; Cancer

Introduction

Health risks from asbestos exposures have been evaluated taking account of data from the past, when exposures at workplaces were much higher than today. The linear no-threshold model, known from the radiation protection, has been applied to asbestosrelated risks although its relevance is unproven and remains arguable both for pleural and lung tumors.[1] Of note, natural fiber emission sources contribute to a global dispersion of chrysotile and amphibole asbestos fibers. Presumably, the natural release dwarfs manmade contributions to the atmospheric dispersion of both fiber types.[2,3] Asbestos fibers have been found at more than 60% routine autopsies including children.[4,5] Finding of fibers does not prove that a disease is caused by asbestos. Inhalation and clearance of fibers occurs permanently being in a dynamic balance. By analogy with[6] other substances in the natural environment,[7-9] it can be assumed that there is a harmless (threshold) fiber concentration in the ambient air. An evolutionary adaptation to a threshold concentration is theoretically possible.[10]

Asbestosandmesothelioma

The targeted search for mesothelioma and other asbestos-related conditions in exposed populations must have resulted in an increased detection rate. Among potential causes of mesothelioma are non-asbestos fibers (e.g. erionite), virus SV40, chronic inflammation (empyema, tuberculosis), ionizing radiation and genetic predisposition.[10-12] The comparison of subjects exposed to asbestos and erionite shows that they have similar characteristics.[13] A majority of malignant pleural mesothelioma (MPM) specimens contained DNA sequences of the SV40 virus.[14] There are indications that SV40 has contributed to the worldwide incidence increase of mesothelioma in recent decades despite asbestos bans.[14] Furthermore, bronchoscopy in people at risk must have contributed to the spread of SV40. It is known that hepatitis virus can be transferred by endoscopy.[15] In Russia, bronchoscopy was performed in patients with asbestos-related bronchitis and those suspected for dust diseases.[16-18]

MPM is not clearly demarcated from other cancers; it had no diagnostic category within the ICD system until the 10th edition.[19] Histologically, many MPMs have a structural similarity to other malignancies. The absence of pathognomonic markers can make the diagnosis complicated, especially that of sarcomatoid MPM.[10,20] According to an estimate, about 10% of MPMs in the United States were misdiagnosed.[21] In one study, the initial MPM diagnosis was validated in 67%, revised in 13% and remained uncertain in 20% of the cases.[22] In asbestos-exposed cohorts, trained pathologists perform targeted search for MPM. Therefore, more MPMs have been detected probably with some overdiagnosis in questionable cases. The shortage of specific biomarkers renders the diagnosis of MPM difficult. Mesothelin was regarded promising, lacking, however, sufficient sensitivity to be used as a standalone marker.[10,23,24] Mesothelin may be over-expressed in various cancers including lung adenocarcinoma.[25] Mesothelin is typically negative in sarcomatoid MPM and in almost a half of cases of epithelioid mesothelioma, especially at early stages.[26,27] Notwithstanding the plethora of old and new markers, none has been sufficiently specific.[10,24,28] According to the 2014 update of the Helsinki Criteria, no specific recommendations can be given for the use of markers in the screening for MPM."[28,29] A general tendency to overestimate validity of immuno-histochemical and molecular markers has been noticed.[30] Furthermore, MPMs may exhibit various molecular setups in different areas of the same tumor, subclones and intra-tumoral heterogeneity.[10,31] Contrary to other cancers, driver mutations have not been clearly determined in MPM.[32] The sensitivity of fluid cytology for MPM remains generally low.[23] The above explains an imprecise delineation of MPM from other cancers, which may enhance the diagnostic yield in exposed people, encompassing borderline and questionable cases.

Chrysotilevs.amphiboles

It is widely believed that serpentine (chrysotile) is less toxic than amphibole (actinolite, amosite, anthophyllite, crocidolite, tremolite) asbestos.[20] However, there are inconsistencies in the literature, in particular, between animal and human data. In some experiments, amphiboles and chrysotile were demonstrated to possess approximately the same level of carcinogenicity both for mesothelioma and carcinoma.[10,33,34] The risk difference between carcinogenic effects of chrysotile vs. amosite and crocidolite for lung cancer in humans was estimated in the range 1:10 to 1:50.[1] The risk ratio of mesothelioma from asbestos of the above-named types was estimated, respectively, as follows: 1:100:500, cited[1] in the review.[20] In a subsequent publication another ratio has been suggested: 1:5:10.[35] It is difficult to explain discrepancies between results obtained in human and animal studies; in particular, there are no reasons to suppose substantial differences in the fiber clearance mechanisms. Chrysotile is predominantly detected in pleura, thus making suppositions about its rapid disappearance from the lung as attest for lower toxicity questionable.[36] The early disappearance of chrysotile fibers may be attributed to a longitudinal or traversal splitting with formation of thin fibrils that can escape detection.[37] As a result of the splitting, the total number of fibrils increases,[37-42] possibly together with carcino-

genicity. Presumably, the thinner a fiber, the higher would be its capacity to cause cancer, as it can better penetrate tissues.[43] Chrysotile was the predominant asbestos fiber in pleura, particularly, in pleural plaques.[44-47] Note that mesothelioma is initially more frequent in the parietal rather than visceral pleura i.e. at a distance from the lung.[48] The pathogenesis of MPM is related to the inflammatory microenvironment created by the fibers in pleura.[32] Chrysotile was reported to induce DNA damage and precancerous changes in cultured cells.[49,50] The relatively high incidence of mesothelioma among workers having contact with amphiboles was supposed to be partly caused by averagely higher exposures.[51] The incidence of mesothelioma is known to be elevated after exposures to pure chrysotile;[36,52] more details are in the preceding report.[10]

There is an opinion that "three D's" - dimensions, durability (biopersistence) and dose - determine the carcinogenicity of asbestos and some other fibers.10,53 The biopersistence being equal, differences in carcinogenicity are associated with the length and thickness of the fibers.[54,55] Long fibers of chrysotile were found to possess a relatively high carcinogenicity as they cannot be efficiently engulfed and cleared by macrophages.[53,56] Interestingly, thin and short chrysotile fibers were found to be the prevailing fiber type detected in the lung and pleura of mesothelioma patients.[57] It was suggested that inhalation of fibers of that kind is associated with a higher risk of MPM.[58] In addition, tremolite in chrysotile products can potentiate carcinogenicity.[59] A review concluded that there is no compelling proof that the increased incidence of MPM in chrysotile workers was caused solely by tremolite.[44] It is possible that tremolite and other amphiboles are more harmful than chrysotile; but further unbiased studies are needed to clarify this. The reportedly higher carcinogenicity of crocidolite from South Africa compared to that from Bolivia might be explained by the fiber width.60 Besides, bias due to the interests of chrysotile producers could have played a role.[10] Works by J. C. Wagner and D.

Bernstein have been discussed previously in this connection.[10] Numerous publications unsupportive of the claims by Bernstein et al. about much higher toxicity of amphiboles were not cited in their reviews.[54,61] Of note, after accounting for the quality of exposure estimation, there were less differences between chrysotile and amphiboles.[62] A similar tendency was noticed in a systematic review, where pooled risk estimates for lung cancer were higher after neighborhood exposures to amphiboles - 1.74 (95% CI 1.18 to 2.57) than to chrysotile - 0.99 (95% CI 0.78 to 1.25); whereas the overall risk tended to be higher in intermediate- rather than in high-quality studies (no poor-quality group was specified): 1.86 (95% CI 1.27 to 2.72) vs. 1.21 (95% CI 0.79 to 1.87).[63] Significant differences between results obtained in high- vs. low-quality studies must be generally indicative of bias due to conflicts of interest,[64] as it is obviously easier to find support for preconceived ideas in the domain of poor-quality and manipulated research rather than in high-quality studies.

Russianscienceonasbestos-relatedhealthrisks

Asbestos-related diseases have been extensively studied in the USSR (reviewed previously).[10] The research activities have somewhat decreased since the last decades together with the number of publications. No risk elevation has been found among inhabitants of the areas adjacent to modern asbestos-processing plants. The prevailing opinion is that, if preventive measures have been taken, modern technologies are acceptably safe while restrictions and bans applied in some parts of the world are superfluous. Fiber emissions from asbestoscontaining roofing materials are deemed negligible. Their concentrations in the indoor air are far below permissible levels. The toxicity of asbestos cement and cardboard is low due to the connection of fibers with cellulose, cement and other substances. Car brakes cause no significant air pollution, while the traffic is safer with asbestos brake pads.[10,65] The low toxicity of chrysotile compared to amphiboles is often stressed in the Russian literature. The asbestos produced in Russia is almost exclusively chrysotile. For an inside observer, it is obvious that behind numerous publications claiming the relative harmlessness of chrysotile has been an unofficial directive and/or spontaneous loyalty of researchers, which doesn't contribute to scientific objectivity. At the same time, some data do not agree with this concept;[66,67] more studies have been reviewed previously.[10]

Discussionandconclusion

The number of publications about asbestos toxicity is growing; it is increasingly difficult to distinguish between objective and biased information. The asbestos research has been influenced by conflicts of interest. The same is true, for example, for the anti-nuclear activism.[9,68] By a given biopersistence of fibers, differences in toxicity can be associated mainly with the length and thickness of fibers, and not so much with the asbestos type.[54,55] Different asbestos types are intermixed in the international trade.[69] The carcinogenicity of asbestos substitutes e.g. carbon nanotubes comes to light these days. Admittedly, carbon nanotubes are diverse: long, stiff, multiwall ones have been classified as possible human carcinogens but some varieties are not yet classifiable.[70] The above-mentioned Helsinki 2014 Criteria stipulate that "even a brief or low-level exposure should be considered sufficient for mesothelioma to be

designated as occupationally related,"[29] which may result in classification of spontaneous cases as occupationally related. As for lung cancer, the Criteria leave space for subjectivity: "Cumulative exposure, on a probability basis should thus be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk."[29] In the author's opinion, current regulations applied in some countries are exceedingly restrictive and should be revised on the basis of independent research to become more realistic. The most promising way to reliable information would be largescale animal experiments. As discussed previously, integrity of all participants is needed for that.[71] There are motives to strangulate the use of amphiboles fuelled by interests of chrysotile producers. Different asbestos types have their technical advantages and, correspondingly, preferred application areas, which is beyond the scope of this report. It can be reasonably assumed that the nonapplication of asbestos-containing brakes, fireproofing, insulation lagging etc. augmented the damage and numbers of civil victims of international conflicts, fires and traffic accidents.

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