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RESPIRATORY CASE REPORTS

Distinguishing Occupational and Environmental Asbestos Exposures: Case Series

Mesleki ve Çevresel Asbest Maruziyetlerinin Ayrımı: Olgu Serisi

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Abstract

Asbestos is a flexible and fibrous silicate that is highly resistant to physical and chemical conditions. Because of these features, it has been utilized for many years in a variety of industries, including insulation, textiles, ships, vehicle production, and construction. Despite the fact that its usage is restricted in Turkey, as in many other countries, significant occupational exposures can occur during the repair of outdated industrial items and the demolition of buildings. Furthermore, environmental asbestos exposure is a major public health concern in several areas of Turkey. Therefore, it does not come to mind to question occupational exposures in the diagnosis of asbestos-related lung disease. The purpose of this article is to highlight the importance and necessity of questioning both occupational and environmental asbestos exposure, as well as some differences in the pathologies that environmental and occupational asbestos exposures may cause in the lungs of patients with asbestos-related disease.

Keywords: *Asbestosis, pleural plaque, occupational asbestos exposure, environmental asbestos exposure.*

Öz

Asbest ısıya, aşınmaya, kimyasal maddelere oldukça dayanıklı, esnek ve fibröz yapıda bir silikattır. Bu özellikleri sebebiyle izolasyon, tekstil, gemi, otomobil üretimi ve inşaat sanayi gibi birçok iş kolunda uzun yıllar boyunca kullanılmıştır. Kullanımı birçok ülkede olduğu gibi Türkiye'de de yasaklanmasına rağmen, eski sanayi ürünlerinin, inşaatların yıkımı ve tamiri sırasında yoğun mesleki maruziyetler olabilmektedir. Ayrıca, doğada diğer mineraller ile karışım halinde bulunan asbest, ülkemizin bazı bölgelerinde yoğun olarak bulunmaktadır. Türkiye'de çevresel asbest temasının yoğun olduğu bölgelerin çokluğu sebebi ile asbest ilişkili akciğer hastalıkları ile karşılaşıldığında çevresel maruziyet sorgulamasının ötesinde mesleki maruziyet sorgulamaları çok da akla gelmemektedir. Biz bu makalemizde olgularla asbest ilişkili hastalık saptananlarda, çevresel maruziyet kadar mesleksel maruziyetinde sorgulanmasının önemi ve gerekliliğini, çevresel ve mesleki asbest maruziyetlerinin akciğerde meydana getirebileceği patolojiler arasında olabilecek bazı farklılıkları belirtmeyi hedefledik.

Anahtar Sözcükler: *Asbestozis, plevral plak, mesleki asbest maruziyeti, çevresel asbest maruziyeti.*

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Asbestos is a fibrous silicate that is highly resistant to heat, water, and physical stress. It has been widely utilized in cement production, fireproof fabric, brake and clutch lining assembly, shipbuilding, insulation, electrical, plumbing works, power plants, and building construction due to its high insulating qualities and lasting structure (1). Furthermore, there is still significant occupational exposure during the repair and demolition of these asbestos-containing products (2).

Environmental exposure to asbestos, which occurs naturally as a combination with other minerals, is also a significant health issue in Turkey. Soil containing asbestos has long been utilized in rural settlements in Turkey's southeastern, central, and eastern Anatolian provinces as roofing, plaster, and whitewash material for heat and water insulation in buildings (2,3).

Asbestos-related diseases are primarily caused by inhalation. Asbestos fiber inhalation causes both benign (pleural plaques, diffuse pleural thickening, pleural effusion, asbestosis, round atelectasis) and malignant (mesothelioma, lung cancer) lung diseases (1,2). Asbestos use was prohibited in Turkey, as well as in many other nations, because it was determined to be a carcinogen (4). Despite this, new cases of asbestosis are reported because of prior exposures in workers in industries where asbestos is extensively utilized (2). In Turkey, where environmental asbestos exposure is high, it is common not to examine the occupational exposure history. The goal of this article is to raise awareness about the numerous aspects of occupational and environmental asbestos contact as well as the importance and necessity of questioning occupational exposure in patients with asbestos-related disorders and environmental exposure.

CASE

Six male patients who were exposed to asbestos and referred to our clinic for further evaluation were assessed in our case series. Table 1 shows the patients' demographics, smoking history, systemic disease history, environmental asbestos exposure, workplace history, time of asbestos exposure, and occupational and environmental asbestos exposure times.

Case 1: A 69-year-old male patient was admitted to our clinic with complaints of worsening dyspnea and dry cough during the previous two months. The patient's anamnesis revealed that he worked as a plumber, where he worked for 32 years, wrapping fabrics containing asbestos around the pipes for insulation purposes. He was born and raised in Ankara, and he had experienced coronary artery disease (CAD) in the past. Fine crackles were detected in the bilateral lower lobes of the lungs during the respiratory system evaluation. Other system and laboratory values were within normal limits.

The posteroanterior (PA) chest X-ray revealed a significant reticulonodular appearance as well as localized pleural thickening and calcifications (CXR). HRCT revealed more prominent pleural thickening and plaque formation in the mid-upper zone of the lungs, as well as reticulonodular density alterations, parenchymal fibrous bands, and intralobular septal thickening in the bilateral lower lung lobes (Figure 1). The patient underwent a diagnostic bronchoscopy. Light microscopy was used to examine the pathology of a transbronchial biopsy sample taken from the lower lobe of the right lung. On a proteinous background coated with lymphocytes and leukocytes, many alveolar macrophages with anthracosis pigment, and ferriginous bodies were detected (Figure 2). Based on his occupational anamnesis and radiological and pathological data, the patient was diagnosed with occupational asbestosis.

Case 2: A 66-year-old male patient was admitted to our clinic with complaints of dyspnea and increased exercise for the last 6 months. The patient's occupational anamnesis revealed that he worked for 11 years between 1977 and 1989 in the production of firefighting suits made of asbestos and glass wool. The patient was born and raised in Ankara. He had a history of coronary artery disease and chronic obstructive pulmonary disease (COPD). Fine crackles were found in the bases of both lungs during a respiratory system evaluation. He had clubbing on both of his hands. Other system examinations and laboratory parameters were within normal limits. The PA CXR of the right sinus was blunt, and there was right pleural irregularity and reticulonodular appearance in the bilateral lung bases. HRCT revealed nodular pleural thickening and minimal pleural fluid in the right lung, peripheral atelectasis adjacent to it, and reticulonodular densities and parenchymal density disparities in both lungs (Figure 3). To rule out a suspected malignancy, a wedge biopsy was performed from the right pleural thickening area. Pathology revealed chronic pleuritis, the formation of hyalinized fibrous pleural plaque, and ferriginous bodies (Figure 4). Based on occupational history as well as clinical, radiological, and pathological data, the patient was diagnosed with occupational asbestosis and asbestos-related benign pleural thickening and calcification.

Case 3: A 78-year-old male patient was admitted to our clinic complaining of dyspnea that worsened with exertion. The patient, who was born in Malatya and lived in Ankara, the patient's occupational anamnesis revealed a history of 1.5 years working as an electrical maintenance master of blast furnaces in a cement factory and subsequently 2 years working in the fabrication of asbestos water pipes. He then retired after working as a manager for a telecommunications company for 19 years. It was concluded that there had been extensive asbestos exposure for 3.5 years. He had a history of hypertension and coronary artery disease. Physical examination revealed fine end-

inspiratory crackles in the bilateral lung bases, as well as clubbing in his fingers. Further examinations found no signs of pathology. The laboratory parameters were all within the normal range. PA CXR revealed pleural thickening and calcifications in both hemithoraxes, as well as nonhomogeneous infiltrates in both lung bases. In each hemithorax, the HRCT revealed nodular pleural thickening and calcification. Interstitial septal thickenings, traction bronchiectasis, ground glass infiltrations with nodularity, and a honeycomb appearance were found in both lungs (Figure 5). Based on clinical, radiographic, and laboratory findings, the patient was diagnosed with occupational asbestosis and asbestos-related benign pleural thickening and calcification.

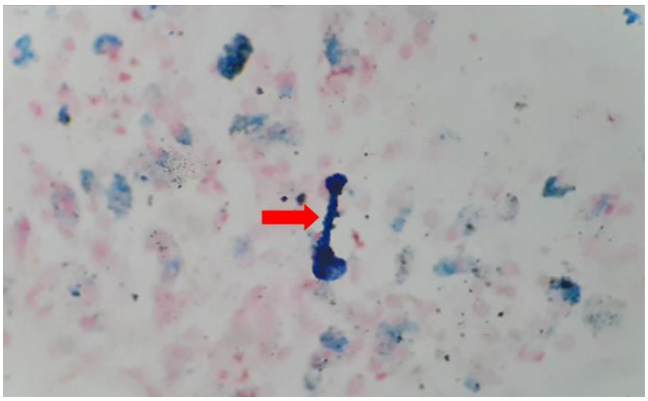

Figure 2: Light microscopic image of Case 1: Hemosiderin-containing macrophages and ferruginous body in pathological examination (shown with a red arrow) (Prussian blue X600)

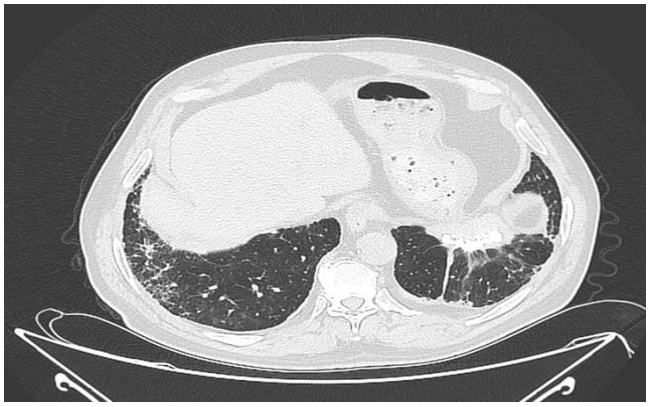

Figure 1: High-resolution computerized tomography parenchyma section of Case 1: Irregular reticular opacities, parenchymal fibrous bands, and intralobular septal thickenings in bilateral lower lung lobes

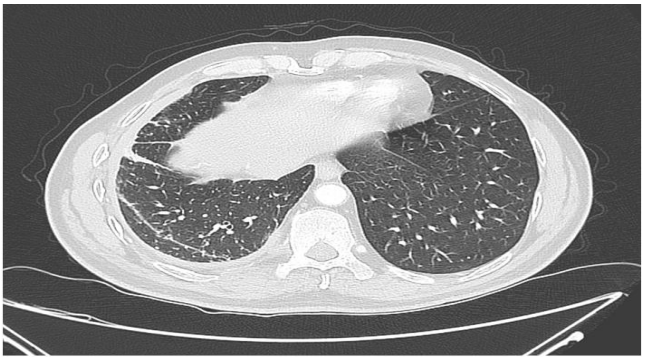

Figure 3: High-resolution computerized tomography parenchyma section of Case-2: Pleural thickening and minimal pleural fluid in the right lung, peripheral atelectasis adjacent to it, parenchymal fibrous bands, irregular reticulonodular densities, and parenchyma

Table 1: The demographic characteristics, smoking history, history of systemic disease, environmental asbestos exposure, workplace history, the time asbestos exposure began, and the duration of occupational and environmental asbestos exposure

Cases	Age (Year)	Smoking (pack/ years)	History of systemic disease	Environmental asbestos exposure	Work place history	The beginning time of asbestos exposure	The duration of occupational exposure (year)	The duration of environmental exposure (year)
Case-1	69	15	CAD	(-)	Plumbing	32 years ago	32	None
Case-2	66	40	CAD, COPD	(-)	Tailor in fire proof fabric production	39 years ago	11	None
Case-3	78	30	CAD	(-)	Waterpipe, Manufacturing, Electrician of furnaces in cement plant	35 years ago	3.5	None
Case-4	52	10	None	(+)	Cement mixing İn construction	52 years ago	None	15
Case-5	70	None	None	(+)	Lathe master	70 years ago	None	20
Case-6	46	26	None	(+)	Lathe master, Security guard, Worker in Magnesite production	46 years ago	None	10

CAD:coronary artery disease, COPD: chronic obstructive pulmonary disease, HT: hypertension

Case 4: A 52-year-old male patient was admitted to our clinic with an occasional cough and shortness of breath. The patient, who was born in Diyarbakir's Ergani district, had spent 15 years living in a house coated with asbestos soil. In his occupational anamnesis, he had mixed sand and mortar during bridge construction between 1993 and 1995. He had been laying granite stones on roadsides in the landscaping profession since 1995. He has no history of chronic disease. During the physical examination, no pathological findings were found. On PA CXR, pleural plaques were observed in the bilateral hemithorax (Figure 6). On HRCT, LAPs with a mediastinal and hilar diameter of 11 mm transversely, significant calcified pleural plaques in mediastinal sections, and millimetric nodules with the largest dimension of 5 mm were detected in both lung parenchymas. There was silica exposure in the occupational anamnesis of the patient, but there was no occupational asbestos exposure. Environmental asbestos exposure was present for 15 years. There was no radiological or clinical evidence that suggested asbestosis. The patient was diagnosed with benign calcified pleural plaque, which was assumed to be caused by environmental asbestos exposure.

Case 5: A 70-year-old male patient was admitted to our clinic complaining of exertional dyspnea. In his occupational anamnesis, the patient, born in Çorum Sungurlu, had a history of working as a lathe master for 40 years. He had no history of systemic disease. There were no abnormal findings discovered during the physical examination. On the PA CXR, pleural plaques were found in the lungs (Figure 7). On HRCT, bilateral pleural calcific plaques were also present on the bilateral diaphragmatic surfaces. In addition, in his occupational anamnesis, the patient was exposed to metal shavings, but there was no occupational asbestos contact. There was no evidence of asbestosis in the clinical or radiological findings. The patient was diagnosed with benign calcified pleural plaque associated with environmental asbestos exposure.

Case 6: A 46-year-old male patient was referred to our clinic because of calcified plaques on the PA CXR. The patient had no active symptoms. The patient, born in Kütahya, had a history of living in an asbestos-plastered house from birth until age 10. His occupational anamnesis had a two-year history of working as a construction worker. Afterward, he worked as a security guard for ten years, and for the last six years, he has been working in the production of magnesium sulfate heptahydrate. He had no history of systemic disease. The physical examination was normal. On the PA CXR, he had calcified pleural plaques. Irregular pleural thickening, nodular pleural plaques, and calcifications were observed in both hemithorax on HRCT (Figure 8). While there was silica contact in his occupational anamnesis, there was no occupational asbestos contact. Asbestosis was not detected in clinical or radiological examinations. The patient was diagnosed with benign calcified pleural plaque associated with environmental asbestos exposure.

DISCUSSION

In the early 1900s, small enterprises were closed as a requirement of the industrial age, and large enterprises engaged in mass production began to open. Along with increasing industrial production, many chemical materials, the harms of which are not well known, have been used in the production sector. Asbestos is one of them (5,6). Asbestos, a fibrous mineral, used in the industrial field for many years due to its resistance to physical and chemical conditions (1). Due to these features, it was included in thousands of industrial products, especially from 1945 to 1965 (5,7). It has been used as the primary insulation material in many industrial areas. The use of asbestos peaked in 1972, with approximately 775,000 tons in the United States (8).

A series of articles were written about the incidence of lung cancer among asbestos workers in England, Germany, and America between 1935 and 1953 (9). It has long been known that inhaling asbestos fibers can cause diseases such as benign pleural plaques, pleural calcification, pleural effusion, diffuse pleural thickening, asbestosis, malignant mesothelioma (MM), and lung cancer (1,10).

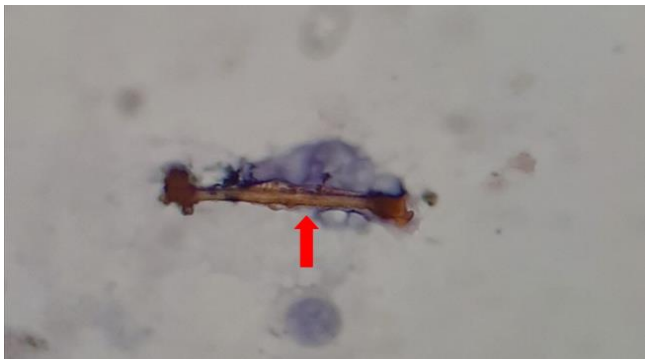

Figure 4: Light microscope image of Case-2: Ferruginous body (shown with a red arrow) (HEX1000)

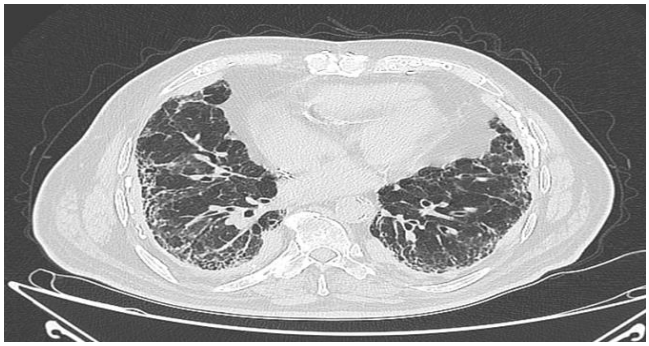

Figure 5: High-resolution CT scan of the lung parenchyma of Case 3. It shows thickening of the interstitial septa, traction bronchiectasis, ground glass infiltrations with nodules, and a honeycomb pattern in the lung parenchyma of both peripheral lobe

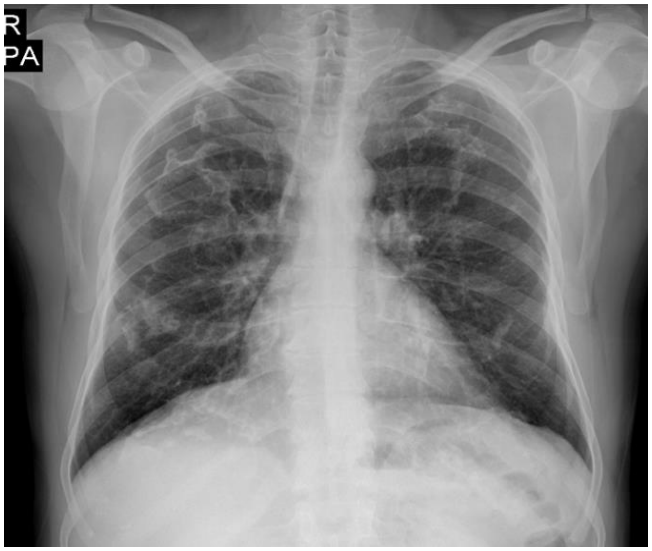

Figure 6: Calcified pleural plaques in the posteroanterior chest X-ray image of Case-4

With the understanding of the effects of asbestos on human health, limitations on its use worldwide began in the late 1980s and early 1990s. Sweden was the first country to ban the use of asbestos in Europe (8). Asbestos extraction, use, and placement on the market have been completely banned in the European Union since 2005 (11). Canada closed its asbestos mines in Quebec in 2012 (8). Asbestos use was prohibited in Turkey as of December 31, 2010, when the regulation published in the Official Gazette on August 29, 2010, numbered 27,687, went into effect (4). However, between 1983 and 2010, 500,000 tons of asbestos were used in many industrial fields in Turkey. The industrial asbestos exposure of the workers in the business fields related to the products in which this amount is used will continue for at least another 30 to 40 years. Therefore, asbestos exposure continues for those working in the repair, maintenance, and demolition of old industrial products (2).

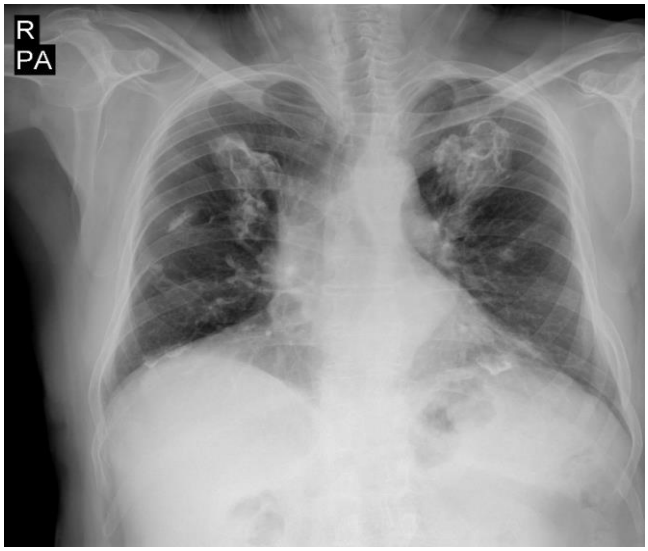

Figure 7: Calcified pleural plaques in the posteroanterior chest X-ray image of Case-5

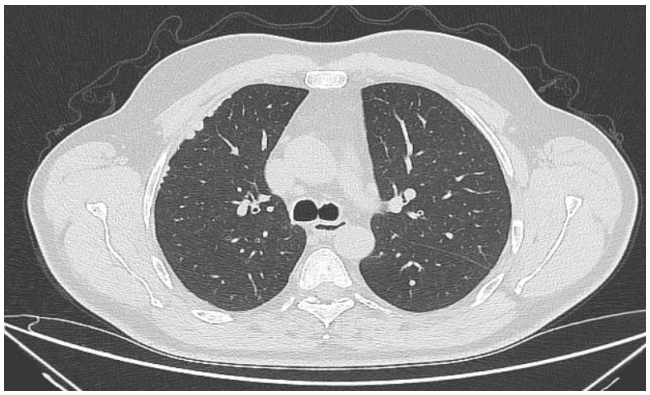

Figure 8: High-resolution CT scan of the right lung parenchyma showing nodular pleural plaques in Case 6

According to the fiber paradigm, all types of asbestos in serpentine and amphibole groups are fibrogenic for the lung. Asbestosis, which develops from lung fibrosis caused by asbestos, occurs approximately 20–30 years after the first exposure (12). In fact, our 3 cases diagnosed with occupational asbestosis were diagnosed 35.3 years after the first exposure.

Asbestosis is radiologically characterized by irregular reticular opacities, parenchymal bands, subpleural curvilinear lines, and a honeycomb appearance in the bilateral lower lobes of the lung. Diffuse interstitial fibrosis findings are also present in the histopathological examination of advanced-stage asbestosis (12). Therefore, radiological and pathological findings in asbestosis are mainly similar to idiopathic pulmonary fibrosis (IPF), and there may be some difficulties in the differential diagnosis. A strong exposure history is considered sufficient for diagnosis in the presence of fibrotic lung disease on HRCT for asbestosis. In the presence of a history of environmental or moderate asbestos exposure, pleural plaques are considered sufficient for diagnosis (13). The most crucial criterion in the differential diagnosis of asbestosis and IPF is a history of asbestos exposure. Rarely, histopathological examination of the tissue is used to confirm the diagnosis. In the histopathological examination, the presence of two or more ferruginous bodies per cm2 together with interstitial fibrosis in the tissue or one ferruginous body per milliliter in BAL is diagnostic for asbestosis (13,14).

According to the Helsinki criteria, asbestos-related disease may develop in the presence of a long exposure history, such as at least one year, asbestos cement factory, and demolition of buildings containing asbestos, or working directly with asbestos (12). In case three, which we mentioned in the case series, we observed that asbestosis developed due to intense asbestos exposure despite the short total exposure time. In the recent PRIMATE study conducted in Italy, methods were investigated to help estimate the cumulative asbestos exposures of 562 patients with MM, both qualitatively and quantitatively. In the study, lifetime asbestos exposure was evaluated by

two methods. Experts' evaluations were used to qualitatively evaluate occupational and non-occupational exposure, while a job exposure matrix (JEM) was used for quantitative evaluation. Experts' evaluations for occupational exposure were defined as the gold standard when the results of the two methods were compared. It was emphasized that no single approach could provide accurate and comprehensive exposure estimation. Generally, the expert method is preferred when assessing exposure in medico-legal settings, while the JEM method may be appropriate for epidemiological studies (15). In another study conducted in Italy, lung fiber burden and fiber type were investigated in 72 people with occupational and environmental asbestos exposure. The results reported no association with asbestos dose in MM, unlike asbestosis, which is dose-dependent (16).Asbestos-related diseases, mostly pleural plaques and MM, have been reported in relation to environmental exposures in regions such as China, Corsica, Greece, Cyprus, California, and Turkey (13,17,18). In a large-scale, field-based cross-sectional study of nonmalignant pleural diseases due to environmental asbestos exposure, 14.4% of participants had pleural plaques, 10.4% had diffuse pleural fibrosis, and 0.4% had asbestosis. This study measured the cumulative exposure level at 14-fiber years/mL^{-1}. It was concluded that the prevalence of asbestosis was low because this value was below 25 fiber years/mL^{-1}, the generally accepted threshold level for asbestosis (19). As a result of high-concentration exposures to asbestos fibers, the possibility of developing asbestosis increases, while lower-concentration exposures increase the possibility of pleural diseases (3,12,13,19). Furthermore, even when the cumulative dose is the same, the immediate exposure dose in environmental asbestos exposure is lower than in occupational asbestos exposure (18).

Rural areas in Turkey where environmental asbestos exposure is most intense are the cities of Eskişehir, Kütahya, Bilecik, Yozgat, Sivas, and Diyarbakır (1,3,18,20). Three of our cases, 4, 5, and 6, had a history of living in asbestos-plastered houses in areas with environmental asbestos exposure. While there was no finding in favor of asbestosis in the imaging of these three patients, they had calcified pleural plaques. It was thought that low concentration and intermittent environmental asbestos exposures might affect these cases.

In this case, series, we aimed to emphasize the importance of questioning occupational exposures as well as environmental exposures, which are common in Turkey, in the differential diagnosis of asbestos-related lung disease. Low-intensity exposures are more common with pleural diseases, while high-intensity exposures, which are mostly occupational, appear as asbestosis. In our case series, asbestosis was present in all three cases with occupational exposure to high concentrations. At the same time, calcified pleural plaques were present in the other three cases with environmental asbestos exposure.

The prospect of this case report is to highlight the need for successful environmental measurements, successful biological measurements, development of exposure indices, and JEMs to more accurately assess the exposure-effect relationship in occupational and environmental asbestos exposures. Epidemiological studies designed with these scales will better understand the burden and causality of asbestos-related diseases. It will also contribute to the development of measures. On the other hand, establishing a causal link between the agent and the disease will enable patients to access legal gains.

CONCLUSION

It is essential to detail the exposure history in diagnosing diseases caused by asbestos in the lungs and pleura. In Turkey, where environmental asbestos exposure is common, it is essential to take a detailed occupational anamnesis in the differential diagnosis of asbestos-related lung diseases. However, in diagnosis, radiological findings, mineralogical examinations in tissue, and demonstration of ferruginous bodies in tissue can be used.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - M.E.G., G.S., A.K., B.A., F.D., C.Ş.; Planning and Design - M.E.G., G.S., A.K., B.A., F.D., C.Ş.; Supervision - M.E.G., G.S., A.K., B.A., F.D., C.Ş.; Funding - M.E.G., F.D., C.Ş.; Materials - M.E.G., G.S., A.K., B.A.; Data Collection and/or Processing - M.E.G., G.S., A.K.; Analysis and/or Interpretation - M.E.G., G.S., A.K., B.A., C.Ş.; Literature Review - M.E.G., G.S., A.K., C.Ş.; Writing - M.E.G., G.S., A.K., C.Ş.; Critical Review - M.E.G., G.S., A.K., C.Ş., F.D., B.A.

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