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Phacoanaphylactic Endophthalmitis in an Owl

G. A. ANDERSON and N. BUYUKMIHCI

An adult screech owl (*Otus asio*) was brought to the Veterinary Medical Teaching Hospital in August 1981. The bird was in poor physical condition, dehydrated, and in shock. The left eye was enlarged and had a luxated lens. The owl would not open the right eye, but no reason for this was evident. Parenteral dexamethasone (4 mg/kg body weight) and oral fluids were administered. The bird continued to deteriorate and died the day of admission.

At necropsy, major gross findings were restricted to the left eye and lungs. The left pupil was constricted markedly. The central half of the cornea was opaque. Hypopyon was present, and the lens was luxated into the anterior chamber. The lungs were mottled with small, bright red patches, and the caudodorsal aspect of the right lung contained approximately 1 ml of free blood within and directly around it. There was marked atrophy of muscles throughout the carcass.

Histologic changes were limited to the left eye, lungs, and kidneys. The left eye had a severe endophthalmitis which centered around the lens. The entire circumference of the lens was disrupted and encompassed by a thick inner zone of polymorphonuclear leukocytes and an outer zone of polymorphonuclear leukocytes and mononuclear inflammatory cells. Many of the polymorphonuclear leukocytes were degenerated and intermingled with chromatin and fragmented lens fibers. These lens fibers varied from individual strands to dense, deeply eosinophilic morgagnian globules. Polymorphonuclear inflammatory cells in the outer zone were less numerous and were mixed with various numbers of macrophages, occasional giant cells, and dense proteinaceous material. Macrophages generally had a foamy appearance and sometimes contained periodic acid-Schiff-positive granules. Small clusters and single inflammatory cells were present in remaining portions of both the aqueous and vitreous cavities. The iris was encased within the inflammatory reaction around the lens. Uveal tract inflammation was characterized by many lymphocytes, plasmacytes, and a few polymorphonuclear leukocytes (figs. 1, 2). The trabecular meshwork and the stroma of the iris and ciliary body were infiltrated densely, whereas the pars plana of the ciliary body and choroid contained only modest numbers of leukocytes. In addition, a few inflammatory cells were

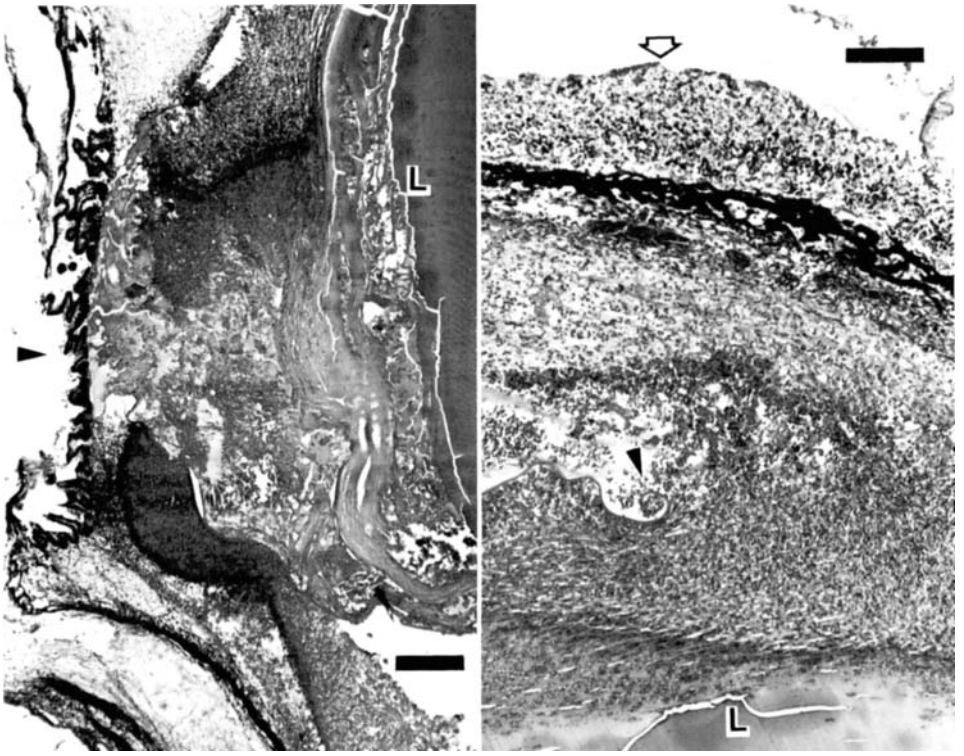

Fig. 1: Photomicrograph; lateral aspect of lens (L) with dense zone of inflammation around fragmented lens and within ciliary body (arrowhead). Bar = 300 μm.

Fig. 2: Photomicrograph; anterior aspect of lens (L) with invasion of polymorphonuclear leukocytes; lens capsule (arrowhead) is broken and distorted; iris (open arrow). Bar = 125 μm.

overlying and within the optic nerve fiber layer of the retina. The pecten was infiltrated densely by lymphocytes and plasmacytes. The remainder of the eye was unremarkable; there was no evidence of a penetrating wound and special stains demonstrated no fungi or bacteria. Additional histologic findings included focal coagulation necrosis with acute pulmonary hemorrhage and mild multifocal acute nephrosis.

Phacoanaphylactic endophthalmitis (phacoallergic endophthalmitis, phacogenic endophthalmitis, lens-induced uveitis) is a generalized inflammatory reaction which primarily involves the uveal tract but involves the lens and vitreous also [1, 8, 13]. Both antibody, particularly immunoglobulin G, and T-cell mediated hypersensitivity reactions can produce the inflammatory changes observed in affected eyes [4, 5, 7]. Since lens proteins are sequestered from immunocompetent cells during fetal life, these proteins have been regarded as classic examples of antigens which have escaped normal induction of immunological tolerance to "self" so that when these lenticular proteins are released into the systemic circulation, they act as antigens [10, 14]. Recent experiments in man and rabbits, however, have shown that lens proteins are common within the aqueous humor of both normal and cataractous eyes [9] and that autologous lens proteins are weak immunogens [8, 10]. Furthermore, experimental results suggested that since lenticular antigens induced varied responses in different rabbits, a central control mechanism involving immune-response and immune-associated genes may be important in the pathogenesis of phacoanaphylactic

endophthalmitis [6–8]. It appears that immunological tolerance to lens proteins can be present normally but can be terminated under certain, as yet undefined, circumstances [10]. Thus, phacoanaphylactic endophthalmitis is a complex immune-mediated disease of the eye that has a multifactorial pathogenesis.

Lens-induced ophthalmitis seldom is encountered in nonhuman animals; when lens material is extruded from the capsule, inflammation is not an invariable sequela [11]. The disease has not been recognized in birds [2, 3, 12]. The concept of phacoanaphylactic endophthalmitis is relatively well established in man [1] but certainly not in other animals. Although caution must be exercised when attempting to correlate strictly histologic features amongst various species, features observed in this owl indicate phacoanaphylactic endophthalmitis is an entity that also can occur in birds.

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