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. Ophthalmological disease is the manifesting feature of WG in 8% to 16% of patients but develops in an estimated 50% to 60% of patients. Orbital disease is the most common ophthalmological manifestation of WG, uveal involvement is uncommon, and granulomatous sclerouveitis is rare. 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 anti-myeloperoxidase enzyme-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. In multiple areas, the choriocapillaris was infiltrated by inflammatory cells and the capillaries were stenotic or occluded by inflammatory cells (Figure 1). 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 choriocapillaris. Only 1 histological description of isolated choroidal involvement by WG exists, to my knowledge. The patient described by Cutler and Blatt...
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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**Online First**

**Detection of Progressive Glaucomatous Optic Neuropathy Using Automated Alternation Flicker With Stereophotography**

Examination of the optic nerve complex (optic nerve head, retinal nerve fiber layer [RNFL], and parapapillary region) is an integral part of the examination for glaucoma. Glaucomatous optic neuropathy (GON) can be diagnosed by characteristic changes in the neuroretinal rim, RNFL, or parapapillary region and often precedes visual field loss.³

The use of flicker chronoscopy for the longitudinal evaluation of glaucomatous change in serial optic nerve photographs was first described by Goldmann and Lotmar.² A new technique, automated alternation flicker (AAF), may facilitate the detection of progressive optic disc change.³ The original technique of alternation flicker involved manual alignment and alternation of serial disc photographs (eg, using 2 overlapping projectors to display sequential images onto a screen).³ Although time-consuming, this approach permitted discerning structural optic nerve changes over time. Automated alternation flicker, however, uses software that automatically aligns 2 images by identifying vascular intersections or other salient image features, superimposing the photos at a subpixel level after global transformations (eg, rotation and magnification), and alternating the images at a user-dictated frequency.

Automated alternation flicker has previously been used to identify changes between monoscopic images, a potential limitation of this technique. Evaluation of serial sets of optic disc stereophotographs has been suggested to be the best standard for monitoring glaucomatous change in both clinical and research settings. In particular, 3-dimensional visualization of the optic nerve complex can provide a clear view of neuroretinal rim tissue changes. Perhaps for this reason, interobserver agreement for optic nerve grading is higher with stereoscopic assessment, and stereoscopic photographs may permit greater sensitivity for and earlier detection of GON compared with monoscopic viewing.³ We describe the combined use of AAF with stereoscopic photography to detect structural progression in 3-dimensional viewing in 4 cases of GON: topographic change with blood vessel movement, neuroretinal rim loss, parapapillary atrophy progression, and disc hemorrhage.

**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-