

Face-Related Ictal Hallucinations and Illusions and Reflex-Type Phenomena

To the Editor: It is generally accepted that pathological excitation of visual cortical areas is responsible for ictal visual hallucinations and that visual association cortex is both necessary and sufficient for this phenomenon. The direct electrical stimulation of the right anterior inferior frontal gyrus was recently shown to result in face-related hallucinations and illusions. Here, we report a patient presenting with complex partial seizures of a probable right frontal origin, who described auras of face-related visual illusions and hallucinations and a susceptibility to face-related reflex-type seizures.

Case Report

A 33-year-old, single, right-handed Nigerian woman presented with a 13-year-long history of refractory diurnal and stereotyped complex partial seizures. These deteriorated with levetiracetam treatment, and she was referred for further investigations. Her seizures initially began with a visual aura in which she would see human faces or face-like objects that seemed to be enlarging and changing in shape or intensifying in color. She also described experiencing visual hallucinations of faces as part of her initial aura. Her habitual seizures were described by her family as starting with a scream, behavioral arrest, staring and unresponsiveness, followed by abduction and dystonic posturing of her right arm and grabbing or wiping automatisms. There was no associated loss of balance. The attacks lasted about 15–30 seconds and were associated with postictal confusion of no longer than 2 minutes. Full recov-

ery followed. She was usually amnesic for the attack; however, sporadically she could recall her ictal experiential phenomena, mainly face-related visual illusions or hallucinations, but rarely auditory hallucinations.

The attacks appeared daily, up to five a day. Infrequently, they could also be brought on by her looking at herself in the mirror or by looking at pictures, paintings, or any shapes or forms resembling faces. Consequently, she recoiled from looking into mirrors and magazines, and her family avoided exhibiting family photos or portraits in their home. Over the years she was prescribed multiple anti-epileptic medications in sufficient doses and for an appropriate duration of time. However, she continued to have frequent attacks while reduction of some of the regimens provoked generalized tonic-clonic seizures. While taking levetiracetam, 1g daily, her attacks became more frequent and were accompanied by falls. She reported feeling “that someone was taking over her body during the attack and trying to push her backward (sic).” She also described a distinct sensation that her spine was pulled by a hook and the skin on her back was being grabbed from behind. Levetiracetam was stopped, and the falls and her ictal experiences of kinesthetic hallucinations and somatic passivity subsequently ceased. She is currently being treated with topiramate, 50 mg b.i.d., and sodium valproate, 1 g daily, and her seizure frequency has decreased to two times a day.

Of germane history, as a child she had been successfully treated for cerebellar medulloblastoma with posterior fossa resective surgery. This was followed by irradiation of the entire cerebrospinal axis and chemotherapy. The secondary complications included hypopitu-

itarism with short stature and primary ovarian failure, which required hormone replacement therapy. She is still taking levothyroxine, 100 μ g daily. Her medical and family history is otherwise unremarkable. No history of affective flattening, thought blocking, or lack of motivation or social withdrawal was reported, and the patient never experienced a psychotic state independent of her seizures. A general mental, cognitive, and physical examination was normal apart from moderate kyphosis, minimal left upper extremity dystaxia, and gait ataxia.

An MRI brain scan showed postoperative changes of midline suboccipital cerebellar surgery with no evidence of mass or abnormal enhancement to suggest residual or recurrent tumor. There was a prominence of the ventricles and CSF spaces about the brain consistent with generalized cerebral volume loss and a mild degree of compensated communicating hydrocephalus. There was no obvious focal cortical abnormality identified and no clear hippocampal volume loss demonstrated.

Her interictal EEG showed right fronto-temporal slowing. In addition, occasional high amplitude delta waves, superimposed sharp waves and epileptiform discharges were seen independently over the right and left temporal and sylvian regions, but more so on the right. Video-telemetry was also performed, during which several habitual attacks were taped, but no face-induced reflex type seizures were captured. After one of the attacks our patient remembered seeing “a face of a monster” and described a vivid visual hallucination. Ictal recorded EEG showed a right prefrontal onset within the muscle artifacts, with spread to mid-frontal and central regions. Later, a clear left frontal slowing

was seen in the delta range, which was then replaced by late activity in the same region. These changes lasted for about a minute.

Discussion

Recent imaging, anatomical, and physiological data support an interaction between multiple highly focal locations in both temporal and prefrontal cortices during face processing.¹ Vignal and colleagues² showed that direct electrical stimulation of the right anterior inferior frontal gyrus resulted in face-related hallucinations and illusions. The authors hypothesized that the remote activation of the primary processing area in the temporal lobe from the secondary processing area of the prefrontal region could explain observed experiential phenomena.

The distinction between illusions and hallucinations resulting from a focal epileptic discharge is not as well understood. We describe a patient with a probable right prefrontal seizure onset experiencing ictal illusions of faces enlarging and changing in shape. At other times, without a clear external stimulus, the patient reported face-related hallucinations as an aura. Under the theory suggested by Vignal et al.,² the face-related experiential phenomena could be viewed here as a result of frontal epileptic discharges projecting to quiescent face-representing regions of the temporal lobe. Alternatively, in the presence of an actual sensory stimulus, the spread from the prefrontal site would distort ongoing activation, resulting in a face-related illusion. Of note is that in our patient pictures of faces also triggered some of the attacks.

To our knowledge, this is the first report to show the face-related ictal hallucinations/illusions and propensity to face-related reflex-type seizures in epilepsy of proba-

ble frontal origin. We cannot exclude the possibility that these experiential phenomena were solely the result of spread to temporal lobes. Furthermore, the observed EEG changes could reflect spread from another part of the brain and possible seizure onset elsewhere with early frontal spread (e.g., from an MRI-undetectable occipital lesion as a result of her past posterior fossa surgery or irradiation). We were unable to record reflex-type seizures during the video-telemetry and consequently were unable to analyze localization of the discharges. However, we propose that during such attacks, faces or pictures of faces act as afferent volleys to trigger pathological hyperexcitable loops in prefrontal and temporal cortical regions, thus leading to uninhibited reflex response and seizure induction.³

Finally, we note that levetiracetam caused worsening of habitual seizures in our patient. It also provoked some unusual ictal experiential phenomena such as somatic passivity and kinesthetic hallucinations, all of which ceased when levetiracetam was stopped. To date, there are only a few reports in the literature regarding the association of levetiracetam with psychosis, none of which pertained to ictal psychotic phenomena.⁴ Deterioration of epilepsy is an uncommon but well-recognized complication of all antiepileptic drugs.

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Parkinson's Disease: Presenting a Gray Box Model

To the Editor: Parkinson's disease is one of the most common neuromuscular disorders. The disease has a variety of movement symptoms. Neural modeling can be useful to analyze the brain behavior and to understand the disease. Modeling the disease is more convenient, inexpensive, and faster than experimental and clinical researches. Computational models could help researchers to predict different treatments and study some hardly accessible states of the disease, such as early mild stages. The important constraint of the