

## CASE 6: HOMONYMOUS HEMIANOPSIA (Slide CC6-1)

1. Our patient has complete loss of vision in both eyes in the right half of the visual field. This kind of visual field deficit must be caused by a lesion on the left half of the brain affecting neurons containing information from both left hemiretinas. In other words, the lesion is proximal to the optic chiasm on the left side. Note that our patient does *not* exhibit macular sparing. The macula, i.e. the most central portion of the retina, is represented in the posterior portion of the calcarine fissure. This region usually receives a dual blood supply from the posterior cerebral and middle cerebral arteries. Thus, in occlusion of the posterior cerebral artery the macular area is often (but not always) spared due to collateral blood flow from the middle cerebral artery. Therefore, our patient most likely has a lesion affecting either the left optic tract proximal to the optic chiasm, the left lateral geniculate nucleus, the entire left optic radiation, or some combination of the above.

2. The mechanical valve in our patient and the recent cessation of anticoagulation therapy are a perfect set-up for clot formation. Clots in the left ventricle can embolize to the CNS causing infarction. Occlusion of which vessel could cause the deficit seen in our patient? Moving from the optic chiasm toward the visual cortex, the optic tract is supplied by small branches arising from multiple vessels, including the anterior choroidal artery, the middle cerebral artery, the posterior communicating artery and the posterior cerebral artery. Thus, isolated infarction of the optic tract is not usually seen, and is, therefore, probably not the site of the lesion in our patient. Other lesions which could affect the optic tract such as an intracranial mass or local hemorrhage would invariably compress the adjacent cerebral peduncle as well, producing contralateral hemiparesis which was not seen in our patient. The lateral geniculate nuclei, as well as the optic radiations are supplied by deep perforating branches of the posterior cerebral artery. Occlusion of a major perforating branch by an embolus could produce infarction of the lateral geniculate nucleus and/or of the entire optic radiation producing a homonymous hemianopsia as seen in our patient. Another possibility is that the posterior cerebral artery itself was occluded by an embolus before the take-off point of the perforating branches. This would cause infarction of not only the lateral geniculate nucleus and optic radiation but also of the entire inferior temporal and medial occipital lobe, (including the primary visual cortex).

Head MRI:

Based on his exam, the ophthalmologist felt that the patient did not have a problem with the the eyes or optic nerves, but rather, a more central process. His office was adjacent to the hospital, where, the CT scanners were heavily booked but, surprisingly, he was able to obtain an MRI scan the same day (see slide). Are these T1 or T2 weighted images? Hint: note that the CSF is white, gray matter is gray, and white matter is black. The left image is more caudal than the right image. Identify the pons, cerebellum, fourth ventricle, midbrain, temporal lobes, temporal horns of the lateral ventricles, and the occipital lobes. Blood flow produces a dark signal which can be seen in the basilar artery in the left image, and in the middle cerebral and posterior cerebral arteries in the right image. A large infarction is present involving the entire left medial occipital lobe and left inferomedial temporal lobe. Since this infarction occurred several weeks previously, the cortex of the left medial occipital and temporal lobe is thin and gliotic (brighter T2 signal relative to the right side), including the left calcar avus. In the left image, a large irregular black area can also be seen. This is due to hemorrhage that occurred at the time of the infarct which was broken down to produce hemosiderin, a paramagnetic substance causing dark signals on MRI. Note also that the left temporal and occipital horns are relatively enlarged due to atrophy of the overlying medial cortex. Images from further up (not shown) demonstrated involvement of the left posterior thalamus as well. Thus, the right homonymous hemianopsia in

our patient was probably caused by an embolus in the proximal portion of the left posterior cerebral artery.

Clinical Course:

The patient did not regain significant vision in his right hemifields. He was promptly restarted on anticoagulation therapy and has remained without further embolic events.