section. Although rare, Rosai-Dorfman disease can be confused with nodular scleritis and should be considered in patients with epibulbar masses and uveitis.

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Granulomatous Choroiditis in Wegener Granulomatosis

Wegener granulomatosis (WG) is characterized classically as the triad of necrotizing granulomatous lesions of the upper and lower respiratory tract, focal segmental glomerulonephritis, and necrotizing vasculitis of small arteries and veins.1 Ophthalmological disease is the manifesting feature of WG in 8% to 16% of patients1 but develops in an estimated 50% to 60% of patients.2 Orbital disease is the most common ophthalmological manifestation of WG, uveal involvement is uncommon,2 and granulomatous sclerouveitis is rare.1-5 I describe the first histological documentation, to my knowledge, of granulomatous choroiditis in WG in the absence of scleritis.

Report of a Case. A 71-year-old man developed protracted nausea, a 6.75-kg weight loss, acute renal failure, and pulmonary hemorrhage. He had positive results on a perinuclear antineutrophil cytoplasmic autoantibody assay and an antitymoperoxidase enzyme-linked immunosorbent assay (level=130.8 U; positive >20.0 U) and negative results on an antiproteinase 3 en-

zyme-linked immunosorbent assay (level=3.9 U; positive >20.0 U). Although he did not have the typical pattern of antineutrophil cytoplasmic antibodies with cytoplasmic staining, his findings were considered most compatible with WG. His renal failure did not resolve with hemodialysis, high-dose methylprednisolone sodium succinate, cyclophosphamide, and plasmapheresis. He had progressively decreasing strength, mental status, and ability to tolerate tube feeding and died approximately 5 months after his initial symptom of nausea developed. A complete autopsy confirmed the diagnosis of WG with necrotizing granulomatous and fibrinous vasculitis with neutrophils and karyorrhectic debris involving the kidneys, testes, appendix, liver, spleen, lungs, pancreas, lymph nodes, small and large intestines, trachea, aorta, pericardium, myocardium, and both orbits (listed in order of decreasing histological severity).

The posterior choroid of both eyes had many foci of granulomatous inflammation similar to those in other organs with mostly epithelioid cells accompanied by lymphocytes and a few multinucleated giant cells (Figure 1). In multiple areas, the choriocapillaris was infiltrated by inflammatory cells and the capillaries were stenotic or occluded by inflammatory cells (Figure 2). Rare minute foci of fibrinoid necrosis with occasional neutrophils and karyorrhectic debris were in the choroid just beneath the choriocapillaris (Figure 2). The choroidal vessels were surrounded by the dense inflammatory infiltrate, but only a rare artery appeared to have its wall infiltrated by lymphocytes without necrosis. Degeneration of the neurosensory retina and scleral inflammation were not seen. Microorganisms were not detected using histochemical stains.

Comment. Choroidal involvement in WG may manifest clinically as uveitis, choroidal folds, retinal epithelial pigmentary changes, choroidal arterial occlusion, or choriocapillaritis.1,2 Only 1 histological description of isolated choroidal involvement by WG exists, to my knowledge.6 The patient described by Cutler and Blatt6

![Image](https://example.com/image.jpg)

**Figure 1.** The posterior choroid of both eyes had many foci of granulomatous inflammation. A confluent area of granulomatous inflammation is to the left in the photomicrograph, while 2 smaller granulomas (asterisks) are to the right (hematoxylin-eosin). Scale bar indicates 100 µm.
Figure 2. The foci of granulomatous inflammation in the choroid contained mostly epithelioid cells accompanied by lymphocytes. The choriocapillaris (asterisk) is occluded by inflammatory cells. A small focus of fibrinoid necrosis has occasional neutrophils and karyorrhectic debris (arrows) (hematoxylin-eosin). Scale bar indicates 25 µm.

died 43 months after the onset of his illness, and there was a light but diffuse infiltrate of lymphocytes throughout the choroid; marked sclerosis of choroidal blood vessels with medial fibrosis and plump, prominent endothelial cells; prominent edema and fibrosis; areas of necrosis and hyperplasia of the retinal pigment epithelium; and degeneration of the overlying sensory retina.

In my patient, both eyes had many foci of granulomatous inflammation in the posterior choroid, rare minute foci of fibrinous necrosis in the choroid just beneath the choriocapillaris, and areas where the choriocapillaris was infiltrated by inflammatory cells. The inflammatory infiltrate in my patient resembled the granulomatous sclerouveitis reported in WG, which contains a mixture of T and B lymphocytes, macrophages, and enhanced expression of adhesion molecules and ligands. I postulate that the difference in the histological appearance of the choroid in my patient's eyes and that reported by Cutler and Blatt is due to the shorter duration of the WG in my patient and its stage of activity at the time of death. However, I cannot exclude the possibility that the difference reflects underlying variation in choroidal manifestation of WG.

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Methods. Baseline and longitudinal digitized optic nerve stereophotographs from 4 patients with progressive GON were taken with a Nidek 3Dx simultaneous stereo camera (Nidek Inc, Fremont, California) and imported into Matched-