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Review Paper

Asbestos-related Lung Diseases: A Brief Update

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ABSTRACT

Health risks from asbestos exposures have been evaluated, considering past professional histories when exposures at workplaces were higher than today. A linear no-threshold (LNT) model has been applied, although its relevance is unproven. Fibers are often found in the lungs and pleura of deceased people. Fiber findings do not prove that a disease is caused by asbestos. It is reasonable to assume that a targeted search for mesothelioma and other asbestos-related conditions in asbestos workers resulted in an increased detection rate. Histological and immunohistochemical characteristics of malignant mesothelioma partly overlap with other cancers, which may contribute to the overdiagnosis in exposed populations. The etiology and differential diagnosis of malignant pleural mesothelioma as well as differences in carcinogenicity between different asbestos types are briefly discussed here. In the author's opinion, current regulations applied in some countries are excessive and should be reconsidered based on independent research. The most promising way to obtain reliable information would be through lifelong bioassays. It can be reasonabl
increases the harm caused by fires, traffic accidents, and armed conflicts.

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1. Introduction

Health risks from asbestos were evaluated based on past professional histories when exposures at workplaces were much higher than today [1, 2]. The linear no-threshold (LNT) model, known from radiation protection, has been applied to asbestos-related risks, although its relevance is unproven and arguable both for pleural and lung tumors [3]. Of note, the natural fiber emission contributes to a global dispersion of chrysotile and amphibole asbestos fibers. Presumably, natural releases dwarf anthropogenic contributions to the atmospheric dispersion of both fiber types [4, 5]. Air, soils, and waters may be contaminated by asbestos and other potentially harmful fibers due to human activities unrelated to asbestos industries, e.g. land excavation, slope reprofiling, and tunneling [6, 7]. Asbestos fibers have been found in more than 60% of routine autopsies, including children [8, 9]. The fiber findings do not prove that a disease is caused by asbestos. Inhalation and clearance of fibers occur permanently [10], in a dynamic balance. By analogy with other substances in the natural environment, it can be assumed that there may be a harmless (threshold) fiber concentration in the ambient air. The notion of "one fiber can kill" seems irrelevant because it is related to the natural substances that would be noxious at higher doses [11].

Asbestos and mesothelioma

The targeted search for malignant pleural mesothelioma (MPM) in asbestos workers must have led to an increased detection rate. Non-asbestos fibers (e.g. erionite), the SV40 virus, chronic inflammation (empyema, tuberculosis), ionizing radiation, and genetic predisposition are all potential causes of mesothelioma [12, 13]. Erionite is a stronger carcinogen than asbestos. Human activities can result in the dispersal of erionite and other potentially carcinogenic fibers in densely populated areas [7]. The comparison of subjects exposed to asbestos and erionite shows that they have similar characteristics, e.g. fiber concentrations in the bronchoalveolar lavage fluid [13]. Furthermore, a majority of MPM specimens contained DNA sequences from the SV40 virus [14]. There are indications that SV40 led to the worldwide increase of mesothelioma in recent decades despite asbestos bans [15]. Among others, bronchoscopy in high-risk people has probably contributed to the spread of SV40. It is known that viruses can be transferred by endoscopy [16]. In Russia, bronchoscopy was performed in patients with asbestos-related bronchitis and those with suspected dust diseases [17-19].

The MPM is not demarcated from other cancers; it had no categorization within the International Classification of diseases (ICD) till the tenth edition [20]. Histologically, many MPMs have structural similarities with other malignancies. The absence of pathognomonic markers can make the diagnosis difficult, especially that of sarcomatoid MPM [21]. According to an estimate, about 10% of MPMs in the United States were misdiagnosed [22]. In one study, the initial MPM diagnosis was confirmed in 67%, revised in 13%, and remained uncertain in 20% of cases [23]. In asbestos-exposed cohorts, pathologists with-relevant experience undertake an appropriate search for MPM. Accordingly, more MPMs are detected, while some overdiagnosis in questionable cases is unavoidable. However, mesothelin was regarded as promising, however, insufficient sensitivity to be used as a standalone marker [2, 24, 25]. Mesothelin may be prominent in various cancers [26]. It is typically negative in sarcomatoid MPM and ~50% of the cases of epithelioid mesotheliomas [27, 28]. Notwithstanding the plethora of old and new markers, none has been sufficiently specific [25, 29]. Available information about the molecular basis of MPM is regarded as poor [30]. According to the 2014 update of the Helsinki Criteria, no specific recommendations can be given for the use of markers in the MPM screening [29, 31]. A general tendency to overestimate the validity of immunohistochemical and molecular markers has been observed [32]. Furthermore, MPMs may show various molecular setups in different areas of the same tumor i.e. subclones and intra-tumoral heterogeneity [33]. Contrary to other cancers, driver mutations have not been determined in MPM [34]. The sensitivity of fluid cytology for MPM remains low [24]. The above explains an imprecise delineation of MPM from other cancers, which may enhance the diagnostic yield of screening in exposed populations.

Chrysotile vs. amphiboles

It is widely believed that serpentine (chrysotile) is less toxic than amphibole (actinolite, amosite, anthophyllite, crocidolite, tremolite) asbestos [21]. However, inconsistencies exist 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 lung cancer [35, 36]. On the contrary, the lung cancer risk difference between chrysotile vs. amosite and crocidolite in humans was estimated to range from 1:10 to 1:50 [3]. The risk ratio of mesothelioma from asbestos of the above types was estimated, respectively, as follows: 1:100:500 [3], cited in the review [21]. In a subsequent publication, quite another ra-

tio has been suggested: 1:5:10 [37]. The same researchers [3] pointed out comparable quantities of pulmonary neoplasms produced in bioassays with various asbestos types [38]. Of note, no reasons exist to believe substantial interspecies differences in the fiber clearance mechanisms. Chrysotile clearance from pulmonary tissues may occur through fiber splitting and relocation to the pleura [39]. One of the causes of the non-detection of chrysotile fibers, interpreted as early disappearance from the lung, is the longitudinal splitting with the formation of thin fibrils that can escape detection [37]. As a result, the total number of fibrils increases [40-45], possibly with carcinogenicity. Presumably, the thinner the fiber, the greater its capacity to cause cancer because it can penetrate tissues more efficiently [46]. Chrysotile was the predominant asbestos fiber found in the pleura, particularly in pleural plaques [38, 47-49]. Note that mesothelioma is initially more common in the parietal rather than the visceral pleura, i.e. at a distance from the lung [50]. The pathogenesis of MPM is related to the inflammatory microenvironment created by the fibers in the pleura [34]. Studies have shown that chrysotile causes DNA damage and precancerous changes in cultured cells [51, 52]. The relatively high incidence of mesothelioma among workers exposed to amphiboles is partly caused by higher average exposures [53]. The incidence of mesothelioma increases after exposure to pure chrysotile [2, 39, 54].

The carcinogenicity of asbestos and some other fibers is largely determined by the "3 D" - dimensions, durability (biopersistence), and dose [55]. When biopersistence is equal, differences in carcinogenicity are associated with fiber length and thickness [56, 57]. Long fibers of chrysotile have relatively high toxicity [58] because they cannot be efficiently engulfed and cleared by macrophages [55, 59]. According to another report, thin and short chrysotile fibers are the prevailing fiber types detected in the lung and pleura of patients with MPM [60]. It has been suggested that the inhalation of fibers of that kind is associated with a higher risk of MPM [61]. In addition, tremolite in chrysotile products can potentiate carcinogenicity [61-63]. A review concluded that no compelling evidence existed that the increased incidence of MPM in chrysotile workers was caused solely by tremolite [38]. Admittedly, tremolite and other amphiboles are more harmful than chrysotile, but further unbiased studies are needed to clarify this. The higher carcinogenicity of crocidolite from South Africa compared to Bolivia may be explained by the fiber width [63]. Besides, bias due to the interests of chrysotile producers could have played a role. The work by David Bernstein has already been discussed in this regard [64]. Numerous publications unsupportive of the claims by Bernstein et al. about the toxicity of most amphiboles were not cited in their review [56, 65]. Of note, after adjusting for the quality of exposure estimation (i.e. doses), fewer differences between chrysotile and amphiboles were observed [66]. A similar tendency was observed in a systematic review, where pooled risk estimates for lung cancer after exposures to amphiboles were higher in the neighborhood-1.74 (95% CI 1.18 to 2.57) than chrysotile-0.99 (95% CI 0.78 to 1.25), while the overall risk was higher in intermediate-quality rather than in high-quality studies (no poor-quality group was observed): 1.86 vs. 1.21 ($P<0.05$) [67]. Significant differences between results obtained in high- quality studies compared to low-quality studies indicate bias due to conflicts of interest [68], as it is easier to find support for preconceived ideas in poor-quality and manipulated studies than in high-quality research. The questionable conclusions by Bernstein et al. in favor of chrysotile vs. amphiboles, as well as support by the Québec Chrysotile Institute, have been pointed out [69, 70].

Russian science on asbestos-related health risks

Asbestos was studied by medical scientists in the Union of Soviet Socialist Republics (USSR), and reviewed previously [2]. The number of publications and research activities has both decreased in recent decades. No risk has been found among residents of the areas adjacent to modern asbestos-processing plants. The prevailing opinion is that, if adequate preventive measures have been taken, modern technologies are sufficiently safe. Fiber emissions from asbestos-containing roofing materials are deemed negligible. Fiber concentrations in the indoor air are much lower than the permissible levels. The toxicity of asbestos cement and cardboard is low due to the aggregation of fibers with cellulose, cement, and other substances. Car brakes cause no significant air pollution, while traffic is safer with asbestos brake pads [2, 71]. Asbestos produced in Russia is almost exclusively chrysotile. The low toxicity of chrysotile compared to amphiboles is often stressed in Russian literature. However, some data contradict this concept [72, 73]; more studies have previously been reviewed [2].

2. Discussion and Conclusion

The number of publications about asbestos toxicity is growing; it is becoming increasingly difficult to distinguish between objective and biased information. Asbestos research has been influenced by conflicts of interest. For example, the same is true for anti-nuclear propaganda [74]. Asbestos use and production are prohibited in some countries [75]. Different fibers are intermixed in

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international trade [76]. The carcinogenicity of asbestos substitutes is under study today; it is largely dependent on the diameter and lengths of the fibers [77]. Long, stiff, multiwall carbon nanotubes have been classified as possible human carcinogens [78]. The above-mentioned 2014 Helsinki Criteria stipulate that "even a brief or low-level exposure should be considered sufficient to define mesothelioma as occupationally related" [31]. This approach leads to the spontaneous classification of cases as occupationally related. As for lung cancer, the criteria leave space for subjectivity, "therefore, cumulative exposure, based on probability, should be considered as the main criterion for attributing a substantial contribution by asbestos to lung cancer risk" [31].

The most promising way to obtain reliable information would be through lifelong bioassays [79]. There are motives to strangle the use of amphiboles fuelled by industrial interests. Different asbestos types have their technical advantages and, correspondingly, preferred application areas, that are beyond the scope of this review. The brake pads' durability is greatly influenced by the types of reinforcing materials used [80]. Failure to use asbestos-containing brakes, fireproofing, insulation lagging, etc. is probably to augment the harm from armed conflicts, conflagrations, and traffic accidents. In conclusion, there are still discrepancies between what is written in the literature and what happens in the real world. Consequently, some decision-makers do not have a clear perspective [81]. This review is another humble attempt to elucidate the matter.

Ethical Considerations

Compliance with ethical guidelines

Not applicable.

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Authors' contributions

Conceptualization and writing: Sergei Jargin.

Conflict of interest

The author declared no conflict of interest.

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