Macular Infarction Following Viperine Snake Bite

Macular infarction has been reported following toxic influences, for example, aminoglycoside toxicity. Venomous snake bites may result in neurologic or hemostatic dysfunction. Viperine (hemotoxic) snake bites may produce coagulopathy, which may result in several systemic complications. Ocular involvement is rare. Common ocular problems encountered after a snake bite are generally neurologic (ptosis, ophthalmoplegia, accommodation paralysis, and optic neuritis). Visual loss may result from direct inoculation of venom into the eye (globe necrosis, keratomalacia, and uveitis), from optic neuritis, or secondary to hemostatic abnormality (vitreous hemorrhage, cortical infarction, and central retinal artery occlusion).

Report of a Case. A 17-year-old girl was bitten by a viperine snake. She was admitted to a local hospital in an unconscious state and administered first aid, anti–snake venom serum, and supportive care. She regained consciousness 14 hours after the snake bite and 6 hours later reported loss of vision in her left eye. She came to us 5 days later. Visual acuity was recorded as 20/20 OD and no light perception OS. Ophthalmological examination disclosed unremarkable anterior segment and normal intraocular pressures in both eyes. Relative afferent pupillary defect was observed in the left eye. Fundus examination revealed optic disc hyperemia, splinter-shaped hemorrhages at the posterior pole, and a cherry-red spot at the center of the macula (Figure 1). Fluorescein angiography demonstrated normal arm-retina (10-second) and arteriovenous transit (11-second) times. Blocked choroidal fluorescence was observed in relation to the nerve fiber layer hemorrhages. The most striking feature on fluorescein angiography was pruning of the perifoveal capillaries. The silhouette of occluded macular capillaries was observed against the choroidal flush. Late-phase angiograms showed optic disc staining (Figure 2).

Systemic examination revealed no deficit. Laboratory investigations showed mild anemia (hemoglobin level, 10.4 g/dL [to convert to grams per liter, multiply by 10.0]), leukocytosis (13 800/µL [to convert to ×10⁹ per liter, multiply by 0.001]), and neutrophilia (74%). Results of renal function tests, abdominal ultrasonography, electrocardiography, echocardiography, and magnetic resonance imaging of the brain were normal. Dual antiplatelet therapy (aspirin, 75 mg/d, and clopidogrel, 75 mg/d), systemic antibiotics, and oral prednisone (40 mg/d tapered by 10

Figure 1. Fundus photograph showing a cherry-red spot at the macula and superficial retinal hemorrhages.

Figure 2. Early (A) and late phase (B) fluorescein angiograms demonstrate pruning of the perifoveal capillaries and optic disc staining, respectively.
mg/wk) were started. Three months later, the visual acuity remained no light perception. Optic disc pallor and gross attenuation of perifoveal vessels were noted. The macula showed pigment clumping and atrophy (Figure 3).

Comment. Snake venom is a complex heterogeneous composition of substances that predominantly affects the synapse (neurotoxic) or coagulation pathway (hemotoxic). Large doses can result in disseminated intravascular coagulopathy (DIC) and ischemic damage to vital organs. Toxic vasculitis has reportedly been caused by certain species of the Viperidae family. Hemorrhagins (complement-mediated toxic components of viperine venom) may induce severe vasospasm, endothelial damage, and increased vascular permeability. Vasospasm and/or DIC may result in vascular occlusion. Fibrin thrombi in the capillaries, perivascular hemorrhages, and necrosis, all observed in our patient, are features of DIC.

The likely cause of visual loss could be (1) ophthalmic artery occlusion with subsequent dislodgement of fibrin emboli into the end arteries at the posterior pole; or (2) retinal necrosis and macular infarction secondary to an aborted DIC process associated with toxic optic neuropathy (venom or ASV serum toxicity).

Ocular complications following a snake bite range from keratomalacia to vitreous hemorrhage, uveitis, optic neuritis, globe necrosis, and visual loss due to cortical infarction. We are unaware of any previous report in the literature of macular infarction following a viperine snake bite. Visual prognosis is poor despite medical treatment.

Jatinder Singh, MS, DNB
Preetam Singh, DOMS
Rajbir Singh, MS
Vipin Kumar Vig, MS

Correspondence: Dr Singh, Sardar Bahadur Dr Sohan Singh Eye Hospital, Katra Sher Singh, Chowk Farid, Amritsar-143006, Punjab, India (drjatinder@rediffmail.com).

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**Slowly Progressive Cancer-Associated Retinopathy**

Cancer-associated retinopathy (CAR) is caused by antiretinal antibodies cross-reacting with pathogens expressed by carcinoma cells. Cancer-associated retinopathy with antirecoverin antibody generally shows rapidly progressive visual deterioration. Autoimmune retinopathy (AIR) is characterized by antiretinal antibody–positive serum in the absence of systemic carcinoma but with slowly progressive visual deterioration, although the pathogenic mechanism is uncertain. We describe a patient with CAR with antirecoverin antibody who had slowly progressive visual deterioration resembling AIR.

Report of a Case. In 2004, we examined a 62-year-old woman with a 10-year history of progressive visual loss and night blindness. In 1994, Goldmann perimetry and electroretinography (ERG) at another hospital showed retinitis pigmentosa–like findings in the right eye and a normal appearance in the left. In 2000, ERG response showed further deterioration in the right eye and a normal response in the left. In 2002, the left eye also exhibited a visual field defect and ERG abnormality. The patient had no history of carcinoma or ocular trauma.

When examined in 2004, our patient’s visual acuities were 20/30 OD and 20/20 OS. Slitlamp examination demonstrated mild iridocyclitis and nuclear cataract bilaterally. Funduscopy examination results demonstrated optic disc pallor, retinal artery whitening, and retinal pigment epithelial atrophy with partial pigmentation in the midperipheral area across 360° (Figure 1A). Fluorescein angiography demonstrated a window defect corresponding to the retinal degeneration (Figure 1B) and late leakage from retinal capillary vessels. Goldmann perimeter demonstrated central and temporal islands in the right eye and a ring scotoma in the left. Results of single bright-flash ERG were unrecordable bilaterally. Western blot analysis detected no serum autoantibodies. One year later, a visual field defect developed in the left eye. Western blot analysis detected serum autoantibody against a 23-kDa protein (recoverin) (Figure 2A). Although the slowly progressive vi-