

Pleural plaques in smoking-associated fibrosis and pulmonary asbestosis

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Dear editor

Bledsoe et al presents an interesting study examining the disparities between radiologic and pathologic diagnoses of asbestosis in cases referred for consultation in pulmonary malignancy.¹ The authors conclude that the clinical diagnosis of asbestosis cannot be reliably distinguished from interstitial fibrosis in heavy smokers. These findings highlight the confounding role of cigarette smoking in the diagnosis of asbestosis when it is based on non-pathologic criteria. Clinical and radiographic characteristics of lung injury following particle exposure (including fibers in which one diameter of the particle is 3× that of the other by definition) are often comparable.² The results of this investigation¹ support further evaluation of a role for cigarette smoking in interstitial fibrosis. Furthermore, asbestos exposure can cause several non-malignant diseases of the pleura and lungs (ie, pleural effusions, pleural plaques, diffuse pleural fibrosis, rounded atelectasis, and asbestosis). Malignancies are also associated with asbestos exposure (ie, lung and laryngeal cancers and mesothelioma).^{3,4} Relationships between the dose–response and prevalence of asbestos-related diseases are complex.^{3,5} The injury requiring the least exposure, and which accordingly demonstrates the highest prevalence, is pleural plaque; 80% of individuals significantly exposed to asbestos (total dose of 0.1 fiber-year or less) will have plaques on the chest X-ray while only 0.5%–8% of an unexposed population will reveal such findings.⁶ Mesothelioma impacts 2,500 to 3,000 workers annually in the United States and its risk is elevated at a total asbestos dose of between 0.1 and 1.0 fiber-year. Those diseases requiring the greatest asbestos exposure are lung cancer and asbestosis; the risk for both is considered elevated at approximately 25 fiber-years. Bledsoe et al identify 24 cases with International Labour Organization (ILO) profusion score of ≥1, out of which only six cases show histological evidence of asbestosis. Of the remaining 18 cases, 16 are identified to have significant smoking history whereas two subjects had unknown smoking status. They observe radiographic evidence of pleural plaques in 82 (44%) of the cases included in the study. It would strengthen the conclusions of the study to know if those diagnosed with asbestosis demonstrated a higher prevalence of pleural plaques than those with cigarette smoking-related fibrosis.¹

Disclosure

The authors have no conflicts of interest in this communication.

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Authors' reply

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Dear editor

In our report,¹ we describe 24 cases of pulmonary fibrosis with ILO profusion ≥ 1 that were split into two groups based on histopathologic findings – six cases with evidence of asbestosis and 18 most consistent with smoking-related fibrosis. The mean potential asbestos exposure duration in each of these groups was 25 years. Pleural plaques were identified by chest radiograph in 5/6 (83%) and 13/18 (72%) of those with and without histopathologic evidence of asbestosis, respectively. As detailed in the letter by Sangani et al, pleural plaques are seen frequently and relatively early in the clinical course of patients with asbestos exposure. Therefore, in our study, given a mean asbestos exposure of 25 years, the high proportion of cases with pleural plaques is not surprising.

The detection of pleural plaques by chest radiograph is not entirely sensitive or specific,^{2,3} but with the appropriate exposure history and imaging findings is considered a reliable marker of asbestos exposure. Similarly, the association between pleural plaques and asbestosis on imaging is generally well accepted,² and the presence of plaques may be useful evidence that parenchymal fibrosis is asbestos-related. However, pleural plaques may also obscure assessment

of underlying pulmonary parenchymal disease by chest radiograph,⁴ making assessment of fibrosis and establishment of the etiology of fibrosis difficult. Furthermore, while some studies have shown a correlation between pleural plaques and microscopic asbestosis,⁵ others have found that plaques do not predict histologic asbestosis.⁶ In our study, in the majority of cases with ILO profusion ≥ 1 and despite the presence of asbestos exposure and pleural plaques, smoking-related fibrosis as the cause of interstitial fibrosis could not be excluded given histomorphology consistent with what has been described in smoking-related fibrosis and the absence of asbestos bodies. Our findings suggest that mild pulmonary fibrosis that is attributed to asbestosis on chest radiograph may not be reliably distinguished from other causes of interstitial fibrosis, such as that related to smoking, including in patients with asbestos exposure and pleural plaques.

Disclosure

The authors have no conflicts of interest in this communication.

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