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Pulmonary Angiitis and Granulomatosis: Necrotizing Sarcoid Granulomatosis and Churg-Strauss Syndrome

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NECROTIZING SARCOID GRANULOMATOSIS

Necrotizing sarcoid granulomatosis (NSG) is a form of angiitis and granulomatosis that predominately involves the lung and is characterized by sarcoidlike granulomas, necrosis of lung tissue, and vasculitis. The disease pursues a fundamentally benign course and is responsive to corticosteroids; only exceptionally is there extrathoracic involvement. Of paramount importance is its differentiation from Wegener granulomatosis (WG), granulomatous infections, and sarcoidosis. NSG is relatively uncommon, with around 100 cases published since the first description of the disease by Liebow, in 1973.[1–11]

Clinical Manifestations

At presentation, the mean age is 50 years, with a broad range from 11 to 75 years. Rare pediatric cases have been reported.[6,7] Presenting symptoms include cough (38%), fever (28%), chest pain (34%), dyspnea (22%), and weight loss (16%). Up to 25% of patients with NSG are asymptomatic.[3–5,9,10,12] Women outnumber men in a ratio of 2.2 : 1.

Most patients have multiple bilateral nodules or nodular infiltrates on chest radiographs; however, unilateral nodules or ill-defined infiltrates may also be seen.[1,8–11] The lesions range in size from a few millimeters to 5 cm in diameter.[9,10] The lower lobes are most often affected.[2] Cavitation and pleural effusions may occur.[8] Enlarged hilar lymph nodes are uncommon in most series,[1,8,11] but Churg observed them in 65% of cases.[2] In a small percentage of patients, manifestations outside the lung may occur,[2] with ocular,[2,7,8] spinal cord,[6,7] and hypothalamic[2] lesions described.

Pathologic Manifestations

The gross pathology varies depending on whether the pulmonary lesions are multiple or solitary, bilateral or unilateral, nodular or ill-defined, and cavitating or solid.[2,8] The primary histologic features consist of granulomatous pneumonitis, necrosis, and vasculitis. The granulomas are sarcoidlike and are composed of nodular aggregates of epithelioid histiocytes and giant cells (Fig. 69-1*A*, *B*). They often form confluent masses and are frequently distributed along lymphatic routes and around bronchioles and blood vessels, as in sarcoidosis.[1,6,8,9] The size of the necrotic areas may vary from small foci to large regions.[8] The inflammatory vasculitic lesions of NSG may consist of giant cells, necrotizing granulomas (Fig. 69-2), and lymphocytes (Fig. 69-3). Both pulmonary arteries and veins are affected.

Clinical Course

Patients with NSG have an excellent prognosis.[5,6] Localized pulmonary lesions may be surgically resected, but in those cases in which there is residual lung disease, corticosteroids are usually effective therapy.[1,8,9,11] In the minority of cases, persistent or

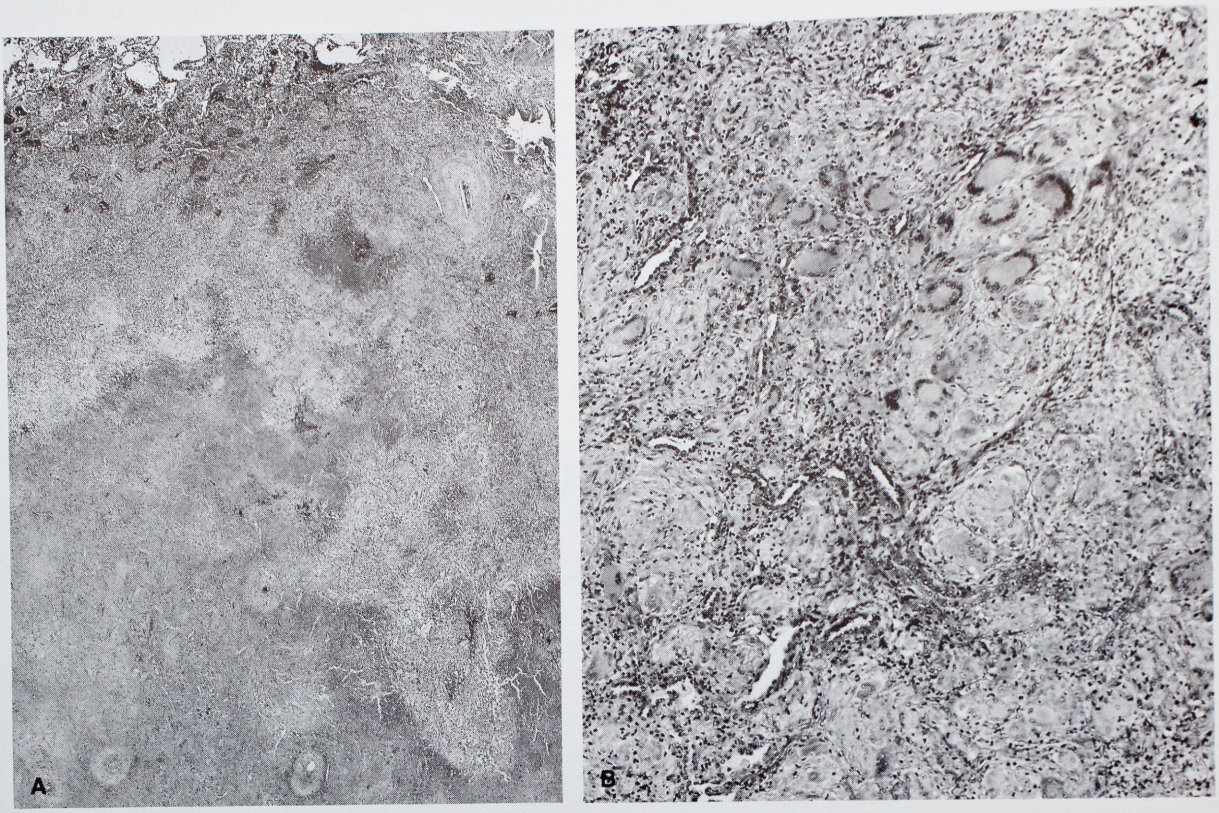

FIGURE 69-1. Necrotizing sarcoid granulomatosis. (**A**) The nodular inflammatory mass shows sharply circumscribed areas of necrosis (*bottom*). (**B**) Higher-power magnification shows that the confluent granulomas consist of numerous giant cells and epithelioid histiocytes at the periphery of the nodule shown in **A**. (H & E stain; low magnification.)

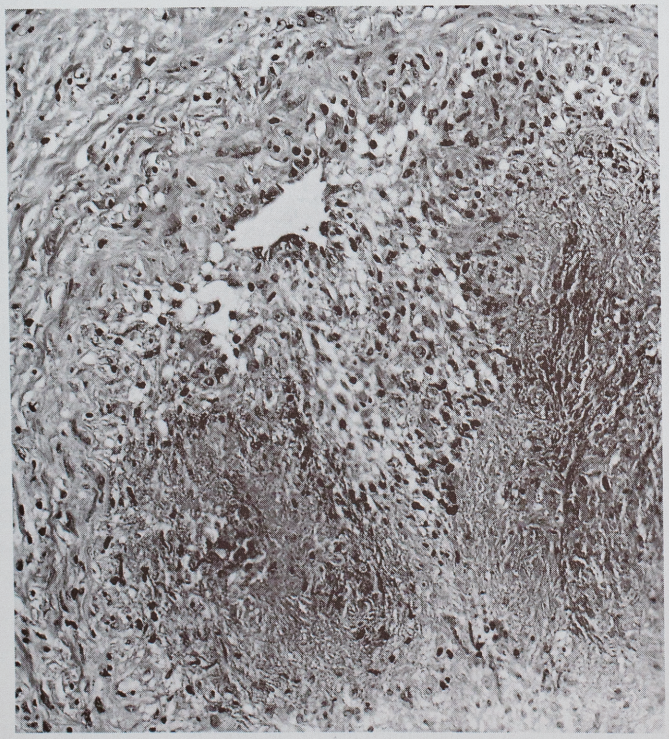

FIGURE 69-2. Necrotizing sarcoid granulomatosis vasculitis. The wall of this large blood vessel is extensively necrotic with narrowing of the lumen. (H & E stain; intermediate magnification.)

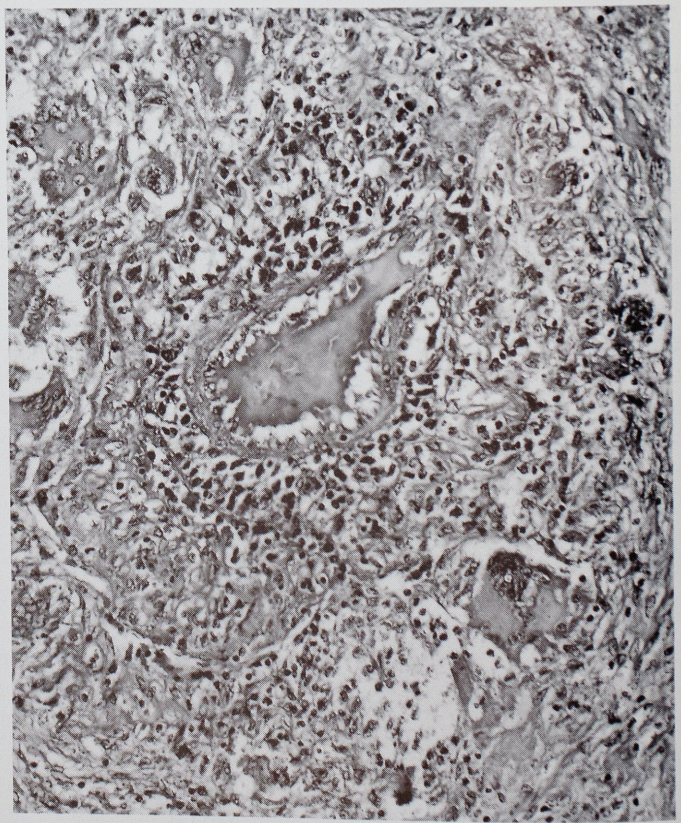

FIGURE 69-3. Necrotizing sarcoid granulomatosis vasculitis. The wall of this blood vessel has an extensive lymphocytic infiltrate; the surrounding tissue also shows scattered, ill-formed granulomas. (H & E stain; intermediate magnification.)

recurrent disease may occur, but there is reluctance to use cytotoxic agents because the only published disease-related deaths in these patients have been due to opportunistic infections resulting from such treatment.[1–3,8,9,11]

Differential Diagnosis

NSG must be differentiated from WG, infection, and classical sarcoidosis. WG is characterized by glomerulonephritis, sinusitis, and elevated tiers of antineutrophil cytoplasmic antibodies (ANCA), which are not features of NSG. In addition, sarcoidlike granulomas are very rare in WG (see Chap. 68).

Necrotizing granulomatous infection is the most important diagnosis to be excluded when considering NSG.[13,14] Exclusion of infection can be accomplished with a combination of cultures or special stains for fungal and mycobacterial organisms. Cultures may be positive even if special stains are negative.[9]

The relationship of NSG to classical sarcoidosis is controversial.[2,8,11] In classical sarcoid, nodular lesions have been described in less than 5% of patients.[15,16] In NSG, the disease tends to predominantly affect the lung. Enlargement of hilar lymph nodes can occur in both NSG and classical sarcoid, although it is more common in the latter.[2] Pathologic similarities also exist, because open lung biopsy specimens of classical sarcoidosis can show small necrotic foci in up to one third of cases and granulomatous angiitis in up to two thirds of cases.[17,18] In addition, both disorders are steroid responsive and have an excellent prognosis.

CHURG-STRAUSS SYNDROME

Churg-Strauss syndrome is characterized by asthma, peripheral blood eosinophilia, and systemic vasculitis.[19–26] Many of these cases had previously been classified as polyarteritis nodosa with asthma.[19,22,26–30]

Clinical Manifestations

Churg-Strauss syndrome typically evolves through a prodromal phase, a vasculitic phase, and a postvasculitic phase,[19,22,24] although these phases do not always occur in a specific order.[24] During the prodrome, patients typically have asthma, allergic rhinitis, blood eosinophilia, and eosinophilic pneumonia or gastroenteritis.[19–22,24,31] The duration of each phase varies greatly, and symptoms may wax and wane. During the vasculitic phase, patients have systemic vasculitis, and it is in this phase that the diagnosis is most easily established.[19] Hypoallergen injections[32–34] and exposure to inhaled antigens such as *Actinomycetes thermophilous*[35] have been reported to precipitate the vasculitic phase. Asthma and allergic rhinitis may persist in the postvasculitic phase, but complications of neuropathy and hypertension may cause difficulty.[19,22]

Other pulmonary abnormalities of Churg-Strauss syndrome include abnormalities in the chest radiograph and pleural effusions.[19,22] Rarely, pulmonary hemorrhage has been reported.[36] A variety of head and neck manifestations can occur, including nasal obstruction or rhinorrhea, nasal polyps, septal perforations,[37] and conjunctival involvement.[38,39]

Other organs that are often affected include the heart,[22,24,26,40–42] gastrointestinal tract, kidney, skin, and peripheral nervous system. Cardiac failure is the most common cardiac manifestation, followed by pericarditis and hypertension. Restrictive cardiomyopathy, acute myocardial infarction, and arrhythmias are less common. Abdominal pain is the most frequent gastrointestinal finding, followed by diarrhea and gastrointestinal bleeding.[19,22,24,43]

Renal involvement is seen in less than 50% of patients in most series.[22,24] Renal disease is usually mild, but renal failure may occur.[19,24,44] Involvement of the ureters,[19,45] prostate,[46,47] and penis can also occur.[19,22,44] Skin involvement occurs in the form of nodules, purpura, and erythremia or urticaria.[48,49] Up to one half of patients may have arthritis or arthralgias.[19,22] Up to two thirds of patients may have mononeuritis multiplex, and about one fourth of patients may have central nervous system manifestations.[19,22]

Blood eosinophilia is the key laboratory feature of the vasculitic phase. Erythrocyte sedimentation rate and C-reactive protein levels may also be elevated.[19,22,24] Antimyeloperoxidase antibodies (*i.e.*, P-ANCA) are often found in patients with Churg-Strauss syndrome[50,51]; however, antiproteinase-3 antibodies (*i.e.*, C-ANCA) can also be encountered.[52] The majority of patients have increased serum IgE levels.[19,22,24,53] Rarely, patients without asthma, but with a history of allergic disease, appear to have the Churg-Strauss syndrome.[54–56]

Pathologic Manifestations

Vasculitis, tissue eosinophilia, and extravascular granulomas are the characteristic pathologic features of Churg-Strauss syndrome, but it is uncommon to encounter biopsy specimens with all three features. The lungs in patients with Churg-Strauss syndrome may show vasculitis, asthmatic bronchitis (Fig. 69-4), eosinophilic pneumonia, and extravascular granulomas.[19,26,57] Extravascular granulomas consist of a central zone of necrotic eosinophils surrounded by palisading histiocytes and giant cells (Fig. 69-5*A, B*). These granulomas can become calcified and fibrotic.

The vasculitis predominantly affects medium-sized muscular arteries or veins. Vessel walls are infiltrated by lymphocytes, plasma cells, eosinophils, epithelioid cells, and giant cells (Fig. 69-6). Fibrinoid necrosis is present in a minority of cases (Fig. 69-7). Nonspecific cicatricial changes are relatively common. Inflammatory changes may lead to vascular obstruction or thromboses, which may result in infarcts. Aneurysms are uncommonly seen.

Cardiac involvement is common and may consist of pericarditis, eosinophilic and granulomatous myocarditis, myocardial fibrosis, coronary arteritis, myocardial infarction, and endomyocardial fibrosis.[19,26,40,41,56,58] Eosinophilic infiltrates, vasculitis, and allergic granulomas may also affect other organs, such as the liver, kidney, gastrointestinal tract, skin, and nose.

Diagnostic Criteria

There has been a shift in the accepted criteria for the diagnosis of Churg-Strauss syndrome,[26] from a pathologic to a clinical approach. The original criteria proposed by Churg and Strauss emphasized pathologic criteria. However, more recent studies have shown that it is uncommon to encounter all of the classic histopathologic features in tissue biopsy specimens from patients with this syndrome.[22,24] This is due to the transient nature of the tissue infiltrates and the evolution of the disease through different phases. In addition, it has become recognized that both drugs[59] and parasitic infections[60,61] can induce pathologic changes similar to those seen in Churg-Strauss syndrome; thus, the pathology is not

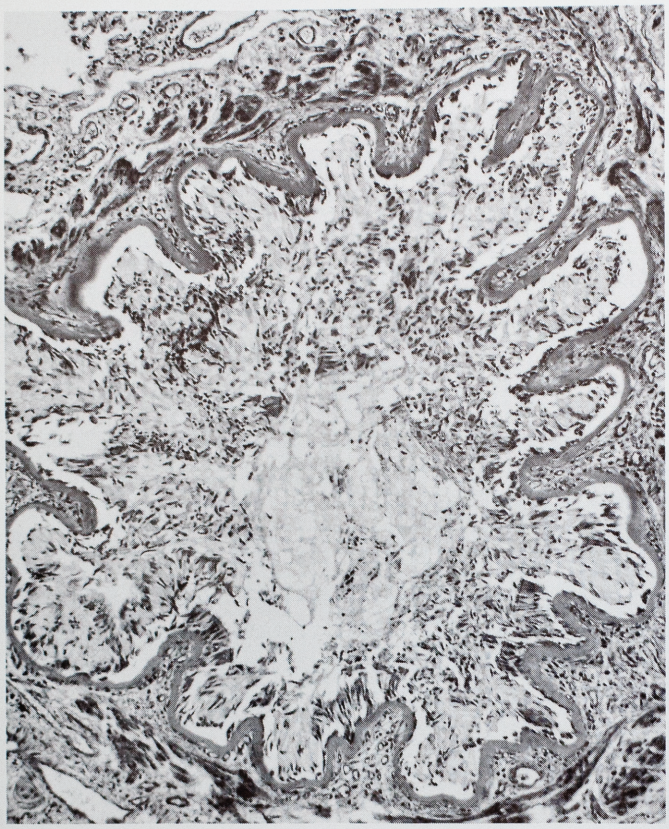

FIGURE 69-4. Churg-Strauss syndrome, asthmatic bronchitis. This bronchus shows mucus plugging with desquamated bronchial epithelium, prominent hyalinized and thickened subepithelial basement membrane, and hypertrophic contracted smooth muscle. (H & E stain; low magnification.)

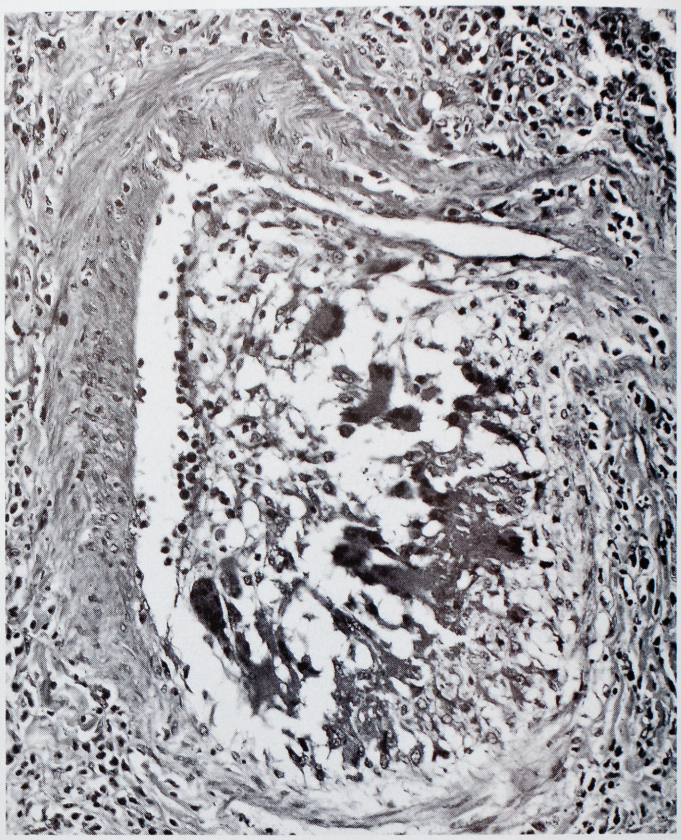

FIGURE 69-6. Churg-Strauss syndrome, vasculitis. This vessel shows intravascular granuloma with eosinophilic debris and numerous giant cells. (H & E stain; intermediate magnification.)

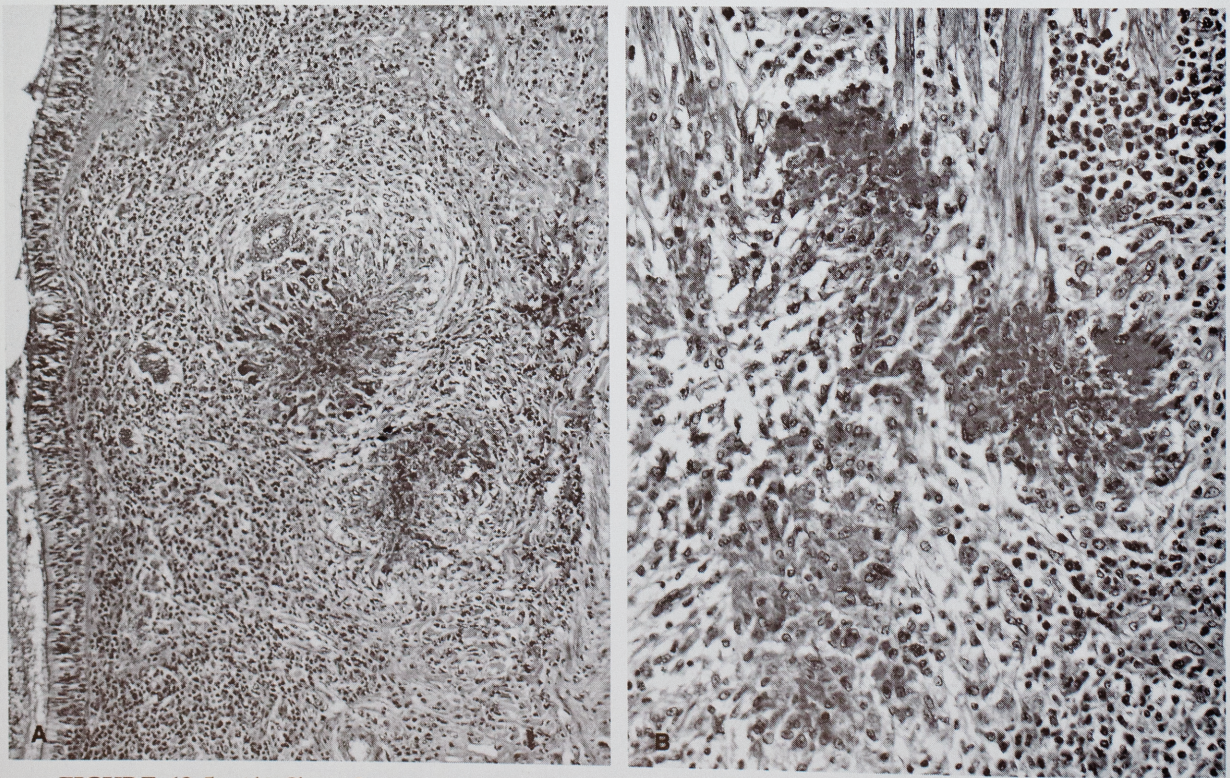

FIGURE 69-5. (**A**) Churg-Strauss syndrome, allergic granuloma. The wall of this bronchiole shows an allergic granuloma consisting of central necrosis with eosinophilic debris. (H & E stain; low magnification.) (**B**) Higher magnification of eosinophilic debris surrounded by epithelioid histiocytes and multinucleate giant cells. (H & E stain; intermediate magnification.)

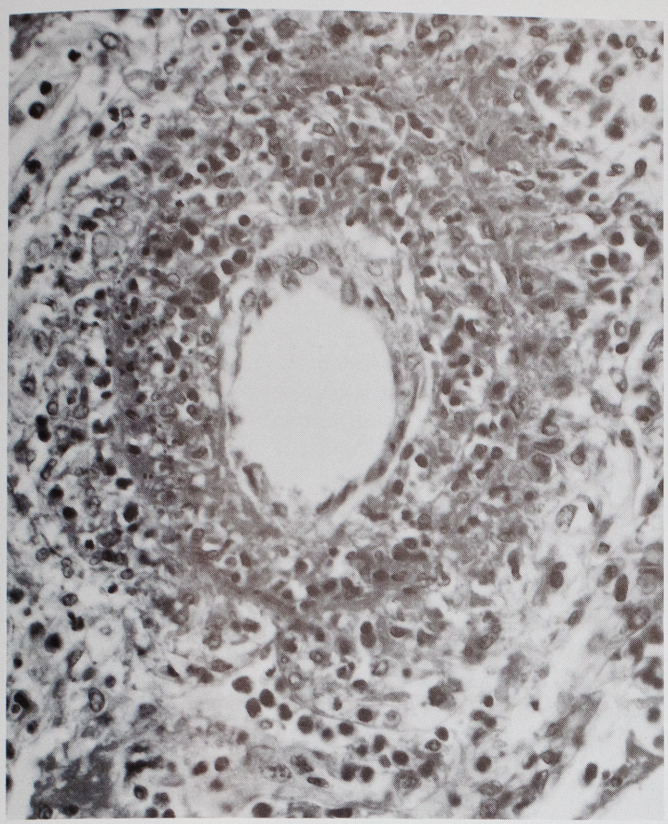

FIGURE 69-7. Churg-Strauss syndrome, vasculitis. This vessel shows fibrinoid necrosis and a dense inflammatory infiltrate that is rich in eosinophils. No granulomas are seen. (H & E stain; intermediate magnification.)

diagnostically specific. This led Lanham and colleagues to propose primarily clinical criteria for the diagnosis of Churg-Strauss syndrome.[22] These include asthma, peripheral blood eosinophilia (*i.e.*, $>1.5 \times 10^9$/L), and systemic vasculitis involving two or more extrapulmonary organs.

In 1990, two sets of diagnostic criteria for Churg-Strauss syndrome were proposed by a subcommittee of the American College of Rheumatology: a traditional format classification and a classification tree.[62,63] According to the traditional format, the presence of four of six of the following criteria results in an accurate diagnosis with a sensitivity of 85% and a specificity of 99.7%.[62] The following are the six proposed criteria:

1. Asthma
2. Eosinophils greater than 10% of the white blood cell differential count
3. Mononeuropathy, including multiplex, or polyneuropathy
4. Nonfixed radiographic pulmonary infiltrates
5. Paranasal sinus abnormalities
6. A biopsy specimen containing a blood vessel with extravascular eosinophils.[62]

The major criteria used in the classification tree include asthma, eosinophilia greater than 10%, and a history of allergy.[62] The latter approach includes systemic vasculitis patients who have a peripheral eosinophilia greater than 10% and a history of allergy other than drug sensitivity.[62]

Differential Diagnosis

Churg-Strauss syndrome must be differentiated from WG,[14] eosinophilic pneumonia,[64] allergic bronchopulmonary fungal disease (ABPFD),[65] parasitic infection,[60] and drug-induced vasculitis.[59,66] Compared with patients with WG, those with Churg-Strauss syndrome tend to have less severe renal disease and less destructive upper airway disease. In addition, in WG, peripheral blood eosinophilia greater than 5% occurs in only 12% of patients. When it occurs it is rarely marked, and the frequency of asthma is not increased over that of the general population. Although marked eosinophilia can occur in 6% of lung biopsy specimens from WG patients, the necrotizing granulomas show necrotic neutrophils, whereas those of Churg-Strauss syndrome show necrotic eosinophils.[14,67]

Systemic vasculitis distinguishes Churg-Strauss syndrome from eosinophilic pneumonia and ABPFD. Asthma and a systemic illness similar to Churg-Strauss syndrome can be caused by *Strongyloides stercoralis*[61] and *Toxocara canis*,[60] and similar pathologic changes have been reported in visceral larva migrans.[60] In addition, carbamazepine may cause a systemic eosinophilic and granulomatous vasculitis.[59]

Therapy and Prognosis

Corticosteroids are frequently effective in the treatment of Churg-Strauss syndrome. Traditionally, cyclophosphamide has been used in cases not responsive to steroids, but some advocate the use of immunosuppressive drugs from the outset because a substantial percentage of patients may develop end-stage organ disease.[19]

Cardiac complications (*i.e.*, congestive heart failure or myocardial infarction) are the most common causes of death, followed by renal failure, cerebral hemorrhage, gastrointestinal perforation or hemorrhage, status asthmaticus, and respiratory failure.[19]

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