Wikipedia Excerpt: Search, Silica

Pathology

When small silica dust particles are inhaled, they can embed themselves deeply into the tiny alveolar sacs and ducts in the lungs, where oxygen and carbon dioxide gases are exchanged. There, the lungs cannot clear out the dust by mucous or coughing.

When fine particles of silica dust are deposited in the lungs, macrophages that ingest the dust particles will set off an inflammation response by releasing tumor necrosis factors, interleukin-1, leukotriene B4 and other cytokines. In turn, these stimulate fibroblasts to proliferate and produce collagen around the silica particle, thus resulting in fibrosis and the formation of the nodular lesions. The inflammatory effects of crystalline silica are apparently mediated by the Nalp3 inflammasome.

Furthermore, the surface of silicon dust can generate silicon-based radicals that lead to the production of hydroxyl and oxygen radicals, as well as hydrogen peroxide, which can inflict damage to the surrounding cells.

Characteristic lung tissue pathology in nodular silicosis consists of fibrotic nodules with concentric "onion-skinned" arrangement of collagen fibers, central hyalinization, and a cellular peripheral zone, with lightly birefringent particles seen under polarized light. In acute silicosis, microscopic pathology shows a periodic acid-Schiff positive alveolar exudate (alveolar lipoproteinosis) and a cellular infiltrate of the alveolar walls…..