Complex Visual Hallucinations, Presumably Due to Subarachnoid Hemorrhage, Treated Successfully With Risperidone

To the Editor: Visual hallucinations have several known nonpsychiatric etiologies and may be treated accordingly. We present, with interest, a case of a 56-year-old woman who developed complex visual hallucinations (CVH) status post-cerebrovascular accident (CVA) in her right middle cerebral artery (MCA) distribution. The hallucinations appear to have been successfully treated with risperidone.

Case Report

Our patient is a 56-year-old, right-handed woman who presented with a large right-MCA aneurysm and several small left-MCA aneurysms. Her right-MCA aneurysm was successfully surgically repaired; no operative intervention was required for the left aneurysms. In addition to transient sensory and motor deficits, her son noted that after the surgery she would report seeing a woman and a black cat. She had no previous visual dysfunction, although, interestingly, her insight into the CVH improved even before medication treatment. Our patient returned 3 weeks later with new hemorrhages in her right-MCA area, with ischemia. No new surgeries were performed, and her condition stabilized after 1 week with appropriate medical intervention. During this admission, her CVHs persisted, and the authors were consulted because of her "behavioral disturbances." There was no apparent association between the hallucinations and sleep or time of day. There were no reports of complex partial or generalized seizures. Per her son's report, she had no previous history of psychiatric illness, visual hallucinations, or auditory hallucinations. She had no history of substance abuse. She did not have fluctuating awareness or alertness; she had a Richmond Agitation And Sedation Scale score of 0 and 26/30 on the Mini-Mental State Exam. Risperidone 1.5 mg/day was prescribed in an attempt to treat her CVHs, which resolved within 7 days of initiating treatment.

Discussion

Although the etiologies for CVHs have been reviewed elsewhere,[1] our patient's phenomenology merits a discussion of delineating between Charles-Bonnet syndrome secondary to cortical stroke. With respect to the latter, CVHs may develop secondary to activated occipital cortical regions adjacent to stroke-damaged areas with associated visual field deficits,[2] whereas stroke is generally regarded as a condition in which decreased afferent input that yields CVHs of a particular quality (straight ahead, colored, people, insight), although it also occurs in patients with visual-field defects and normal central acuity.[3] The theories proposed to explain the etiology of both conditions fall into two categories. The "deafferentation" theory proposes that the lack of visual sensory input into the cortex causes spontaneous neuronal discharge leading to abnormal visual perceptions not unlike the phantom-limb syndrome. The second, "perceptual release" theory, is based on the premise that a normal sensory input inhibits irrelevant impulses from the conscious perception of images. Where there is a reduction in sensory input, the threshold to suppress irrelevant images cannot be achieved, and previously subconscious perceptions are "released" into consciousness, resulting in CVHs.[4]

Risperidone has potent serotonin type-2 receptor antagonism and has been suggested by some authors to have efficacy in treatment of CVH through the blockade of such receptors in the visual cortex.[5]

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