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Imaging of asbestos-related lung and pleural diseases as an endemic exposure in Egypt

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Abstract

Background: Asbestos refers to a group of naturally occurring silicate minerals which have been traditionally used in building materials and household products. Inhalation of asbestos fibers, however, has been associated with adverse health outcomes, with the disease manifestations principally affecting the thorax. The aim of our study is to detect and evaluate the different radiological patterns of asbestos-related lung and pleural disease and its complications

Results: MDCT examination was able to assess and distinguish asbestosis as well as asbestos-related lung and pleural disease besides detection of any associated complications.

Conclusion: This study demonstrates that while reporting of malignant asbestos-related pleural disease is adequate, there is room for improvement in the reporting of more benign disease.

Keywords: Asbestos, Mesothelioma, Bronchogenic carcinoma, Round atelectasis, Pleural plaques, MSCT

Background

Asbestos is a group of minerals that occur naturally as bundles of fibers. These fibers are found in soil and rocks in many parts of the world. Asbestos was often used in buildings for insulation, flooring, and roofing and sprayed on ceilings and walls. They are primarily divided into two major categories, serpentine (chrysotile), and amphibole (amosite, crocidolite, tremolite, anthophyllite, and actinolite) fibers. The fibers are insoluble in water/inorganic solvents and are largely chemically inert. Inhalation of asbestos fibers, however, has been associated with adverse health outcomes, with the disease manifestations principally affecting the thorax. There can be a considerable latency period between exposure and the development of disease (may span many decades) [1].

Amphibole asbestos fibers are considered more hazardous to the human body with their fine, straight fibers having greater capability for deposition in smaller,

more distal airways. Similarly, the smaller, finer amphibole particles are more prone to inhalation as they are more easily mobilized from their source. Serpentine fibers are broader and mostly get deposited in the larger airways. This allows the body's natural defense mechanisms (i.e., mucociliary clearance) to better clear these fibers [1].

Occupational asbestos exposure is associated with several benign lung and pleural diseases, particularly asbestosis, pleural plaques, visceral pleural fibrosis, rounded atelectasis, and benign pleurisy, and several malignant diseases, mainly mesothelioma and lung cancer [2].

Sources of asbestos exposure The risk of asbestos exposure occurs mainly through the processing, manufacturing, and end-use of asbestos. Manufacturers commonly use asbestos in the following products: products containing asbestos cement like pipes, shingles, clapboards, sheets, vinyl-asbestos floor tiles, asbestos paper used in filtering and insulating products, textile products, and spray products used for acoustic, thermal, and fireproofing purposes [3].

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Occupations associated with asbestos include insulation workers, boilermakers, pipefitters, plumbers, steamfitters, welders, and janitors [3].

It has been clearly established that asbestos-related interstitial fibrosis (i.e., asbestosis) are associated with an increased risk of lung cancer, although asbestos-related lung cancer may occur in the absence of asbestosis. However, there is persistent controversy surrounding several aspects of asbestos-related diseases, particularly with respect to the consensus statement “workers with asbestos-induced pleural abnormalities are at increased risk for lung cancer compared with workers with similar exposures without these pleural abnormalities.” Pleural plaques are the lesions most commonly observed among asbestos exposed subjects [4].

Health surveillance of formerly asbestos exposed individuals focus on early detection of asbestos-related diseases, such as lung fibrosis (asbestosis), pleural plaques, mesothelioma, and lung cancer in particular. One main concern is the early and clear identification of lesions with a high risk of malignant changes and their undelayed clinical work-up. False positive results may lead to unnecessary and often painful diagnostic interventions, which create high costs when applied to a large cohort and also may discredit the whole program [5].

Screening by low-dose chest computed tomography (CT) scan was associated with a significant reduction of lung cancer mortality in some current or former heavy smokers between the ages of 55 and 75 years with a smoking history of at least 30 pack-years (one pack-year equals smoking one pack [20 cigarettes] per day for 1 year) [4].

Methods

This prospective observation study was conducted on 40 patients (32 males and 8 females) with asbestos-related

lung disease with an age range from 37 to 78 years mean \pm 55.40 years.

Cases were referred to the radiology department for multidetector computed tomography (MDCT) assessment after obtaining required consents and were approved by the ethical committee in our department.

The complaints varied between dyspnea, chest pain, cough, hemoptysis, fatigue, and loss of weight.

Inclusion criteria

This includes patients coming to the radiology department complaining of chest symptoms with history of asbestos exposure.

Exclusion criteria

None.

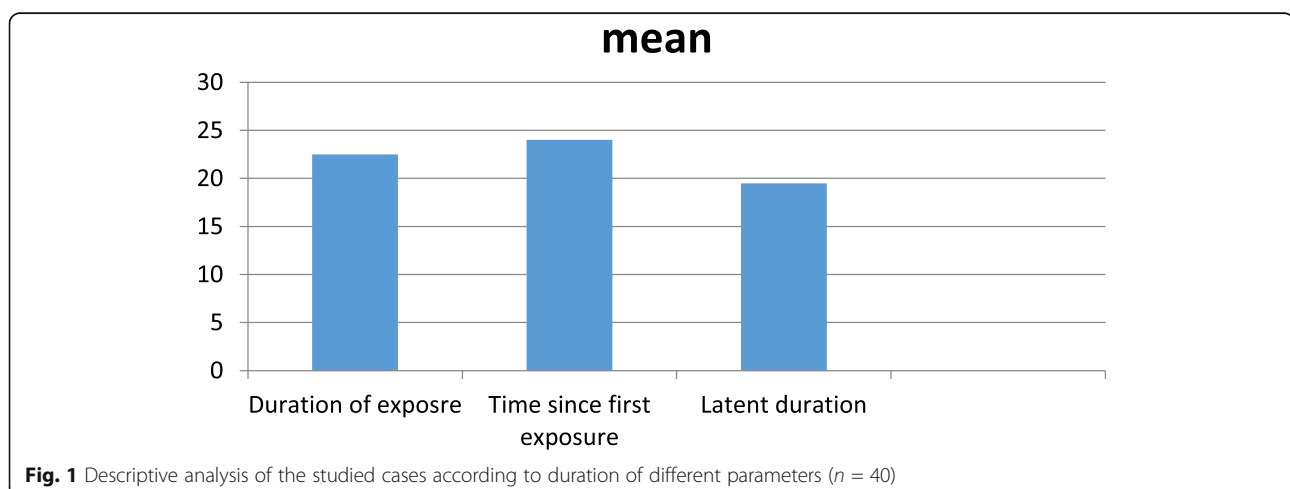
All cases were subjected to the following:

- Written consent and explanation of the technique and its aim
- Past medical history
- General asbestos exposure history: any direct contact with asbestos (source, intensity, and duration of exposure), age at first exposure, and years since first exposure
- Occupational exposure history

Protocol for MDCT

MDCT examination of the whole lung in supine position during one breath-hold with deep inspiration without administration of contrast material was applied (SOMATOM Sensation 16, Siemens Medical Solutions, Forchheim, Germany). A standard low-dose MDCT protocol was used: 120 kV, 10 mA for individuals with less than 80 kg, 20 mA for individuals with 80 kg and more, 16 \times 0.75 mm collimation, rotation time 0.5 s, table feed/rotation 18 mm. Images were reconstructed in three different ways.

Low-dose CT refers to scanning techniques which use tube current less than 100 mAs in an attempt to deliver



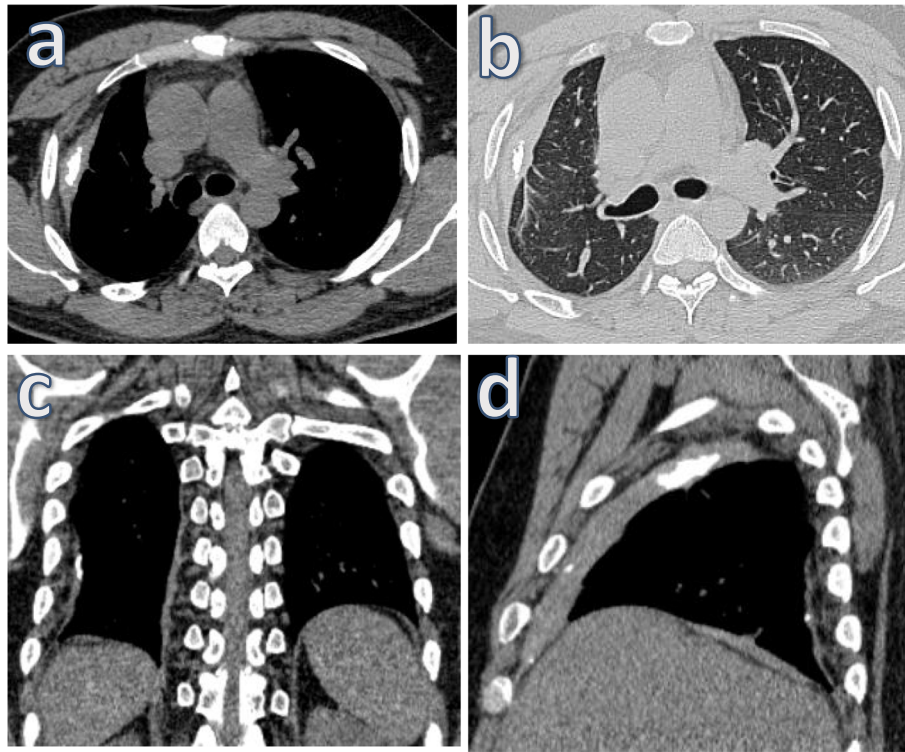


Fig. 2 A male patient 40 years old, a smoker, working as a concrete board cutter for 20 years. (a) axial mediastinal window, (b) axial lung window, (c) coronal mediastinal window and (d) sagittal mediastinal window non contrast CT scan of the chest showing right sided pleural plaques involving the costal and diaphragmatic pleura with dense curvilinear calcifications denoting benign asbestos related pleural disease.

reduced radiation dose to the patient while maintaining diagnostic quality images.

The first stack of images was reconstructed with 5 mm effective slice thickness applying an increment of 4 mm with a medium smooth soft tissue convolution kernel (Siemens B30 kernel) window setting (center (C) = 80 HU, window (W) = 400 HU) for analysis of soft tissue,

mediastinal, and pleural changes. The next stack of images was reconstructed as 1-mm-thick sections with a reconstruction increment of 0.5 mm and a sharp kernel (Siemens B50 kernel) (C = - 600; W = 1500) for detection of pulmonary nodules, and the last stack of images was reconstructed as a high-resolution set with 1-mm-thick sections every 10 mm with a B80 ultra sharp

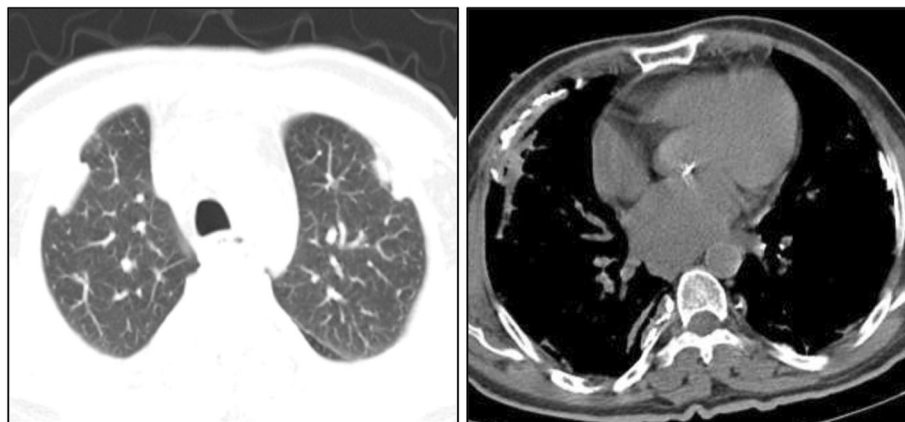


Fig. 3 A 65-year-old male smoker patient, working in ship building for 30 years, was diagnosed as having calcified pleural plaques for 2 years with a latent period of 28 years. Axial CT scan lung and mediastinal windows showing bilateral curvilinear calcified pleural plaques involving the costal pleura

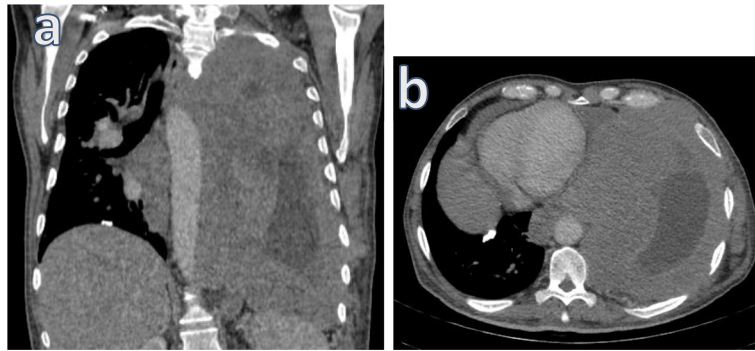


Fig. 4 A male patient 48 years old, a smoker with history of occupational exposure to asbestos for 31 years. (a) Coronal (b) axial mediastinal window contrast enhanced CT scan of the chest showing left sided mesothelioma in the form of non-uniform circumferential pleural thickening mounting to formation of pleural masses with left pleural effusion, total left lung collapse and contralateral mediastinal shift. Right diaphragmatic pleura calcified plaque is noted. Pericardial effusion and mediastinal lymphadenopathy are also noted.

reconstruction kernel ($C = -600$; $W = 1500$) for analysis of additional asbestos-related changes.

Image interpretation

Images were interpreted independently by three observers, two experienced in thoracic imaging (15–20 years' experience) and one novice. The inter-observer agreement was about 90%, and controversy was only in the list of findings.

Statistical analysis

All statistical calculations were done using computed program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) release 15 for Microsoft Windows (2006).

Results

This study was conducted on 40 patients with asbestos-related lung disease with the following results:

The mean duration of exposure of asbestos was 22.73 years, and the mean time since first exposure was 23.75 years with a latent duration about 19.36 years (Fig. 1).

In this study, 40% of patients showed malignant lesions in the form of mesothelioma in 27.5% and bronchogenic carcinoma in 12.5% of cases, and benign lesions were found in 60% of cases in the form of calcified pleural plaques in 40%, pleural effusion in 7.5%, lung fibrosis in 7.5%, and round atelectasis in 5% (Figs. 2, 3, 4, 5, and 6).

Calcified pleural plaques were found in 32 cases (80%), of which 16 cases showed associated malignancy (40%) and 16 cases showed calcified pleural plaques only (40%) (Table 1).

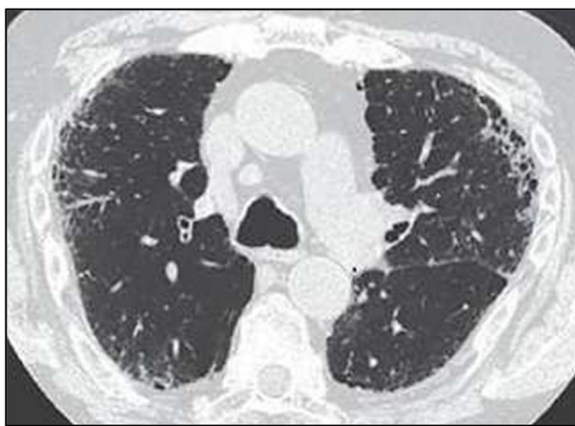


Fig. 5 A 56-year-old male smoker patient, working as a concrete board cutter for 32 years, was diagnosed as having asbestosis for 6 years with a latent period of 26 years. Axial CT scan lung window showing bilateral subpleural reticulation and coarsening of pulmonary interstitium associated with honeycombing denoting interstitial fibrosis



Fig. 6 A 56-year-old male smoker patient, working in plasters for 13 years, was diagnosed as having round atelectasis for 3 years with a latent period 10 years. Axial CT scan lung window showing left lower lobe subpleural soft tissue density with comet tail sign and underlying pleural thickening denoting round atelectasis

Table 1 Distribution of the studied cases according to asbestos related disease ($n = 40$)

Asbestos-related disease	No.	%
Mesothelioma	11	27.5
Bronchogenic carcinoma	5	12.5
Calcified pleural plaques without associated malignancy	16	40
Pleural effusion	3	7.5
Lung fibrosis	3	7.5
Round atelectasis	2	5

There were no statistically significant differences between the malignant and benign groups as regards smoking status (p value 0.237) as well as the presenting symptoms. However, there was a significant difference between the two groups as regards duration, time of exposure, and latent period as cancer susceptibility increases in patients with longer duration of exposure and longer latent period (Table 2).

On univariate analysis, the significant factors affecting malignancy of lesions were duration of exposure and time since first exposure (Table 3).

Discussion

Asbestos-related disease is a worldwide problem. Pleural plaques (PP), asbestosis, malignant mesothelioma, pleural effusion, diffuse pleural thickening, and bronchogenic carcinoma constitute asbestos-related diseases with the pleural plaques being the most common manifestation. Detection of early pleural and parenchymal changes on computed tomography (CT) is more sensitive than chest X-ray [6].

In the present study, we aimed to detect and evaluate the different radiological patterns of asbestos-related lung disease and its complication by including

Table 2 Comparison between the two studied groups according to duration of different parameters

	Malignant (<i>n</i> = 24)	Benign (<i>n</i> = 16)	Test of Sig.	<i>p</i>
Duration of exposure to asbestos				
Min.–Max.	37.0–78.0	45.0–70.0	<i>U</i> = 94.0*	0.007*
Mean ± SD.	55.46 ± 12.49	55.31 ± 7.51		
Median	55.0	54.0		
Time since first exposure (years)				
Min.–Max.	10.0–36.0	13.0–35.0	<i>t</i> = 3.019*	0.005*
Mean ± SD.	20.04 ± 6.59	26.75 ± 7.22		
Median	19.0	29.0		
Latent duration (year)				
Min.–Max.	3.0–8.0	1.0–5.0	<i>t</i> = 4.231*	< 0.001*
Mean ± SD.	5.67 ± 1.20	2.47 ± 1.34		
Median	6.0	2.0		

Table 3 Univariate and multivariate analysis for the parameters affecting cancer

	Univariate		Multivariate	
	p	OR (95% C.I.)	p	OR (95% C.I.)
Duration of exposure to asbestos	0.009*	1.148(1.035–1.275)	0.110	0.312(0.075–1.303)
Time since first exposure (years)	0.010*	1.152(1.035–1.283)	0.112	3.347(0.753–14.874)

40 patients with asbestos-related lung disease with mean age of the studied patients being 55.4 years with male predominance at 80%. Of patients, 55% were smokers, 30% were ex-smokers, and 15% were non-smokers.

In the current study, we found that the mean duration of asbestos exposure was 22.73 years and the mean time since first exposure was 23.75 years with a latent duration about 19.36 years.

In a study by Ahn et al. [7], they found that the mean duration of Asbestos exposure for the compensated workers was 16 years. The most common duration of exposure involved the group exposed to asbestos for 10–20 years (eight cases). The mean duration of the latency period was 22.6 years. The most common duration of the latency period was 20–30 years.

In the present study, we found that 40% of patients showed malignant lesions in the form of mesothelioma in 27.5% and bronchogenic carcinoma in 12.5% of cases. On the other hand, 60% showed non-cancer lesions in the form of pleural effusion in 7.5%, calcified pleural plaques in 40%, lung fibrosis in 7.5%, and rounded atelectasis in 5%. In a study by Çoşgun et al. [6], it was found that pleural plaques due to environmental asbestos exposure were found in 66 of the 75 patients on chest CT distributed as follows: 64 (96.6%) costal plaques, 44 (66.6%) diaphragmatic plaques, and 9 (13.6%) pericardial plaques.

Comparing patients diagnosed with malignant and benign lesions, there were no significant differences as regards age, sex, smoking status, and presenting symptoms.

As regards smoking status in contrary to our result, the bulk of epidemiologic evidence implicates asbestos as a carcinogen, the effect of which is augmented by cigarette smoking. A synergistic relationship between the two carcinogens is commonly accepted, and a review of 23 studies addressing smoking and asbestos exposure lends support to a multiplicative interaction [8].

In a retrospective study of 98,912 asbestos workers, Frost et al. [9] demonstrated that the interaction between smoking and asbestos exposure was greater than the additive (i.e., multiplicative) to the occurrence of lung cancer, while lung cancer risk remained increased even 40 years after smoking cessation.

In the current study, we found that there were significant differences between the two groups as regards duration, time of exposure, and latent period as cancer susceptibility increases in patients with longer duration of exposure and longer latent period, and by univariate analysis, the significant factors affecting malignancy of lesions were duration of exposure and time since first exposure.

This was also detected by other studies as they revealed that the risk of malignant mesothelioma MM is very low in the first 10–15 years [10]. The mean latency period has been repeatedly found to be 30–40 years, and more than 90% of MM were diagnosed more than 15 years after the first asbestos exposure [11, 12].

However, MM cases were reported with a very brief latency period and epidemiologic studies support the hypothesis that heavy asbestos exposure may result in a shorter induction period [13].

Bianchi et al. [14] investigated 325 mesothelioma cases that occurred in the shipbuilding industry; 15.7% (50 cases) had latency < 10 years.

The consensus of international experts is that a minimum of 10 years from the first exposure is required to attribute MM to asbestos exposure [15]. This difference may be caused by a short history of occupational asbestos use and relatively younger compensated workers compared to other countries. For example, reviewing the series of 557 MM of the pleura in Italy, latency period ranged from 14 to 75 years (mean, 48.8 years; median, 51.0 years) [14].

Moreover, Mastrangelo et al. [16] showed that a significant increase in asbestos risk was found with increasing cumulative asbestos exposure, but not with time since first exposure, peak exposure, duration of exposure, age, and smoking. It can be seen that the significant risk factors were cumulative exposure to asbestos, time since first exposure and peak exposure for pleural plaques, and time since first exposure for diffuse pleural thickening.

Conclusion

This study demonstrates that while reporting of malignant asbestos-related pleural disease is adequate, there is room for improvement in the reporting of more benign disease.

Abbreviations

MDCT: Multidetector computed tomography; CT: Computed tomography; HU: Hounsfield unit; PP: Pleural plaque; C: Center; W: Window; SPSS: Statistical Package for the Social Science; MM: Malignant mesothelioma

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Authors' contributions

MF shared in the study conception and design, collecting patients' data, processing CT findings at the CT work station, and shared in writing and correcting the manuscript and revision. MK shared in the study conception and design, acquisition of data, analysis and interpretation of data, and drafting of manuscript. YH shared in the study conception and design, clinical examination, acquisition of data, analysis and interpretation of data, and drafting of the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analyzed during the study are available upon reasonable request.

Ethics approval and consent to participate

No individual data was included in the study.

This study was approved by the Research Ethics Committee of the Faculty of Medicine at Cairo University Kasr El-Aini in Egypt in March 2018, reference number I-110318.

All patients included in this study gave verbal informed consent to participate in this research.

If the patient was unconscious at the time of the study, written informed consent for their participation was given by their legal guardian.

Consent for publication

All patients included in this study gave written informed consent to publish the data contained within this study.

If the patient was unconscious when consent for publication was requested, written informed consent for the publication was given by their legal guardian.

Competing interests

The authors declare that they have no competing interests.

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