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Case Reports

Functional Neuroimaging Correlates of Medically Unexplained Vision Loss

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Hysteria has long been a puzzling and intriguing issue in neuropsychology. This old and complex term refers to conditions otherwise described as "somatoform" or "conversion" disorder. Conversion disorder refers to a hypothesis based on psychoanalytic theory: it involves sensory or motor symptoms, which cannot be explained by any known medical condition, that are not consciously produced and are considered to be associated with psychologic factors. The Diagnostic and Statistical Manual of Mental Disorders-5 (DSM 5) proposed renaming conversion disorder as "functional neurologic disorder."[1,2] Neurology outpatient clinics report 10%–30% of patients complaining of medically unexplained symptoms, which are, therefore, thought to be "nonorganic," "functional" in nature and bear a poor prognosis for recovery.[3,4]

As far back 1870, Jean-Martin Charcot first hypothesized that hysteria may reflect some sort of dynamic, functional brain abnormality.[5] The last decade has witnessed a number of functional neuroimaging studies that have provided support for Charcot's theory, revealing abnormal brain perfusional patterns in patients with functional symptoms. These studies showed a selective decreased activity of frontal, prefrontal, and subcortical circuits involved in motor control during hysterical paralysis,[6–8] reduced activity of somatosensory cortices during hysterical anesthesia,[9] and a reduced visual cortex activity during functional visual loss.[10] However, increased activation has been detected by functional neuroimaging studies in limbic regions, including the cingulate and orbitofrontal cortex, during conversion symptoms involving different sensory or motor modalities.[11] However, the neural mechanisms that might unconsciously lead to psychologic stressors resulting in physical, medically unexplainable symptoms, still remain to be clarified.

Functional visual loss is defined as any decrease in vision where the origin cannot be attributed to a pathologic or structural abnormality.[12] It occurs in 1%–5% of patients attending ophthalmology clinics and often runs a chronic course.[13] In 1910, Freud conceptualized unexplained visual loss as a type of "unconscious conversion," but the question of whether a patient with hysterical visual impairment can or cannot "see" or whether he or she is unaware of the visual stimulus remains unresolved.[14]

Case Report

Ms. A, a 23-year-old woman, with no medical history other than migraine without aura, was admitted to the hospital for acute, bilateral amaurosis; and after an extensive neurophysiologic and neuroimaging workup, she was diagnosed as having a functional disorder. The only medication she was on was combined oral contraception. She was admitted in the emergency department for sudden, complete bilateral vision loss in the form of loss of light perception in both

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eyes, without headache. Ophthalmic examination revealed no abnormalities, such as relative afferent pupillary defect, and both pupils were equal, round, and fully reactive to light. Intraocular pressures were within the normal range, and the anterior and posterior segments of both eyes appeared normal.

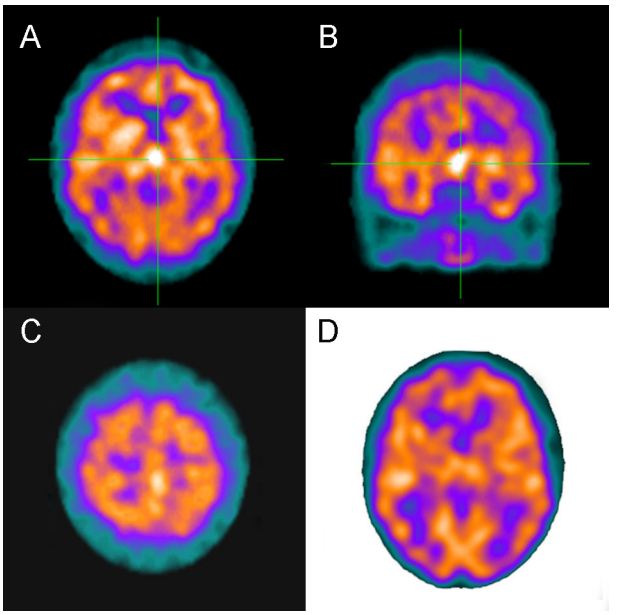
She was referred for neurologic examination, which did not reveal any other clinical deficit, apart from bilateral visual loss (blind visual nasal/temporal fields in both eyes) and precautionary gait (although she navigated her space with uncertainty, she did not bump into objects in the room); in particular, the motor functions, finger-to-nose testing, and cognitive state were unremarkable. She did not react emotionally to visual stimuli. No mood or psychotic disorder was evident at the clinical interview, nor was there any family history of mental disease. However, she was quite indifferent to her health problem, although she did relate a certain degree of anxiety about some relationships in the family context. In particular, she reported a conflicting relationship with her mother, after her parents divorced.

Visual evoked potentials showed no abnormality in the electrophysiologic response pattern (normal amplitude and latency of the P100). Magnetic resonance imaging (MRI) with gadolinium and diffusion-weighted imaging ruled-out any signal intensity alterations in the posterior visual pathway, and an MR angiographic study of extracranial and intracranial vessels was unremarkable. Based on her history of migraine, aura of the basilar type was then postulated. Functional neuroimaging investigations were performed 18 hours after she had reported onset of vision loss: a ^{99m}Tc-hexamethylpropylene amine oxime single-photon emission computed tomography (SPECT) of the brain was qualitatively evaluated by a skilled nuclear medicine specialist and showed focal hyperperfusion in the left thalamus and posterior cingulate cortex, without any other perfusional abnormality consistent with migrainous aura (Figure a,b,c).

She made a complete and spontaneous recovery on the fourth day and was discharged with the diagnosis of "blindness of conversive nature in a patient affected by migraine without aura."

Two months later, the hexamethylpropylene amine oxime SPECT of the brain was repeated: the thalamic/cingulate hyperperfusion was no longer detectable, suggesting that it had been a transient alteration in perfusion, related to the functional visual loss (Figure d).

FIGURE. **Brain HMPAO SPECT Alterations During and After the Functional Visual Loss. Brain HMPAO SPECT Carried Out During the Visual Loss, Qualitatively Evaluated by a Skilled Nuclear Medicine Specialist, Showed a Focal Hyperperfusion in the Left Thalamus on Axial (A) and Coronal (B) Scans and in the Left Posterior Cingulatus on Axial Scans (C); After 2 Months, the Thalamic Perfusional Alteration was No Longer Detectable (D). HMPAO SPECT = Hexamethylpropylene Amine Oxime Single-photon Emission Computed Tomography.**



Discussion

Recent functional brain imaging studies have revealed neural circuits involved in complex mental processes potentially related to conversion disorder. Moreover, abnormal activity of the thalamus and posterior cingulate has been reported in some cases of functional loss of motor and sensory functions.[11] In a previous study,[10] brain activation induced by periodic (monocular) 8-Hz visual stimulation by whole-field color flickers was investigated by functional MRI in 5 patients with unexplained visual loss who fulfilled Diagnostic and Statistical Manual of Mental Disorders-IV criteria for conversion disorder and 7 asymptomatic volunteers. Compared with controls,