The word asbestos is a collective term that refers to a family of fibrous hydrous silicate minerals, including a serpentine (chrysotile) and amphiboles (crocidolite, amosite, tremolite). Because of its heat resistance and insulating properties, asbestos has been extensively used in most industrialized countries. The health effects of asbestos were described early in the 20th century, but it was only in the last few decades of this century that an intense medical and political debate was started, resulting in the ban of the use of these minerals in many countries and increasing restrictions in others. Even if the use of asbestos has dramatically decreased, leading to a virtual disappearance of severe cases of asbestosis (lung fibrosis), pleural manifestations are and will still frequently be diagnosed in the next decades. This is due to the very long latency between asbestos exposure and its manifestations and to the extensive use of thin-section computed tomography (CT) in the surveillance of exposed workers. In addition, malignant and non-malignant pleural diseases can be induced with low cumulative exposures, and many workers are still exposed to asbestos materials remaining in buildings.

The pleura, principally the parietal pleura, is a main target for asbestos fibers and is involved far more often than the lung parenchyma, despite the fact that the lung is the first organ reached by inhaled fibers. The precise mechanisms of translocation of fibers toward the pleura and the physiopathology of pleural fiber-induced diseases are still not well understood.

The non-malignant forms of asbestos-related pleural disorders include pleural plaques, benign pleural effusion, and diffuse pleural thickening. Although circumscribed pleural plaques and diffuse pleural thickening are considered under this broad category of asbestos-related pleural fibrosis, these processes are distinct entities that are likely to involve different pathogenic mechanisms and have different clinical consequences (Schwartz 1991).

8.1 Pleural Plaques

Pleural plaques are the most common thoracic lesions found in persons exposed to asbestos (Hillerdal and Lindgren 1980). These lesions predominantly involve the parietal pleura and are not associated with adhesions between the visceral and parietal pleural layers, unless there has been a complicating pleurisy or a surgical exploration (Schwartz 1991). Pleural plaques are most commonly found on the posterior wall of the lower half of the pleural spaces, laterally on the costal pleura, over the vertebrae, on the mediastinum, on the central part of the diaphragm,
and, more rarely, close to the costochondral junctions on the anterior wall (Roberts 1971). They are almost never located at the apices of the pleural cavities or in the costophrenic angles. Plaques also can be found on the pericardium. Pleural plaques are most often multiple and bilateral but unilateral asbestos-related plaques have been reported in up to 25–35% of asbestos-exposed patients with plaques (Fisher 1985; Gevenois et al. 1998; Neri et al. 1994). They have also been described to occur on the visceral pleura, within the interlobar fissures (Rockoff 1987; Sargent et al. 1981; Solomon et al. 1979), and exceptional cases of calcification within the fissures have been reported (Sargent et al. 1981; Rockoff 1987; Remy-Jardin et al. 2004).

8.1.1 Histopathology

Macroscopically, pleural plaques are discrete, raised, and irregularly shaped areas (Roberts 1971). They vary in size from a few millimeters to 10 cm. The thinner plaques are only slightly raised above the pleural surface, while the thicker plaques are either smooth or show a fine or coarse nodularity. Most of the plaques have a leathery consistency, but as pleural plaques tend to calcify with time, more heavily calcified plaques are brittle and can be fractured (Roberts 1971).

Microscopically, the pleural plaques consist of undulating dense fibrous bands of avascular collagen, lying parallel to the surface (Roberts 1971). They are relatively acellular with only occasional spindle-shaped fibroblasts, do not contain histologically visible asbestos bodies (Meurman 1968; Roberts 1971), and are covered with a normal mesothelium (Gefter and Conant 1988).

8.1.2 Physiopathology

The mechanism of the induction of pleural plaques is unknown, but they are so frequent that they probably represent the “normal” tissular reaction to the abnormal presence of fibers in the lung or in the pleura. Indeed, in animal studies, the development of plaques requires a normal immune response, whereas less well-organized pleural reactions occur when the immunity is modified by cytotoxic agents (Sahn and Antony 1984).

In contrast with the fact that pleural plaques are specifically associated with asbestos exposure, mineralogical studies have rarely detected such fibers in the parietal pleura. Boutin et al. (1996) hypothesized that the distribution of asbestos fibers in the pleura is heterogeneous and that these are concentrated in certain areas, probably derived from Wang’s pores and/or milky spots present at the surface of the parietal pleura. These structures are connected with the subpleural lymphatic network and are involved in the physiological clearance mechanisms of the pleural space (Kanazawa et al. 1979). In humans, milky spots are almost invisible on healthy pleura. However, direct examination of the pleura using thoracoscopy or autopsy frequently visualizes foci of anthracosis near lymphatic vessels of the parietal pleura (Boutin et al. 1991). They are found in more than 90% of urban dwellers at autopsy (Mitchev et al. 2002). They contain macrophages and lymphocytes. The pigmentation of these “black spots” is due to accumulation of inhaled exogenous dusts (coal, soot), and they are mainly located in the posterior and lower part of the costovertebral gutter and on the diaphragm (Mitchev et al. 2002). Most importantly, it has been shown that asbestos fibers, including long carcinogenic amphibole fibers, are heterogeneously distributed in the parietal pleural and that they concentrate in these black spots, whereas few or no fibers are detected in macroscopically normal pleural areas. The accumulation of fibers in these black spots make them possible starting points for asbestos-induced lesions of the parietal pleura (Fig. 8.1). The correspondence between the number, type, and dimensions of fibers in black spots and in the respective lungs is consistent with a mechanism of direct migration of fibers from the alveoli into the pleural cavity through the

![Fig.8.1. Macroscopic aspect of the parietal pleura of a past heating worker exposed to asbestos, coal, and soot dusts (autopsy specimen). Pleural plaques are visible in close contact with foci of anthracosis (“black spots”)](image-url)