Diagnosis of Asbestosis*

Primum Non Nocere

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The preceding essay "Asbestos-Related Disorders: A Realistic Perspective" explores the diagnostic uncertainty confronting the clinician when asked by the asbestos-exposed patient, "Do I have asbestosis?"

Little has changed in diagnosis and treatment of asbestosis since 1986, when a committee of experts proposed useful clinical diagnostic criteria that did not require lung biopsy.[1] The most significant development over those 10 years has been the widespread availability of thin-section high-resolution CT of the chest. While providing a strikingly more detailed image of the lung parenchyma, high-resolution CT may not add diagnostic sensitivity or specificity for patients whose plain chest roentgenogram is on the borderline between normal and abnormal (International Labour Organization categories 0/1 to 1/0).[2] Unfortunately, it remains true in 1997 that we have no therapy to offer the patient with asbestosis, although rapid advances in the understanding of the basic pathogenesis holds promise for intervention trials in the near future.

For an opposing viewpoint see page 1424

A subcontext of this article is the frustration many pulmonologists feel in being called upon to make or exclude a diagnosis of asbestosis in patients whose disease is in a subclinical or very mild stage of progression. Like idiopathic pulmonary fibrosis, asbestosis begins as a silent alveolitis in the years after initial exposure. The alveolitis may be present and progressive for decades before it can be detected by symptoms, exam, roentgenogram, or lung function. Asbestosis characteristically progresses at a slow pace over decades, so that the clinical expression of an exposure in youth may not come until as late as the seventh or eighth decade. Unfortunately, there are also patients who progress much more rapidly. The factors that make one exposed individual progress to clinical asbestosis while his similarly exposed coworker remains apparently disease-free are currently being elucidated. A better understanding of cellular switching on the path from alveolitis to fibrosis may also lead to effective ways to modulate the lung's chronic inflammatory response to inhaled asbestos, thus providing a form of secondary prevention in the exposed individual without disease.

The legal culpability of several large asbestos manufacturing companies in actively suppressing scientific information about asbestos health effects over several decades produced outrage in thousands of employees exposed during those years. Many now seek out medical opinions to determine whether they qualify for compensation under class action lawsuits; others wish only to find out whether they are among the affected. A part of the frustration of clinicians stems from the difficult task of making a diagnosis in cases where the disease is still mild and the manifestations subtle. At this early stage of disease, diagnostic uncertainty is greater, and in the absence of treatment there is no clinical therapeutic advantage to be gained by early diagnosis, although many who are exposed desire prognostic information.

A major point raised is whether claims of asbestosis are being made in excess of the true number of cases of asbestosis and other asbestos-related diseases. This would seem to be an easy question to answer, but in fact it is not. It is appropriately pointed out that the number of cases determined depends on the sensitivity and specificity of the criteria used in a case definition. But for asbestosis in the United States, we have no accurate means to estimate the true prevalence of the disease. There are neither uniform diagnostic criteria nor specific surveillance programs designed to capture even a representative sample of cases. Hence, any estimates of the numbers of cases must be just that—estimates based on reasonable assumptions, but estimates that are not currently subject to verification. The estimate cited by the National Institute of Occupational Safety and Health (NIOSH) in the 1994 *Work-Related Lung Disease Surveillance Report*,[3] an authoritative resource on occupational lung disease and prevalence, is based on multiple cause of death data from the death certificates and collected from all reported deaths by the National Center for Health Statistics. The NIOSH authors caution that "limitations of multiple cause of death data include under- or over-reporting of conditions on the death certificate by certifying physicians."[3] Estimates of the national prevalence of asbestosis

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would need to be based on an estimate of the numbers of individuals with asbestos exposure and the time elapsed since exposure (because of the long latency). However, measurements of the prevalence of asbestosis in selected high-risk groups have been performed and are helpful in assessing the accuracy of the estimates.

One recent 56-center survey of asbestosis prevalence in building construction workers applied uniform diagnostic criteria in evaluating 9,605 asbestos-exposed sheet metal workers between 1986 and 1993 for whom at least 20 years had elapsed since entering the trade.[4] Among these volunteers, who may have self-selected according to their heavier exposure categories, the prevalence of parenchymal fibrosis was 12.3% or approximately 1,180 prevalent cases among those screened.

It is appropriately pointed out by Rosenberg that "without utilizing uniform criteria, occurrence data . . .are not comparable." The estimates of US asbestosis prevalence subsequent to this statement illustrate some of the pitfalls inherent in quick estimates of disease prevalence based on multiple unconnected databases.

Nevertheless, let us assume this estimate is true, and further assume that the number of legal claims outnumbers the cases of asbestosis. What is the appropriate medical response? To answer this question, we should return to the clinical setting and address the clinical question of diagnostic criteria for asbestosis in patients with an appropriate occupational exposure history. In this circumstance, our practice should be, as in all other situations, guided by informed clinical judgment. The expert panel[1] was as acutely aware of all these issues in 1986 as we are today, and wisely settled on a thorough but noninvasive evaluation which would be expected to have a high degree of sensitivity and specificity, recognizing that all tests have some false-positives and false-negatives. The expert panel pointed out that the more clinical criteria were met, the higher the sensitivity and specificity of the evaluation.

These clinical criteria were tested against the gold standard of lung biopsy, in the study by Gaensler et al.[5] The study found an approximately 5% false-positive rate for the clinical criteria, *ie*, that 95% of the clinically diagnosed cases did indeed have asbestosis, and 5% had other disease. The 95% specificity of the clinical criteria for asbestosis suggested by this study is consistent with the other literature on clinical diagnosis. Given this specificity, should we be recommending and performing open or thoracoscopic lung biopsies on more patients who ask the question, "Do I have asbestosis?" Does the number of asbestos-related personal injury claims provide a sound rationale for taking steps to improve the specificity of diagnosis from 95% up to 97 or 99%?

I would argue emphatically no, and for two reasons. The first is that a problem of overdiagnosis of asbestosis could be largely corrected simply by the more widespread application of the clinical diagnostic criteria. The problem of overdiagnosis, if it exists, is not any failure of these criteria, but only a failure to apply them. The second reason is that the use of more frequent invasive procedures would subject patients to the risks of general anesthesia and surgery without providing any clinical benefit. The surgical mortality of thoracoscopic lung biopsy is approximately 1%,[6] but it might be lower—approximately 0.5%—in patients with mild asbestosis. Surgical morbidity would be expected in the range of 5 to 10%. Such procedures would not improve quality of life or survival either in the 95% with asbestosis, or (because of the modest benefits of therapy in nonvasculitic interstitial disease) in the 5% with nonasbestos disease or no disease. The cost of each such thoracoscopic procedure at our institution is approximately $19,000. Who would pay these costs?

Good clinical practice requires attention to clinical features that may help in distinguishing a treatable pulmonary vasculitis from asbestosis. Such findings as systemic symptoms, renal disease, the presence of serologic markers of vasculitis, or even an unusually rapid progression may indicate a patient with a potentially treatable condition. By the same token, judicious clinical practice requires avoiding the risk of invasive procedures when the patient stands to gain no therapeutic benefit from the information added by biopsy. Our current approach to asbestosis diagnosis, given the limitations of our technology and therapy, is already quite realistic.

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[†]Available from publications dissemination, NIOSH, 4676 Columbia Parkway, Cincinnati, OH 45226-1998; FAX (513) 533-8573.