

# Letters to the Editor

## CORRESPONDENCE RE: STRICTLAND-MARMOL LB, FESSLER RG, ROJANI AM. NECROTIZING SARCOID GRANULOMATOSIS MIMICKING AN INTRACRANIAL NEOPLASM: CLINICOPATHOLOGIC FEATURES AND REVIEW OF THE LITERATURE. MOD PATHOL 2000;13:909-3.

**To the Editor:** The temporal lobe mass described in this paper is hard to classify, but it is difficult to justify a diagnosis of necrotizing sarcoid granulomatosis. This diagnosis requires the exclusion of infection and the identification within the mass of *all* three of the following lesions: prominent, well-formed, nonnecrotizing granulomas; significant parenchymal necrosis; and granulomatous vasculitis that cannot be attributed to direct extension from the parenchymal granulomas (1–4). The mass described in this paper includes granulomas and necrosis but no vasculitis. A diagnosis of necrotizing sarcoid granulomatosis is not warranted because only two of the three diagnostic criteria are present. Mycobacterial and fungal infection are unlikely because the special stains and cultures were negative and the patient responded to steroid therapy. In view of the past history of sarcoidosis, it is likely that the temporal lobe mass represents *nodular sarcoidosis* (5). Small foci of necrosis such as are illustrated in Figure 2 of the paper can be encountered occasionally in the centers of sarcoid granulomas (6).

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**In reply:** We agree with Dr. Kay that the diagnosis of necrotizing sarcoid granulomatosis requires three elements: granulomas, necrosis, and vasculitis (1). The specific features of these requirements, however, warrant some clarification. The granulomas in this case are well defined, both necrotizing and non-

necrotizing. The majority of granulomas (>60%) have some component of necrosis. Other reports have described necrosis in 21% of granulomas in cases of sarcoidosis that showed any necrosis (2). Measurements of the area of necrosis in our case, performed by image analysis, reveals >40% of involved areas to be necrotic. This is significantly greater than reports describing necrosis in typical or nodular sarcoidosis, where necrosis is very limited and in some instances consists only of scattered necrotic cells. The definition of vascular involvement in necrotizing sarcoid granulomatosis is not restricted to the identification of granulomas within the vessel wall. Three vascular lesions have been described in necrotizing sarcoid granulomatosis: destruction of a portion of the wall of a large vessel by intraluminal granulomas; compression of small vessels by surrounding granuloma and often occlusion of their lumina by intimal fibrosis; and destruction of the wall of large and small vessels by an infiltrate of lymphocytes, plasma cells, and histiocytes (1). In this case, we did not find transmural or intramural granulomas; however, in this limited resection, elastic stains revealed only three vessels with defined elastic lamina. Compression of vessels with occlusion of lumina by intimal fibrosis was readily evident, as was involvement of the vessel wall with inflammatory infiltrate. Thus, we feel that the designation of necrotizing sarcoid granulomatosis in this case is appropriate and correct.

The letter does, however, raise questions about the relationship between sarcoidosis, nodular sarcoidosis, and necrotizing sarcoid granulomatosis. The concept of necrotizing sarcoid granulomatosis simply being a variant of sarcoidosis (1) in a manner similar to the relationship between sarcoidosis and nodular sarcoidosis certainly has merit. Although there are clinical and histologic similarities between these entities, there remain significant differences. Hence, until we are in a position to better define their etiopathogenesis, it would seem appropriate and necessary to continue to consider necrotizing sarcoid granulomatosis as a separate nosological entity.

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