tic neuritis,3 globe necrosis, and vitreous hemorrhage,4 uveitis, optic neuritis following snake bite range from keratomalacia (venom or ASV serum toxicity). Associated with toxic optic neuropathy ary to an aborted DIC process asso-
crrosis and macular infarction second-
at the posterior pole; or (2) retinal ne-
enclusion with subsequent dislodge of 
could be (1) ophthalmic artery oc-
culopathy (DIC) and ischemic dam-
result in vascular occlusion. Fibrin 
meability. Vasospasm and/or DIC may 
induce severe vasospasm, endotheli-
components of viperine venom) may 
rhagins (complement-mediated toxic 
cellaneous composition of sub-
heterogeneous composition of sub-
Comment. Snake venom is a complex 
cy of the Viperidae family.5 Hemor-
gage following snake bite. Visual prognosis is poor de-
med treatment.

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Slowly Progressive Cancer-Associated 
Retinopathy

Cancer-associated retinopathy (CAR) is caused by antiretinal an-
tibodies cross-reacting with patho-
gens expressed by carcinoma cells. Cancer-associated retinopathy with antirecoverin antibody generally shows rapidly progressive visual de-
terioration.1,3 Autoimmune retinopa-
thy (AIR) is characterized by anti-
retinal antibody–positive serum in 
the absence of systemic carcinoma 
but with slowly progressive visual deterioration,1,3,4 although the patho-
genic mechanism is uncertain. We 
describe a patient with CAR with an-
tirecoverin 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 progres-
ssive visual loss and night blind-
ness. In 1994, Goldmann perim-
etry 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 abnor-
mality. The patient had no history 
of carcinoma or ocular trauma.

When examined in 2004, our pa-
tient’s visual acuities were 20/30 OD 
and 20/20 OS. Slitlamp examination 
demonstrated mild iridocyclitis and nuclear cataract bilaterally. Fundu-
scopic examination results demon-
strated optic disc pallor, retinal ar-
tery whitening, and retinal pigment 
atrophy with partial pig-
mentation in the midperipheral area 
across 360° (Figure 1A). Fluores-
ccein angiography demonstrated a 
window defect corresponding to the 
retinal degeneration (Figure 1B) and 
late leakage from retinal capillary 
vessels. Goldmann perim-
ometry showed global visual field 
deterioration,1,3,4 indicating an auto-
immune mechanism. Slowly pro-
grressive visual deterioration was 
noted in both eyes, and a slowly pro-
grressive pattern of visual field 
deterioration was observed. 

Slowly Progressive Cancer-Associated 
Retinopathy

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Figure 2. (A) Al- 

Figure 3. At 3-month follow-up, coarse pigment clumping and atrophy at the macula and gross attenuation of the macular arterioles are evident. Note also the optic disc pallor.

mg/wk) were started. Three months later, the visual acuity remained no light perception. Optic disc pallor and gross attenuation of perifoveal ves-
sels were noted. The macula showed pigment clumping and atrophy (Figure 3). The likely cause of visual loss could be (1) ophthalmic artery occlusion with subsequent dislodge of fibrin emboli into the end arterioles at the posterior pole; or (2) retinal ne-
crosis and macular infarction secondary to an aborted DIC process asso-
ciated with toxic optic neuropathy (venom or ASV serum toxicity).

Ocular complications following a snake bite range from keratomalacia to vitreous hemorrhage,1 uveitis, optic neuritis,3 globe necrosis, and visual loss due to cortical infarction.5 We are unaware of any previous re-
port in the literature of macular in-
farction following a vipersine snake bite. Visual prognosis is poor de-
spite medical treatment.
visual dysfunction suggested AIR, systemic screening detected a 2-mm bronchioloalveolar carcinoma without metastasis, and the patient was treated with lobectomy. Subsequently, CAR was diagnosed. A few carcinoma cells exhibited cytoplasmic immunoreactivity for recoverin (Figure 2C). One month after the lobectomy, the patient’s visual acuity decreased and late-phase fluorescein angiography demonstrated cystoid macular edema in the left eye and marked leakage from retinal capillary vessels bilaterally. Oral administration of prednisolone, 40 mg/d, was initiated and then tapered across 5 months. At the last follow-up examination, the patient’s visual acuity remained unchanged (20/30 OD and 20/20 OS), with no further progression of the visual field defect.

Comment. Patients with AIR exhibit slowly progressive visual deterioration mimicking retinitis pigmentosa, cystoid macular edema, and retinal vascular edema on fluorescein angiography.1,4 Some patients have systemic benign tumors.4 The patient we describe was diagnosed as having CAR with antirecoverin antibody 11 years after the initial visual symptoms appeared despite clinical features resembling AIR.

Our group previously described a patient with CAR who had aberrant expression of recoverin in many carcinoma cells.2 In the current patient the causative tumor was quite small and only a few carcinoma cells expressed the recoverin antigen, suggesting that the slow clinical course correlated with the low number of recoverin-immunopositive tumor cells, unlike patients in previously reported cases.1,2

Based on the clinical course and immunohistochemical findings, we infer that lung adenocarcinoma or preneoplastic lesions, such as atypical adenomatous hyperplasia5 expressing recoverin, were present when the initial visual symptoms were noticed, suggesting that some patients with AIR have an occult preneoplastic or malignant tumor expressing recoverin. We conclude that regular screening for systemic cancer may be necessary to save the lives and vision of patients with presumed AIR.

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Cascade-like Oculomotor Misinnervation

Report of a Case. A 6-month-old boy with a history of microcephaly and a type II atrioseptal defect was investigated for oculomotor abnormalities. There was no family history of oculomotor abnormalities.

Examination showed an absence of adduction of the left eye on attempted right gaze and during attempted convergence. Instead, simultaneous abduction of both eyes was observed (Figure 1A). However, rhythmic adductive movements with an amplitude of approximately 10° were elicited upon sucking (Figure 1D). We observed both a slight narrowing of the left palpebral fissure and adductive movements simultaneously. Exotropia was observed in the primary position and abduction of the left eye was limited on left gaze (Figure 1B and C). Upgaze and downgaze were normal. No ptosis or enophthalmos were correlated with these findings. Pupillary function was normal, with no unusual changes in eye movements. Right-eye motility was unremarkable.

Comment. Congenital ocular misinnervation can occur in a variety of forms. It typically involves the sixth cranial nerve. Most common is the Duane, or congenital retraction, syndrome, which consists of hypoplasia of the sixth nerve (nucleus) and innervation of the lateral rectus muscle by a branch of the oculomotor nerve. Depending on the relative contribution of third nerve fibers to the medial and lateral rectus, the patient is first with either predominant limitation of abduction or addition. Some of our patient’s clinical features can be explained by an extreme form of the Duane syndrome, where most, if not all, oculomotor nerve branch fibers originally directed to the medial rectus innervate the lateral rectus, thus leading to simultaneous abduction on attempted adduction (also referred to as synergistic divergence). Interestingly, although no left-eye adduction could be elicited on lateral gaze or convergence, we observed adductive movements during sucking. Missing innervation of the medial rectus by oculomotor nerve fibers was replaced by fibers, most likely originating from a motor branch of the trigeminal nerve. Thus, the lack of innervation of the lateral rectus (Figure 2A) appears to have triggered a sequence of aberrant nerve sprouting, resulting, initially, in a shift of fibers originally meant for the medial rectus toward the lateral rectus (Figure 2B). Second, and possibly as a consequence of lack of innervation of the medial rectus muscle, a shift of trigeminal nerve motor fibers to the medial rectus took place (Figure 2C), leading to a trigemino-oculomotor synkinesis between the lateral pterygoid or one of the suprahyoid muscles and the medial rectus. The slight narrowing of the palpebral fissure observed during sucking can be explained by synkinetic contraction of the medial muscle against a tight lateral rectus muscle in that specific situation, which led to discrete retraction of the globe.

The combination of the Duane and Marcus Gunn syndromes or other misinnervation syndromes involving the sixth, third, and fifth cranial nerves recurs. Other cases with synergistic divergence and a trigemino-oculomotor synkinesis have been reported (also...