NEWER CONCEPTS IN SILICA AND SILICATE LUNG DISEASE

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Pulmonary disease may be caused by exposure to free silica and a wide variety of nonfibrous silicates. The pulmonary disease caused by chronic exposure to free silica is usually referred to as silicosis or classical silicosis. The lesion typical of silicosis is said to be the silicotic or classical silicotic nodule. Classical silicotic nodules are rounded, whorled, well demarcated very fibrotic lesions clearly demarcated from the background lung. Microscopically they have a narrow rim of dust containing macrophages admixed with randomly oriented collagen fibres, an intermediate zone of concentrically arranged collagen and a central collagenous core which may be variably hyalinised and calcified. On the other hand the mixed duet fibrotic nodule is stellate and microscopically is composed of a central zone of collagen with a peripbery of linearly and radially arranged collagen admixed with dust laden macrophages.[1] The latter is said to be characteristic of pulmonary disease caused by exposure to free silica in combination with less fibrogenic dusts sucb as kaolin,iron oxide or carbon.[1-3]

The occurrence of pneumoconiosis consequent to pure nonfibrous silicate exposure is debatable since commercial silicates are often contaminated by other minerals of known fibrogenicity. Relatively few cases have been described and in many of these no accurate analytical data is available.

When examining histopathological specimens of lungs from cases of so-called classical silicosis I have often been struck by the frequency of lesions other than the classical silicotic nodules. For example in a study of the lungs from North Wales slate workers, who were exposed to dust containing between 30 and 35% free silica, "mixed dust" fibrotic nodules and interstitial fibrosis were noted in a considerable proportion of the cases as well as the classical silicotic nodules.[4] It is probably not surprising in view of the fact that slate contains considerable quantities of mica and other minerals such as chlorite, iron salts and titanium in addition to free silica. On further reflection it will be obvious that there are few if any situations wbere pure exposure to free silica occurs and it is nearly always accompanied by exposure to combined silicates. There is experimental evidence that the toxic effects of free silica on the lung can be modified by the presence of other minerals such as mica, haematite and coal probably by modifying the surface activity of the free silica particles but the results are difficult to predict.[5,6] At present the precise conditions of dose, duration of exposure, mineral composition and physicochemical properties for the development of classical silicotic, mixed dust fibrotic nodules and interstitial fibrosis in humans are not fully understood. Other factors also appear to be important such as subject variation and complicating disease.[7]

In this presentation I would like to outline the results that my colleagues and I have obtained from a study of autopsy lungs from a group of Cornish china clay workers since it sheds some light upon how these lesions develop.[8] It is also one of the few studies of a pure nonfibrous silicate pneumoconiosis in which good pathological and analytical data are available.

The Cornish china clay industry is largely confined to a small geographical area located around St. Austell in the South West of England. The industry started in the 18th century wben china clay and china stone deposits were worked and the products used in British pottery production. Since then the industry has expanded by increasing the production of china clay but china stone usage has ceased.

The lungs from 62 subjects who had worked in the Cornish china clay industry had been referred to the MRC Pneumoconiosis Unit between 1968 to 1981. These were studied both pathologically and mineralogically and occupational histories and chest radiographs, available in 39 cases, were obtained. As the study proceeded it became apparent that there was good agreement between the occupational histories and the mineral content of the lungs. Indeed mineralogical analysis often proved more accurate than the initial occupational history.

On the basis of the mineralogical findings three groups could be distinguished:

1. "China clay" group–kaolinite > 90%, quartz < 1.1% and feldspars < 1% by mass.
2. "China clay and china stone" group–kaolinite < 90%, quartz > 0.9%, feldspars > 1.0% by mass.
3. "Miscellaneous" group–did not meet conditions for groups 1 and 2; it was considered probable that there was exposure to other minerals.

Each lung was graded histopathologically for nodular and interstitial fibrosis and the size of any PMF lesion noted. When the histopathogical gradings were compared with the mineralogical values the following conclusions were reached:

a. Nodular fibrosis correlated better with quartz concentrations than kaolinite
b. Interstitial fibrosis correlated better with kaolinite concentration than nodular fibrosis
c. In the majority of cases it was relatively easy to separate the china clay cases from the china clay and china stone cases histologically.

This study shows that a pneumoconiosis may result from nonfibrous silicates in the absence of free silica, in this case kaolin, and interstitial fibrosis is the predominant lesion. Further studies of this type are necessary to comprehend the toxic effects of free silica and nonfibrous silicates on the human lung.

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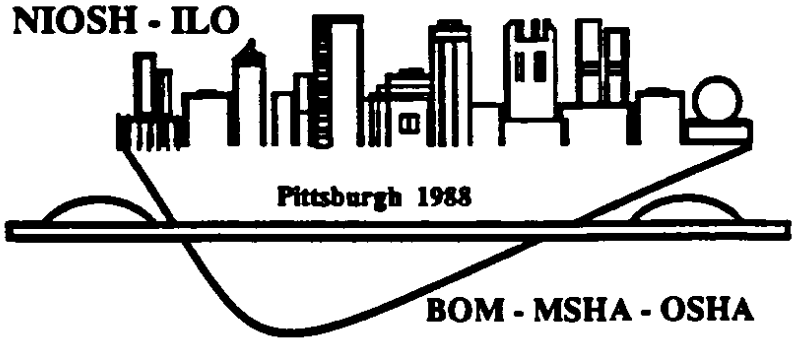

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