Table 8-4 Spectrum of Inflammatory Responses to Infection

Type of Response	Pathogenesis	Examples
Suppurative (Purulent) Infection	Increased vascular permeability Leukocyte infiltration (neutrophils) Chemoattractants from bacteria Formation of "pus"	Staphylococcal pneumonia Tissue abscesses
Mononuclear and granulomatous inflammation	Mononuclear cell infiltrates (monocytes, macrophages, plasma cells, lymphocytes) Cell-mediated immune response to pathogens ("persistent antigen") Formation of granulomata	Syphilis Tuberculosis
Cytopathic-cytoproliferative reactions	Viral transformation of cells Necrosis or proliferation (including multinucleation) Linked to neoplasia	Human Papilloma Virus Herpesvirus
Tissue necrosis	Toxin or lysis mediated destruction Lack of inflammatory cells Rapidly progressive processes	*Clostridium perfringens* Hepatitis B
Chronic inflammation/scarring	Repetitive injury leads to fibrosis Loss of normal parenchyma	Chronic hepatitis (cirrhosis)
No reaction	Severe immune compromise	*Mycobacterium avium* in untreated AIDS (T-cell deficiency) Mucormycosis in bone marrow transplant patients (neutropenia)

Therefore, many pathogens produce similar reaction patterns, and few features are unique or pathognomonic for a particular microorganism. Moreover, sometimes the nature of the interaction between the microorganism and the host determines the histologic features of the inflammatory response. Thus, pyogenic bacteria, which normally evoke vigorous leukocyte responses, may cause rapid tissue necrosis with little leukocyte exudation in a profoundly neutropenic host. Similarly, in a normal patient, *M. tuberculosis* causes well-formed granulomas with few mycobacteria present, whereas in an AIDS patient the same mycobacteria multiply profusely in macrophages, which fail to coalesce into granulomas, which are summarized in Table 8-4 and described below.

There are five major histologic patterns of tissue reaction in infections.

Suppurative (Purulent) Inflammation

This pattern is characterized by increased vascular permeability and leukocytic infiltration, predominantly of neutrophils (Fig. 8-4). The neutrophils are attracted to the site of infection by release of chemoattractants from the "pyogenic" (pus-forming) bacteria that evoke this response, mostly extracellular gram-positive cocci and gram-negative rods. Masses of dying and dead neutrophils and liquefactive necrosis of the tissue form pus. The sizes of purulent lesions range from tiny microabscesses formed in multiple organs during bacterial sepsis secondary to a colonized heart valve, to diffuse involvement of entire lobes of the lung in pneumonia. How destructive the lesions are depends on their location and the organism involved. For example, pneumococci usually spare alveolar walls and cause lobar pneumonia that resolves completely, whereas staphylococci and *Klebsiella* species destroy alveolar walls and form abscesses that heal with scar formation. Bacterial pharyngitis resolves without sequelae, whereas untreated acute bacterial inflammation of a joint can destroy the joint in a few days.

Mononuclear and Granulomatous Inflammation

Diffuse, predominantly mononuclear, interstitial infiltrates are a common feature of all chronic inflammatory processes, but when they develop acutely, they often are a response to viruses, intracellular bacteria, or intracellular parasites. In addition, spirochetes and helminths provoke chronic inflammatory responses. Which mononuclear cell predominates within the inflammatory lesion depends on the host immune response to the organism. For example, plasma cells are abundant in the primary and secondary lesions of syphilis (Fig. 8-5), whereas lymphocytes predominate in HBV infection or viral infections of the brain. The presence of these lymphocytes reflects cell-mediated immune responses against the pathogen or pathogen-infected cells. At the other extreme, macrophages may become filled with organisms, as occurs in *M. avium-intracellulare* infections in AIDS patients, who cannot mount an effective immune response to the organisms. *Granulomatous inflammation* is a distinctive form of

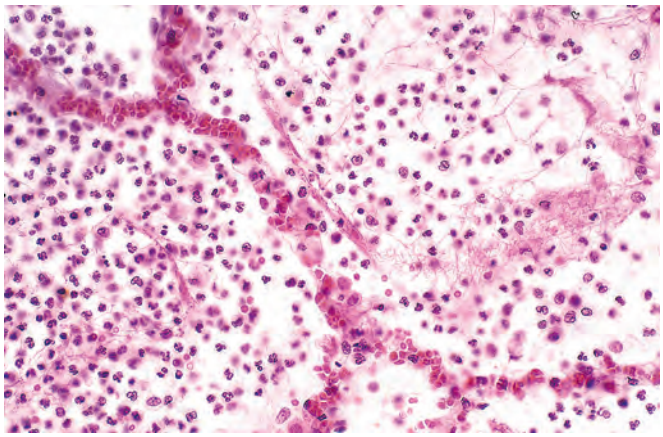

Figure 8-4 Suppurative (purulent) infection. Pneumococcal pneumonia with extensive neutrophilic infiltrate.