Pneumoconiosis

9

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Pneumoconiosis is a nonneoplastic reaction of the lung to inhaled inorganic particles. Most affected patients are diagnosed based on a combination of an occupational history, pulmonary function tests, and radiologic findings. Surgical specimens showing pneumoconiosis are uncommon. The histologic reactions of the lung to the inhaled particles vary, depending on the chemical properties and size of the particulates, the length and dose of exposure, and the individual susceptibility of the host. The characteristic pathologic features in the types of pneumoconiosis most commonly encountered in surgical lung specimens are summarized in Table 9.1 and are illustrated in this chapter.

Table 9.1 Major types of pneumoconiosis and their typical pathologic features

Diseases	Pathologic features
Silicosis	Concentric hyaline nodules; progressive massive fibrosis
Mixed dust fibrosis	Stellate interstitial fibrotic nodules
Asbestosis	Diffuse interstitial fibrosis
Coal workers' pneumoconiosis	Dust macules and collagen fibrosis
Berylliosis	Sarcoid-like granulomas
Hard metal pneumoconiosis	Giant cell interstitial pneumonia

Silicosis

Silicosis (Figs. 9.1, 9.2, 9.3 and 9.4) occurs in reaction to inhaled crystalline silica. Simple silicosis consists of silicotic nodules involving predominantly the upper lobes. Hilar or mediastinal lymph nodes may also be involved. Progressive massive fibrosis is defined by the presence of nodular fibrosis that is greater than 1 cm in size and is the form of disease most likely to be associated with physiologically significant lung disease and disability.

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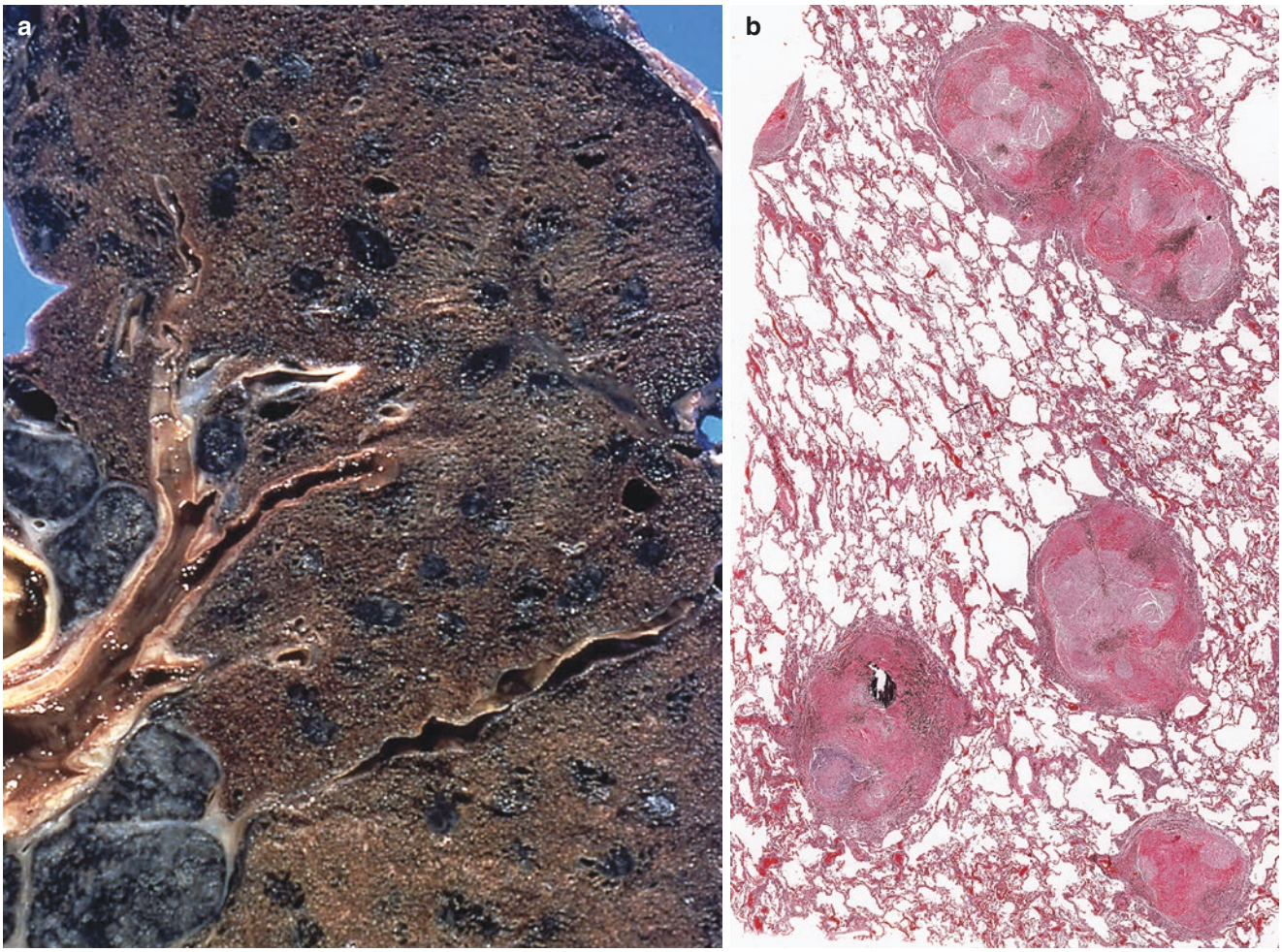

Fig. 9.1 Simple silicosis. (**a**) Cut surface of autopsy lung showing classic simple silicosis with multiple pigmented lung nodules measuring less than 1 cm in their greatest dimension. (**b**) Low-magnification photomicrograph of a section from the same autopsy lung showing multiple silicotic nodules consisting of concentric, hyalinized collagen bundles, and a peripheral rim of inflammatory cells in which dust-laden macrophages predominate

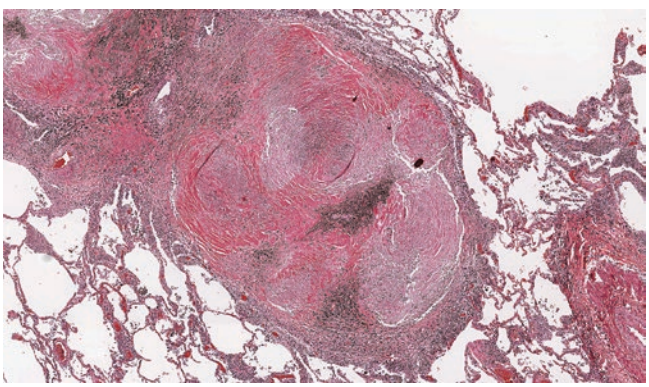

Fig. 9.2 Silicotic nodule. Higher-magnification view of nodule illustrated in Fig. 9.1b showing the coarse collagen bundles typical of silicotic nodules with the usual degree of associated dust-laden macrophages

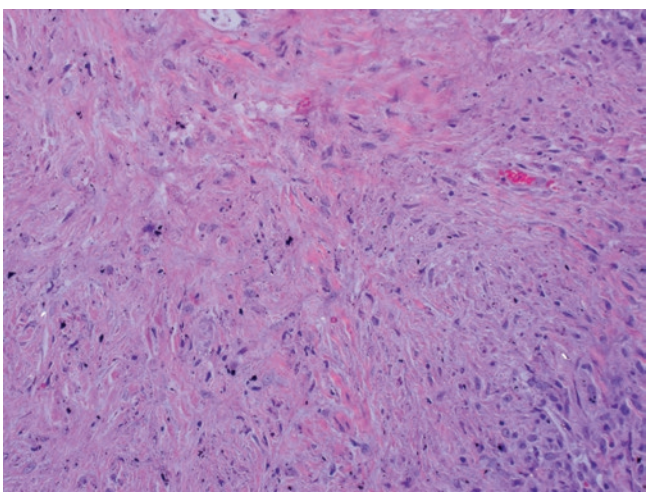

Fig. 9.3 Silicotic nodule. High-magnification photomicrograph taken using polarized light showing small, dimly birefringent crystalline particulates characteristic of free crystalline silica. Finding crystalline particles does not by itself establish the diagnosis of silicosis, which is predicated on the finding of particulates in the appropriate histologic context

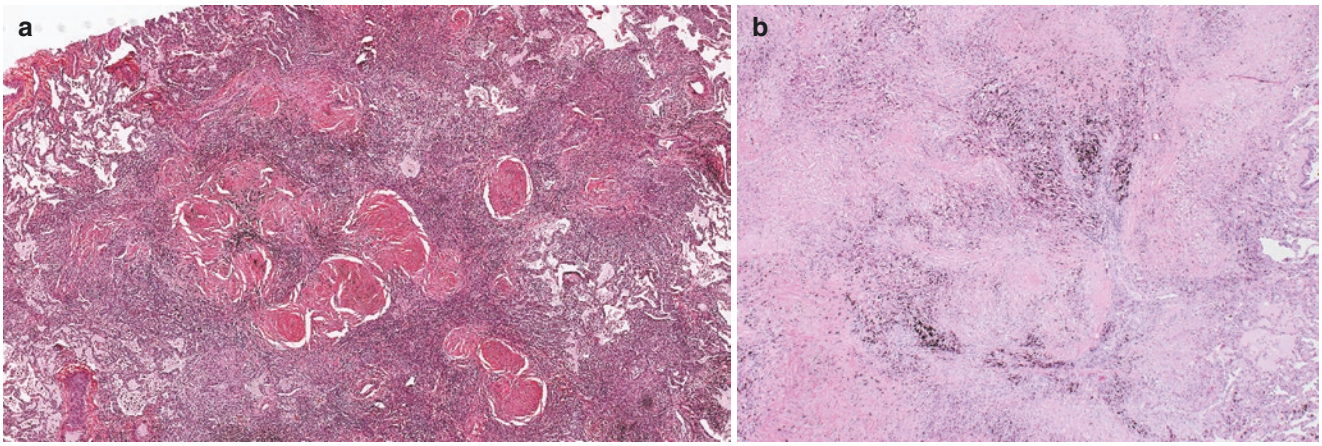

Fig. 9.4 Complicated silicosis (progressive massive fibrosis). (**a**) This low-magnification photomicrograph shows a conglomeration of silicotic nodules to form a larger complex lesion measuring more than 1 cm in greatest dimension. (**b**) Low-magnification photomicrograph showing another example of progressive massive fibrosis that involved nearly all of the right upper and middle lobes in an explanted lung from a patient with complicated silicosis

Mixed Dust Fibrosis

Mixed dust fibrosis (Figs. 9.5, 9.6 and 9.7) occurs when the inhaled particles consist of a mixture of silica and other less fibrogenic dusts such as anthracotic pigment and iron oxides. Compared to those of pure silicosis, the radiographic opacities of mixed dust fibrosis tend to be more irregular and stellate in shape. The main histologic finding is multiple bronchiolocentric stellate dust macules composed of dust-laden macrophages without well-formed silicotic nodules. Nonasbestos ferruginous bodies containing brown or black cores may also be present.

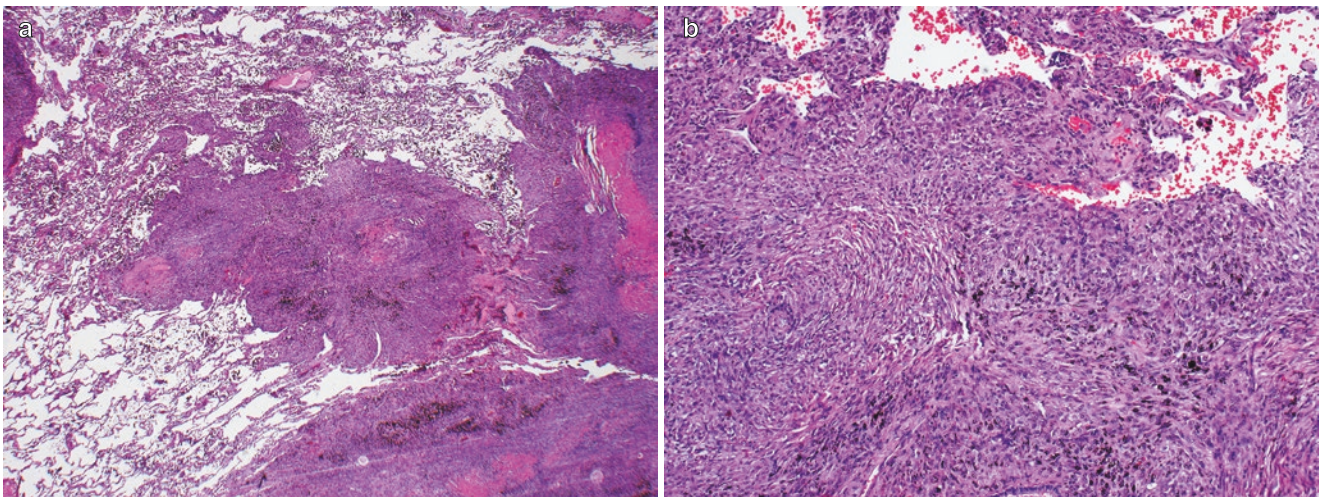

Fig. 9.5 Mixed dust fibrosis. (**a**) Low-magnification photomicrograph showing irregularly shaped dust macule without well-formed nodules. (**b**) Higher-magnification view showing epithelioid and spindled dust-laden macrophages making up the dust macule

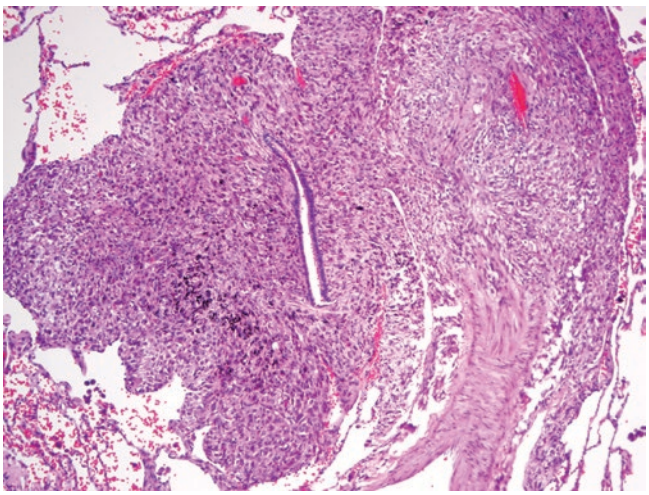

Fig. 9.6 Mixed dust fibrosis. High-magnification view of another dust macule surrounding a bronchiole in a patient with mixed dust fibrosis. The stellate macule consists of macrophages, fibroblasts, and various pigmented particulates without silicotic nodules

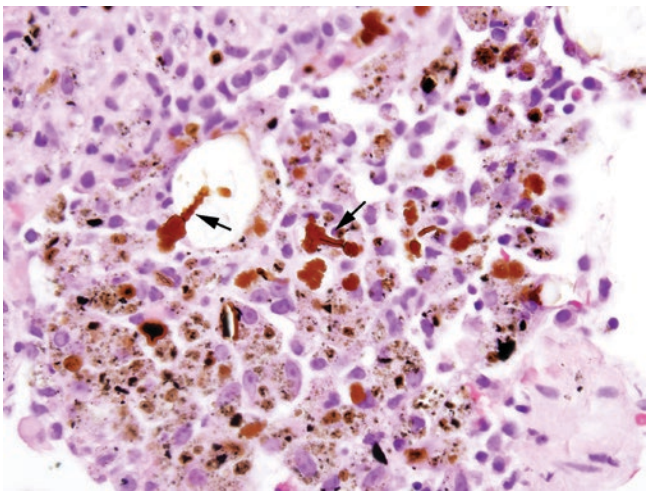

Fig. 9.7 Mixed dust fibrosis with nonasbestos ferruginous bodies. A high-magnification photomicrograph shows pigmented particulates and ferruginous bodies containing central black cores (arrows) that differ from the translucent cores typical of asbestos

Asbestosis

Asbestosis (Figs. 9.8, 9.9, 9.10 and 9.11) is interstitial fibrosis caused by inhaled asbestos fibers. The disease process preferentially involves the lower lobes of the lung and is commonly accompanied by pleural plaques/fibrosis. The interstitial fibrosis of asbestosis ranges from early peribronchiolar fibrosis to late stage disease that may be indistinguishable from usual interstitial pneumonia. Identifying asbestos bodies in a background of interstitial fibrosis in a patient with a supportive occupational history is key to the diagnosis of asbestosis.

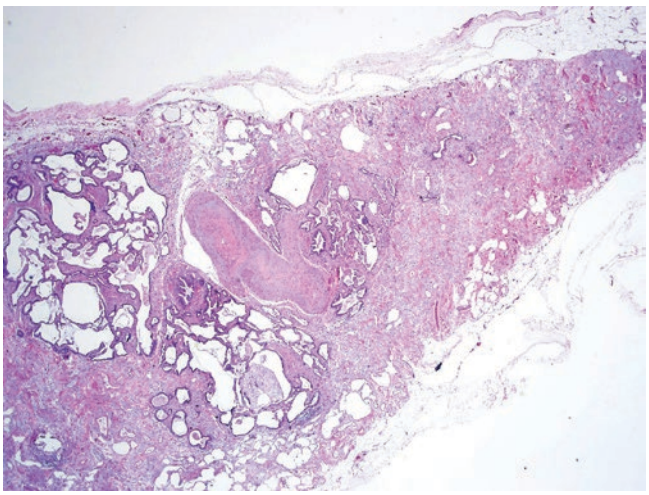

Fig. 9.8 Asbestosis. Low-magnification photomicrograph showing diffuse fibrosis with patchy scarring and honeycomb changes indistinguishable from usual interstitial pneumonia

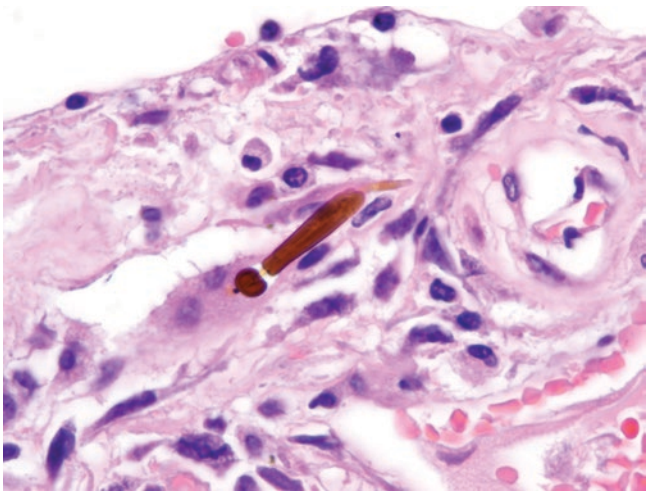

Fig. 9.10 Asbestosis. High-magnification photomicrograph showing a club-shaped asbestos body with a clear central core, a finding helpful in establishing the histologic diagnosis of asbestosis

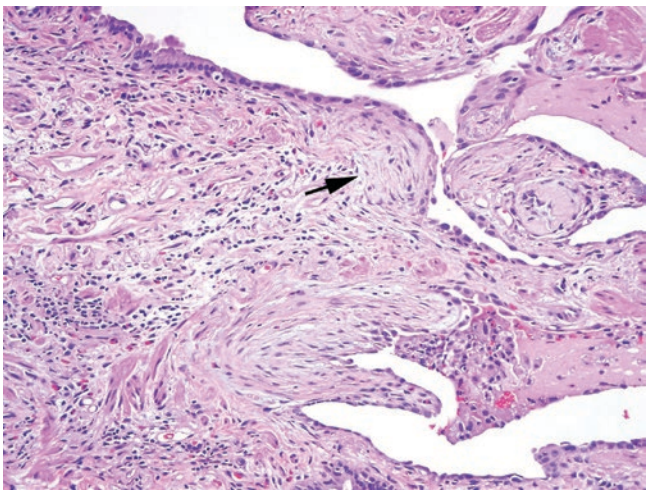

Fig. 9.9 Asbestosis. At high magnification, the interstitial fibrosis includes fibroblast foci (arrow) typical of those commonly seen in usual interstitial pneumonia of unknown cause (i.e., idiopathic pulmonary fibrosis)

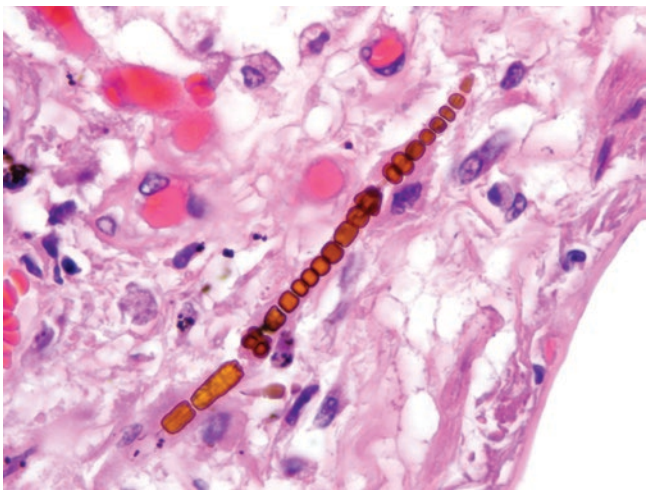

Fig. 9.11 Asbestosis. High-magnification photomicrograph showing another example of an asbestos body with a beaded appearance. Note the clear, refractile central core that distinguishes asbestos bodies from other forms of nonasbestos ferruginous bodies

Coal Workers' Pneumoconiosis

Coal workers' pneumoconiosis (CWP) (Figs. 9.12, 9.13, 9.14 and 9.15) occurs in reaction to inhaled coal dusts. Depending primarily on the silica content of the coal dust, CWP may be complicated by concomitant silicosis or mixed dust fibrosis. Simple CWP is characterized by a combination of dust macules and nodules with associated black coal dust. Complicated CWP is the term applied to those examples in which there is associated progressive massive fibrosis defined as conglomerate nodules measuring more than 1 cm in greatest dimension. As with complicated silicosis, progressive massive fibrosis in CWP can be very extensive and associated with severe respiratory impairment.

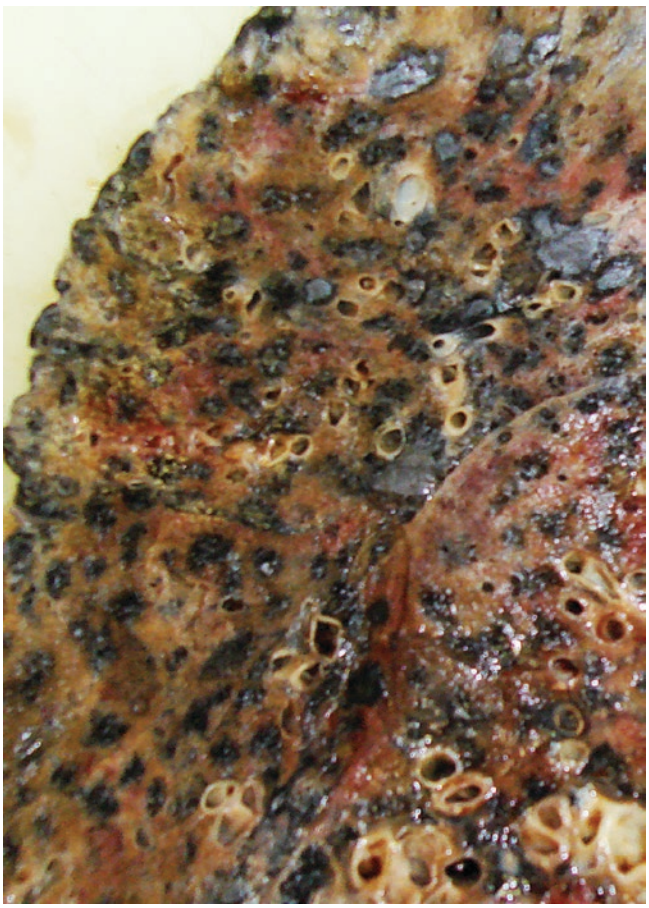

Fig. 9.12 Coal workers' pneumoconiosis (CWP). Cut surface of autopsy lung from a patient with CWP. There are numerous black pigmented macules and nodules with early complicated lesions measuring just over 1 cm in greatest dimension

Fig. 9.13 Simple CWP. Specially prepared Gough section of thinly sliced lung showing darkly pigmented dust macules characteristic of simple CWP

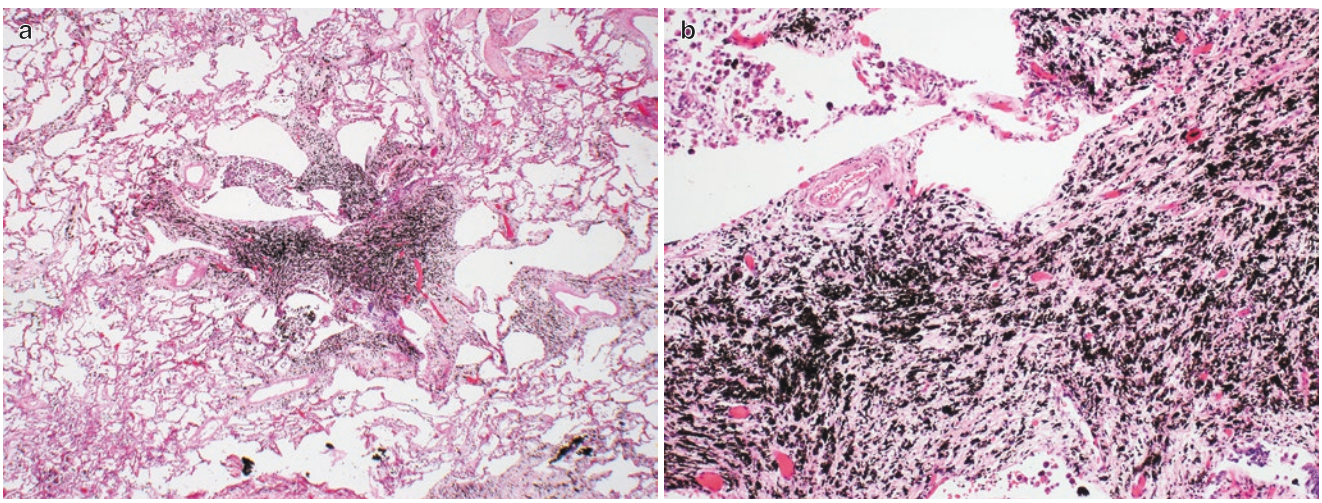

Fig. 9.14 Simple CWP. (**a**) Low-magnification photomicrograph showing a dust macule marked by deposits of black coal dust. (**b**) Higher-magnification view showing black coal dust in a dust macule characteristic of simple CWP

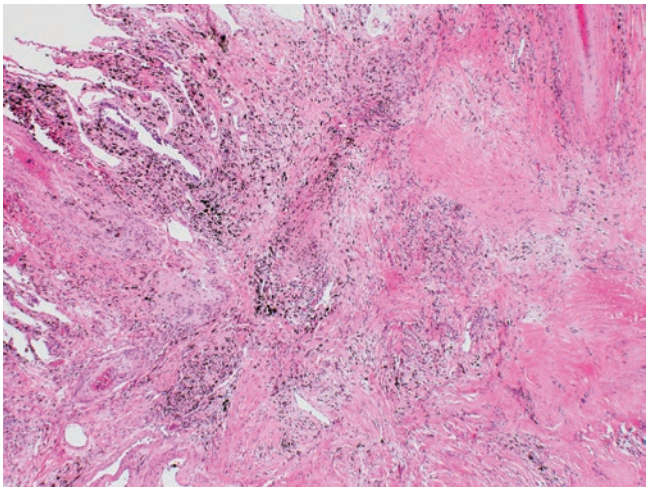

Fig. 9.15 Complicated CWP. Low-magnification photomicrograph of autopsy lung showing large area of progressive massive fibrosis in a patient with advanced CWP

Berylliosis

Berylliosis (Figs. 9.16 and 9.17) is a systemic disease caused by beryllium exposure. Chronic exposure may lead to pulmonary interstitial fibrosis accompanied by non-necrotizing granulomas, causing a pattern of granulomatous inflammation indistinguishable from pulmonary sarcoidosis in some patients. Beryllium is not visible on tissue sections. Differentiation from sarcoidosis requires correlation with exposure history, clinical course, and radiologic findings. The beryllium lymphocyte proliferation test is available only in specialized centers. If the test is positive, it confirms the diagnosis in an exposed worker with granulomatous disease in a lung biopsy.

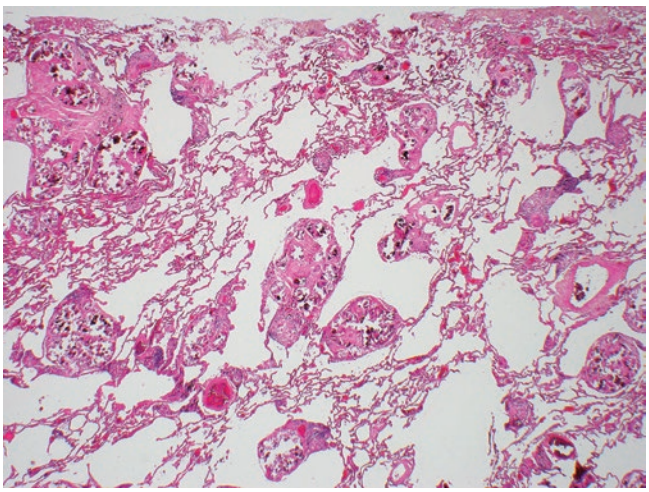

Fig. 9.16 Berylliosis. (**a**) low-magnification view showing interstitial fibrosis associated with multiple non-necrotizing granulomas. In this example, many of the granulomas show prominent cytoplasmic inclusions consisting of concentric calcifications (Schaumann bodies). Schaumann bodies are characteristic of the granulomas seen in berylliosis but are nonspecific and commonly seen in other granulomatous conditions such as sarcoidosis

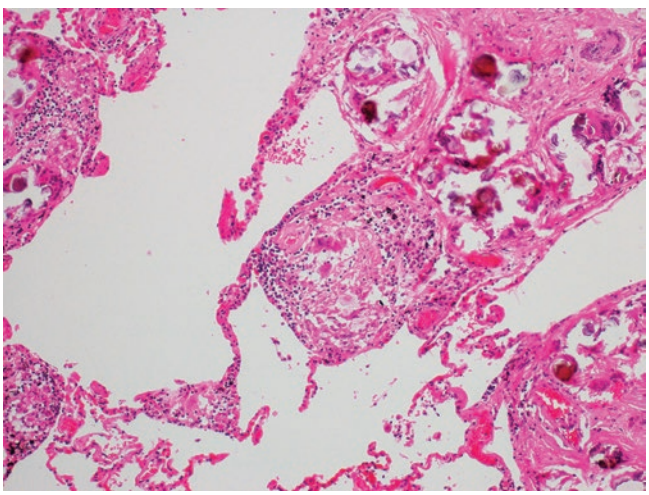

Fig. 9.17 Berylliosis. A high-magnification view of a non-necrotizing granuloma consisting of histiocytes, multinucleated giant cells, and a rim of lymphocytes

Hard Metal Pneumoconiosis

Hard metal pneumoconiosis is a diffuse interstitial lung disease caused by exposure to hard metals such as cobalt, tungsten carbide, titanium, chromium, and nickel. Cobalt is considered the etiologic driver in most patients. A common feature of hard metal pneumoconiosis is the intra-alveolar accumulation of multinucleated macrophages and epithelial giant cells, which is the reason why the disease is often referred to as giant cell interstitial pneumonia (Figs. 9.18, 9.19 and 9.20). Giant cell interstitial pneumonia is not pathognomonic, however, and also occurs in patients who lack evidence of cobalt exposure.

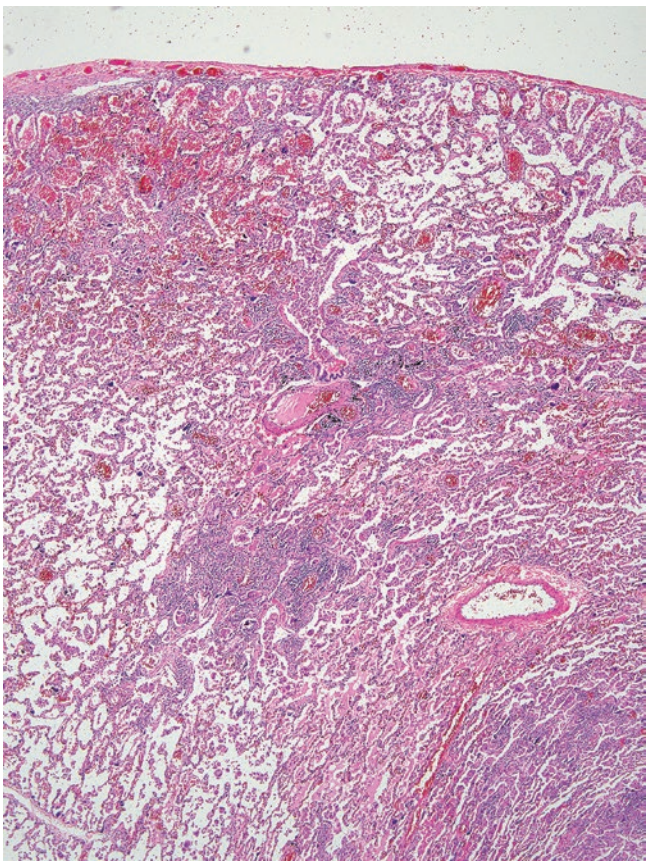

Fig. 9.18 Hard metal pneumoconiosis (giant cell interstitial pneumonia). A low-magnification view showing patchy interstitial thickening and cellular infiltrates distributed in a bronchiolocentric fashion. At this low magnification, you can see numerous multinucleated giant cells, many of them with hyperchromatic nuclei

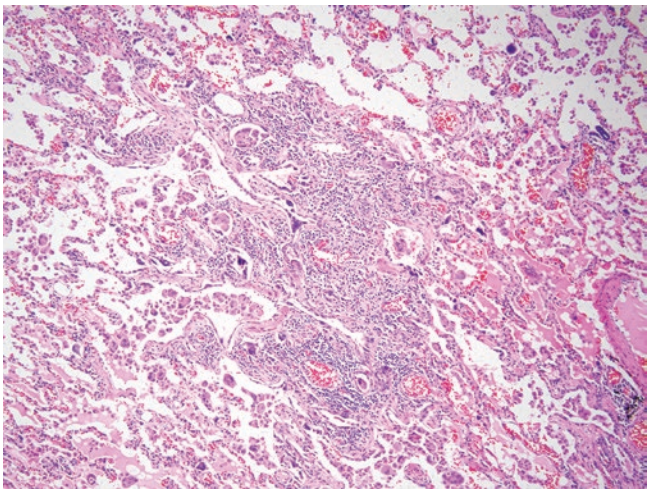

Fig. 9.19 Hard metal pneumoconiosis (giant cell interstitial pneumonia). A higher-magnification view showing expansion of peribronchiolar interstitium by inflammation with prominent multinucleated giant cells composed of both epithelium and alveolar macrophages. The multinucleated epithelial giant cells are TTF-1 positive (not shown) and represent pneumocytes

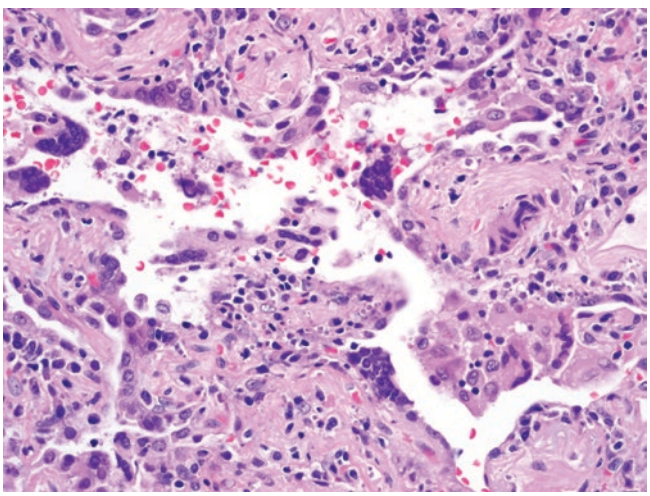

Fig. 9.20 Hard metal pneumoconiosis (giant cell interstitial pneumonia). High-magnification view showing intra-alveolar and surface epithelial giant cells

Other Rare Types of Pneumoconiosis

Other rare types of pneumoconiosis include a pattern of disease resembling simple coal workers' pneumoconiosis in workers exposed to aluminum dust (Figs. 9.21 and 9.22). Other inorganic particulates, including other metals, can occasionally cause occupational lung disease frequently characterized by the accumulation of a combination of dust macules and fibrosis.

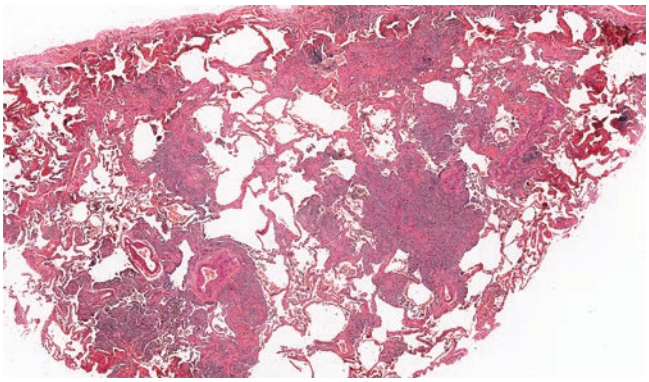

Fig. 9.21 Aluminum pneumoconiosis. Low-magnification photomicrograph showing multiple dust macules in which peribronchiolar interstitium is expanded by prominent collections of dust-laden macrophages

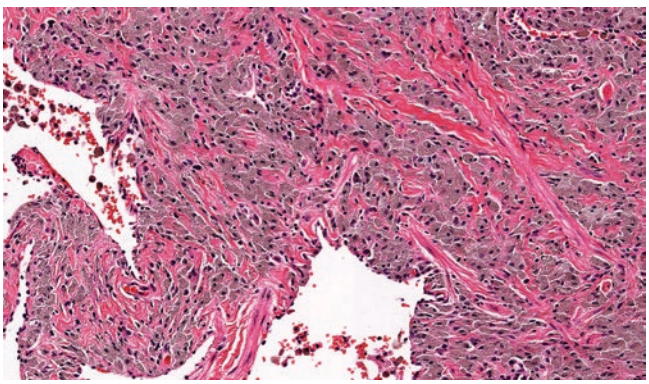

Fig. 9.22 Aluminum pneumoconiosis. High-magnification view of a dust macule showing macrophages containing finely granular, grayish-brown particles characteristic of aluminum

Suggested Reading

Anttila S, Sutinen S, Paananen M, Kreus KE, Sivonen SJ, Grekula A, Alapieti T. Hard metal lung disease: a clinical, histological, ultrastructural and x-ray microanalytical study. Eur J Respir Dis. 1986;69:83–94.

Aronchick JM, Rossman MD, Miller WT. Chronic beryllium disease: diagnosis, radiographic findings, and correlation with pulmonary function tests. Radiology. 1987;163:677–82.

Balmes JR, Abraham JL, Dweik RA, Fireman E, Fontenot AP, Maier LA, et al. Sensitivity and D. Chronic beryllium. An official American Thoracic Society statement: diagnosis and management of beryllium sensitivity and chronic beryllium disease. Am J Respir Crit Care Med. 2014;190:e34–59.

Churg A. The diagnosis of asbestosis. Hum Pathol. 1989;20:979.

Craighead JE, Abraham JL, Churg A, Gree FH, Kleinerman J, Pratt PC, et al. The pathology of asbestos-associated diseases of the lungs and pleural cavities: diagnostic criteria and proposed grading schema. Report of the pneumoconiosis Committee of the College of American pathologists and the National Institute for Occupational Safety and Health. Arch Pathol Lab Med. 1982;106:544–96.

Davis JM, Chapman J, Collings P, Douglas AN, Fernie J, Lamb D, Ruckley VA. Variations in the histological patterns of the lesions of coal workers' pneumoconiosis in Britain and their relationship to lung dust content. Am Rev Respir Dis. 1983;128:118–24.

De Vuyst P, Dumortier P, Rickaert F, Van de Weyer R, Lenclud C, Yernault JC. Occupational lung fibrosis in an aluminium polisher. Eur J Respir Dis. 1986;68:131–40.

Honma K, Abraham JL, Chiyotani K, De Vuyst P, Dumortier P, Gibbs AR, et al. Proposed criteria for mixed-dust pneumoconiosis: definition, descriptions, and guidelines for pathologic diagnosis and clinical correlation. Hum Pathol. 2004;35:1515–23.

Jederlinic PJ, Abraham JL, Churg A, Himmelstein JS, Epler GR, Gaensler EA. Pulmonary fibrosis in aluminum oxide workers. Investigation of nine workers, with pathologic examination and microanalysis in three of them. Am Rev Respir Dis. 1990;142:1179–84.

Jones RN. The diagnosis of asbestosis. Am Rev Respir Dis. 1991;144(3 Pt 1):477–8.

Khoor A, Roden AC, Colby TV, Roggli VL, Elrefaei M, Alvarez F, et al. Giant cell interstitial pneumonia in patients without hard metal exposure: analysis of 3 cases and review of the literature. Hum Pathol. 2016;50:176–82.