



## Case Presentation

# Improvement in Compensation for Chronic Poststroke Homonymous Hemianopsia After Initiation of a Selective Serotonin Reuptake Inhibitor: A Case Report

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## Abstract

Homonymous hemianopsia (HH) is a common adverse outcome after stroke. Spontaneous improvement more than 6 months poststroke is thought to be unlikely, and traditional visual rehabilitation techniques lack clear evidence of efficacy. The case presented is of a 22-year-old woman who demonstrated improved compensation of her stroke-induced HH after the initiation of a selective serotonin reuptake inhibitor (SSRI). There is evidence supporting the use of SSRIs to improve poststroke cognitive impairment, motor impairment, and depression. This is the first case, however, demonstrating the potential novel use SSRIs to improve HH compensation after stroke.

**Level of Evidence:** To be determined.

## Introduction

Homonymous hemianopsia (HH) is a common adverse outcome after stroke and generally is caused by lesions in the occipital lobes or optic radiations [1]. HH is correlated with lower quality of life, diminished social functioning, increased dependence/disability, a greater incidence of falls, and overall worse rehabilitation outcomes [1-3].

Suggested treatments for HH include compensatory mechanisms such as optical aids and visual training [1,3-5]. There is a diminishing chance of recovery from HH as time progresses after neurologic injury, and recovery beyond 6 months is unlikely in patients with stable brain disease (ie, stroke). Thus, at greater than 6 months postinjury, "documented improvement in the visual fields of [patients with HH] undergoing rehabilitative therapy would be a sign of therapeutic efficacy" [6]. We present the case of a patient with HH secondary to a remote stroke who demonstrated improvement in compensation for her visual field deficit after being started on a selective serotonin reuptake inhibitor (SSRI).

## Case Report

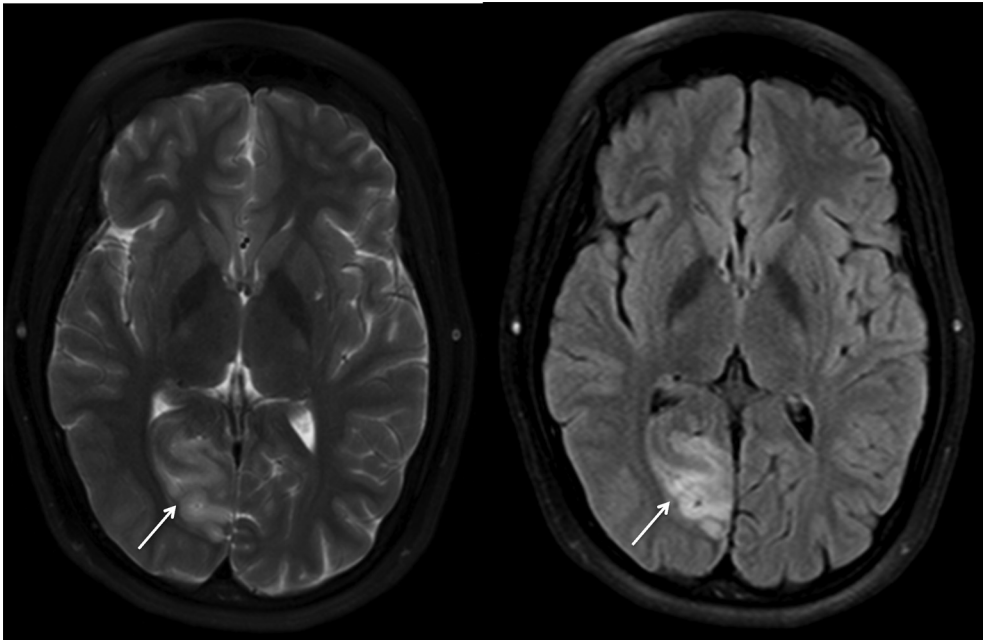
A 22-year-old female patient with a history of stroke, migraines, tobacco abuse, and obesity presented to an

acute inpatient rehabilitation facility after sustaining a mild traumatic brain injury, multiple pelvic fractures, and lumbar vertebral column fractures as a restrained passenger in a motor vehicle collision (MVC).

The patient's stroke occurred 1 year before the MVC. Her visual symptoms began as floaters and flashers on the left side of her visual fields of both eyes, which gradually moved toward the midline and respected the vertical. The lateral visual fields subsequently darkened to blackness. The patient occasionally used tobacco and alcohol and was on lisdexamfetamine and Depo-Provera (Pfizer Inc, New York, NY) at the time of her stroke. Magnetic resonance imaging of the brain at the time of her stroke demonstrated an infarct in the right posterior cerebral artery territory (Figure 1). Vascular imaging demonstrated stenosis of the P3 segment of the right posterior cerebral artery.

To determine the etiology of the stroke in an otherwise-healthy young patient, a laboratory evaluation for hypercoagulable states along with a transesophageal echocardiogram to rule out cardiac sources of emboli were performed and were negative. The patient was discharged on aspirin 325 mg daily. The patient returned home after her stroke and did not undergo any visual rehabilitation.

The patient did have follow-up with neuro-ophthalmology shortly after her stroke. At that time,



**Figure 1.** Magnetic resonance imaging of the brain. Note hyperintense area on T2-weighted (left) and fluid-attenuated inversion recovery (right) sequences demonstrating an area of abnormal signal in the right occipital lobe representing an acute to early subacute infarction in the right posterior cerebral artery territory (white arrow).

Humphrey visual field testing showed a congruent left HH with minimal sparing of paracentral field superiorly (Figure 2, top). The patient subsequently had follow-up with ophthalmology 3 weeks before admission (and 3 days before her MVC). At that point, she did not demonstrate improvement in visual fields or compensation, neither subjectively or objectively, by confrontation or Goldmann visual field testing (Figure 2, middle).

After the patient's MVC, she had a brief loss of consciousness (exact duration unknown but suspected to be less than 10 minutes) and her initial Glasgow Coma Scale at time of admission was 15. Computed tomography scan demonstrated continued evidence of her remote posterior cerebral artery infarct without any acute intracranial abnormalities (Figure 3). The patient did not have any retrograde amnesia but did report 3-4 days of anterograde amnesia after the accident.

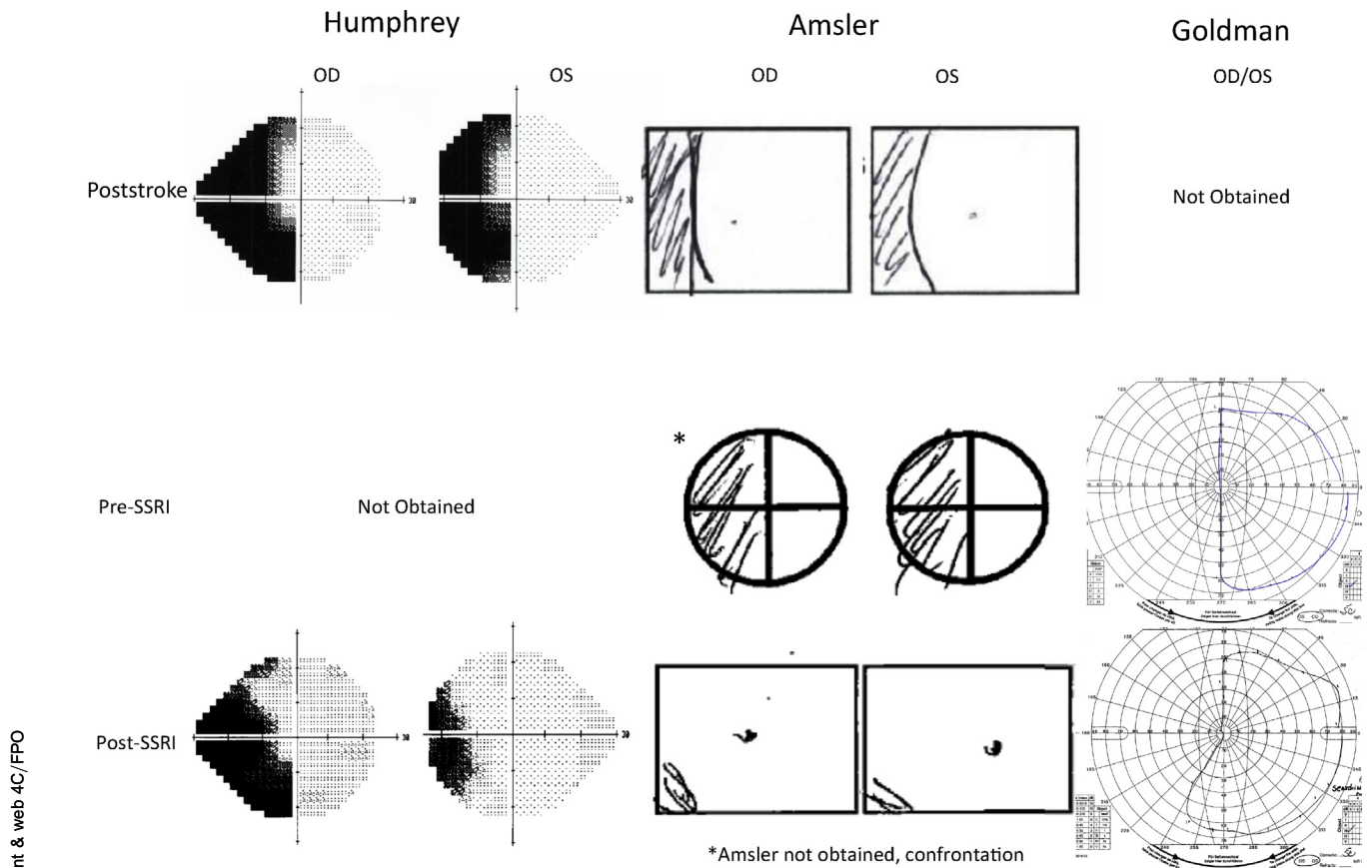
At the time of admission to acute inpatient rehabilitation for her traumatic injuries sustained in the MVC, the patient reported that all of her poststroke symptoms had abated with the exception of unchanged HH. On physical examination, she continued to have a complete left visual field cut in both eyes on confrontation visual field testing. She had not driven since her stroke, given her visual field deficits. The patient remained upset about not being able to pick her daughter up from school and felt that her visual deficits led to difficulty maintaining a job. She also reported running into people at times, difficulty avoiding environmental hazards, and having a fear of large crowds and shopping in stores.

During the patient's stay in acute inpatient rehabilitation, she was noted to have recurrent episodes of tearfulness. She reported feeling depressed and overwhelmed for several months. The patient was started on citalopram 20 mg daily on acute rehabilitation day 8 to treat her suspected depression. Over the following week, the patient described gradual but significant improvement in her HH to the point where she had minimal left sided deficits on confrontation visual fields. Patient was not started on any other neurotropic medications during her hospitalization, and there were not any other medications changes made around the time of initiation of citalopram.

The patient reported continued subjective improvement in her visual fields and quality of life 3 months after discharge from acute inpatient rehabilitation. Amsler and Humphrey visual field testing at that time showed improvement in compensation for her HH compared with her previous neuro-ophthalmology visits (Figure 2, bottom), although loss of fixation was noted on Humphrey testing. Goldmann visual fields continued to show complete left HH.

## Discussion

Spontaneous recovery of visual fields in patients with HH decreases as time progresses after neurologic injury. In a study by Zhang et al [6], 254 patients with HH were monitored for improvement in their visual fields. Although approximately 55% of patients seen within 1 month demonstrate spontaneous recovery, no patients with stable underlying brain disease (such as stroke) and



**Figure 2.** Visual field testing, the top images represent her left (OS) and right (OD) visual fields immediately after her stroke; middle images represent the patient's visual fields at the her visit shortly before being started on a selective serotonin reuptake inhibitor; and the bottom images represent her visual fields 3 months after initiation of a selective serotonin reuptake inhibitor. The left column demonstrates the patient's Humphrey visual field tests: darker areas represent greater vision loss. The middle column shows the patient's Amsler/confrontation testing with darker areas represent greater vision loss. The right column represents the patient's Goldmann testing with areas within the drawn circles representing preserved vision.

stable cognitive status demonstrated recovery after 6 months. Only 5.3% of patients with HH demonstrated complete recovery of their visual fields.

Attempts at compensation strategies for patients with HH consist of oculomotor training to explore visual fields and optical aids such as prisms to help expand the visual field and relocate images [3]. These visual rehabilitation programs tend to require specialized equipment, training, and expertise and definitive benefit of these techniques remains unclear [1].

In this case, the improvement in the patient's vision is felt to be the result of improvement in compensation for her deficits (similar to the type of improvement seen with optical aids and visual training) and not the result of improvement in her visual fields. Amsler, Humphrey, and Goldmann testing all require constant fixation on a target. Patients who are using compensation techniques such as scanning may have falsely enlarged visual fields and unreliable testing results. In this case, the discrepancy in improvement in Humphrey and Amsler fields compared with Goldmann testing is felt to be due to loss of fixation noted during Humphrey (and assumed to also have occurred during Amsler testing) without loss

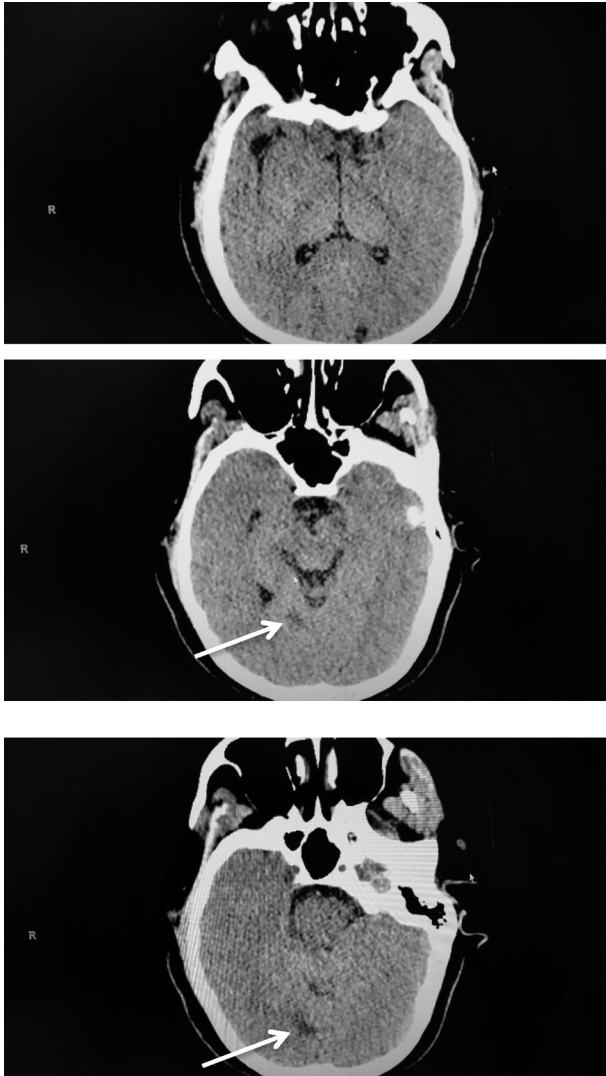
of fixation during Goldmann testing (which is constantly under examiner supervision). Static visual field testing (ie, Humphrey) may be more susceptible to loss of fixation compared to kinetic visual field testing (ie, Goldmann) [7,8].

Given that spontaneous improvement has not been noted in patients with stroke after 6 months and that our patient did not receive any formal visual field training or optical aids, the improvement seen by our patient is most likely as the result of improved compensation secondary to the initiation of citalopram.

SSRIs have been suggested as a treatment for a wide range of poststroke sequelae, including cognitive impairment, motor impairment, dependency, disability, depression, and anxiety after stroke [9,10].

Various mechanisms for the beneficial effects of SSRIs have been suggested including directly through neurogenesis, neuroplasticity, neuroprotection, and adrenergic effects, as well as indirectly through effects on depression [11].

Maya-Vetencourt et al [12] found that fluoxetine, an SSRI, helped to improve visual acuity in adult rats with amblyopia. This improvement was felt to be a direct



**Figure 3.** Noncontrast computed tomography scan of the brain after the patient's motor vehicle collision. Top image demonstrates posterior cerebral artery territory without stroke. Bottom 2 images demonstrate the remnant areas of remote infarct (white arrow).

benefit of increased serotonin transmission leading to increased neuroplasticity, especially because it was associated with reduced intracortical inhibition and the increased expression of brain-derived neurotrophic factor, a key driver of neuroplasticity at the molecular level. Furthermore, these effects were negated by the coadministration of the benzodiazepine, diazepam. To the best of our knowledge, this is the only study to date demonstrating a direct effect of SSRIs on the visual cortex.

There is additional evidence of a direct effect of SSRIs on neuroplasticity in the motor cortex. Espinera et al [13] demonstrated that citalopram, an SSRI, led to improved functional recovery, which was likely due to increased neurotrophic factors, neuromigration, and neurogenesis. Pariente et al [14] found that a single dose of fluoxetine led to improved motor function via

increased activation of the motor cortex in patients with pure motor hemiplegia. A single dose of fluoxetine was unlikely to have had indirect effects via improvement in patient depression.

The Fluoxetine for Motor Recovery After Acute Ischemic Stroke (FLAME) trial showed that patients with hemiplegic stroke treated with fluoxetine had improvement in motor outcomes at 3 months compared with patients receiving a placebo. Although patients with depression were excluded from this trial, those receiving fluoxetine did have lower onset of newly diagnosed depression [15]. Other studies also have shown there have been improvements in function and overall outcome independent of depression [16-18].

Although these studies suggest that SSRIs have a direct beneficial effect on the neuroplasticity of stroke patients, other studies have demonstrated indirect benefits of SSRI on function through improvements in depression. Poststroke depression is common and is seen in approximately one third of stroke survivors [19]. A review by Robinson and Spalletta [20] found a correlation between poststroke depression and impaired functional status, with improvement in depression leading to improvement in functional status. A Cochrane Review by Mead et al [10] demonstrated improved dependence and disability after the initiation of an SSRI in patients with stroke with more marked improvement in patients with preexisting depression. One possible mechanisms for these findings include impaired participation in rehabilitation in depressed patients [21].

After the initiation of an SSRI, the patient in this case demonstrated improved visual compensation after a remote ischemic stroke. We believe the SSRI was the cause of this recovery, given that spontaneous recovery is extremely unlikely at 1 year and the improvement was temporally related to the initiation of the SSRI.

It is unclear in this case whether the improvement in her compensation was secondary to improvement in her depression leading to improved visual functioning or due to direct effects on the neural visual system. Previous studies have used functional magnetic resonance imaging and diffusion tensor imaging to visualize lesions in the visual pathways and subsequent cortical reorganization [22-24]. With the use of these techniques, the improvement in visual fields after the initiation of an SSRI has the potential to be more completely evaluated.

Further research is needed to further explore these questions. To our knowledge, the beneficial effect of SSRIs to improve vision has not been documented in the literature in human patients. SSRIs may prove to be an adjunctive treatment for HH and the potential for therapeutic synergy with more traditional rehabilitation techniques has not been explored. A more formalized randomized control study is needed to further evaluate this potential.



## Conclusion

Visual field deficits are common sequelae of stroke, and late spontaneous recovery is rare. SSRIs have been demonstrated to have beneficial effects in a wide range of poststroke conditions. This case report suggests the potential for a novel use of SSRIs to improve compensation for HH after stroke.

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## Disclosure

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