


New-Onset Fixation-Off Sensitivity or Scotosensitivity in an Adult Male After Bilateral Embolic Strokes of the Parietal and Occipital Regions

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Abstract

A 50-year old male suffered new-onset clinical and subclinical seizures provoked by acute bilateral embolic strokes in the parietal and occipital regions as a complication of aortic valve replacement surgery. After successful treatment of these refractory symptomatic seizures with intravenous propofol and antiseizure medications, video-EEG recordings revealed acute and transient fixation-off sensitivity or scotosensitivity that resolved within 10 weeks.

Keywords

fixation-off sensitivity, scotosensitivity, electroencephalogram (EEG), EEG, adults, cerebral infarction, stroke, seizures, embolic

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Introduction

Fixation-off sensitivity (FOS) is characterized by posterior focal or generalized epileptiform discharges or seizures that are induced by elimination of central vision/fixation typically by eye closure.¹ FOS was first and most commonly observed in patients with idiopathic childhood occipital epilepsies, but has also been described in subjects with symptomatic or cryptogenic focal and generalized epilepsies, even in nonepileptic individuals.² Though FOS may persist into adulthood, such cases are thought to represent a prolongation of the same phenomenon that arose in childhood often representing symptomatic forms of epilepsy.³ FOS has been postulated to be associated with occipital hyperexcitability.⁴

The rare phenomenon of scotosensitivity is defined as the emergence of interictal or ictal activity induced by the obstruction of retinal light stimulation achieved by eye closure or by maintaining complete darkness.⁵ FOS is purportedly differentiated from scotosensitivity by the elimination of central vision and fixation in the presence of light with the use of modified Ganzfeld stimulation or Frenzel lenses and observing for the characteristic high-amplitude occipital or generalized paroxysmal discharges. However, most cases described as scotosensitivity are suspected to represent FOS.¹

We present the case of an adult male whose electroclinical findings are consistent with FOS or scotosensitivity following recurrent secondarily generalized seizures likely symptomatic from multiple bilateral acute ischemic insults to the posterior head regions. FOS or scotosensitivity, as the cardinal feature of symptomatic occipital lobe epilepsy, has been rarely reported

in the literature^{6,7} with no reports of such phenomenon as an acute and transient finding in poststroke seizures in this age group.

Case Report

A 50-year-old right handed, Caucasian male had aortic valve replacement surgery for aortic stenosis. Shortly after the procedure, the patient had a witnessed generalized tonic-clonic seizure (GTCS). Treatment with intravenous lorazepam was initiated. The patient had recurrent seizures requiring repeated doses of lorazepam and a loading dose of intravenous levetiracetam. Because of the inadequate remission of his seizures, the patient then received a loading dose of intravenous fosphenytoin. Despite these urgent measures, the patient continued to experience GTCS that were preceded by limb posturing and versive head turning to the left. After being transferred to the intensive care unit, he was intubated, started on continuous video-EEG monitoring (CEEG), and placed on a propofol drip.

The CEEG recorded both clinical and subclinical events characterized by independent, bilateral electrographic seizures broadly emerging from the posterior head regions. Three clinical

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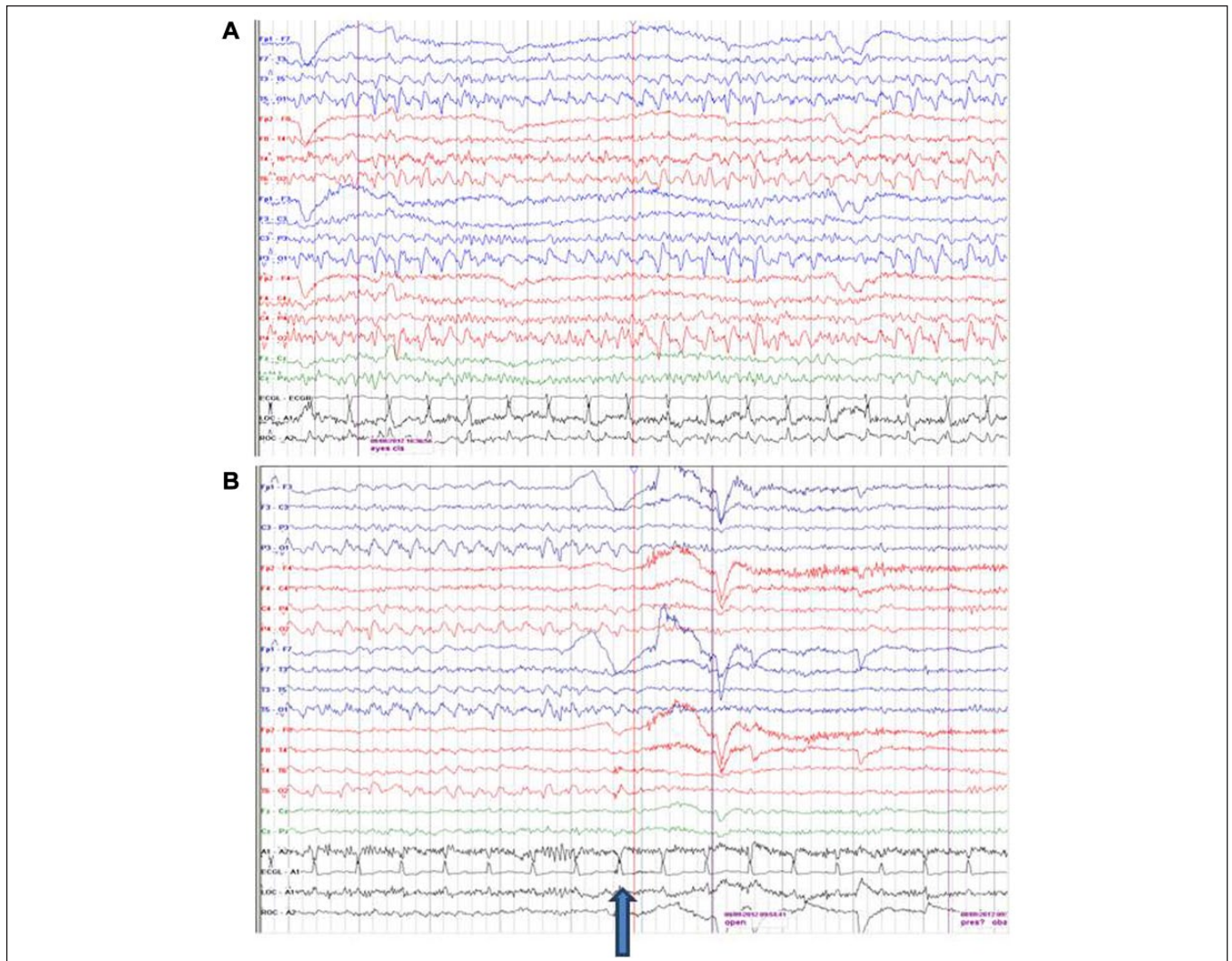


Figure 1. (A) Bilateral rhythmic repetitive sharply contoured occipital negative discharges. Fixation-off sensitivity (FOS) versus scotosensitivity phenomenon in occipital regions with eye closure. (B) The discharges end in response to eye opening (arrow). Awake EEG (bipolar longitudinal montage, 10-20 electrode system, 10-second duration, average amplitude 100 μ V, LFF [low frequency filter] 1 Hz, HFF [high frequency filter] 70 Hz).

seizures were recorded (as described above) that correlated with the emergence of rhythmic 2-Hz activity across the right hemisphere with maximal negativity in the posterior channels prior to extension into the left hemisphere after 2 to 5 minutes. Moreover, there were 4 subclinical electrographic seizures characterized by the emergence of rhythmic 3-Hz activity in the bilateral posterior regions that evolved in frequency more prominently across the left hemisphere. The propofol drip was titrated until modified burst suppression was achieved approximately 6 hours after seizure onset, and was subsequently maintained for 24 hours with the combination of propofol, levetiracetam, and fosphenytoin.

In the next 24 hours of recording, the propofol drip was slowly tapered with the CEEG transitioning from a modified burst suppression pattern into a background composed of diffuse, rhythmic alpha activity at 10 to 12 Hz that was minimally reactive to external stimulation. The background was interrupted

by brief runs of bilateral 1.5 to 2.0 Hz rhythmic delta activity with superimposed, independent sharp waves in the right and left posterior head regions. For the next 24 hours, no further clinical seizures were recorded on CEEG as the patient was successfully weaned off propofol and extubated. The EEG continued to reveal a poorly reactive background with diffuse alpha activity.

The following day, a routine EEG with video was completed. The study showed mild diffuse background slowing and frequent runs of high amplitude, spike and sharp wave discharges at 3- to 4-Hz frequency in the occipital regions bilaterally (maximal at O1 and O2) lasting 20 to 30 seconds in duration that emerged within 0.5-1 second of eye closure and terminated with eye opening. On video, the patient remained conversant, appropriate, and cooperative during these discharges (Figure 1). The complexes did not spread to other locations. A follow-up routine EEG was performed the

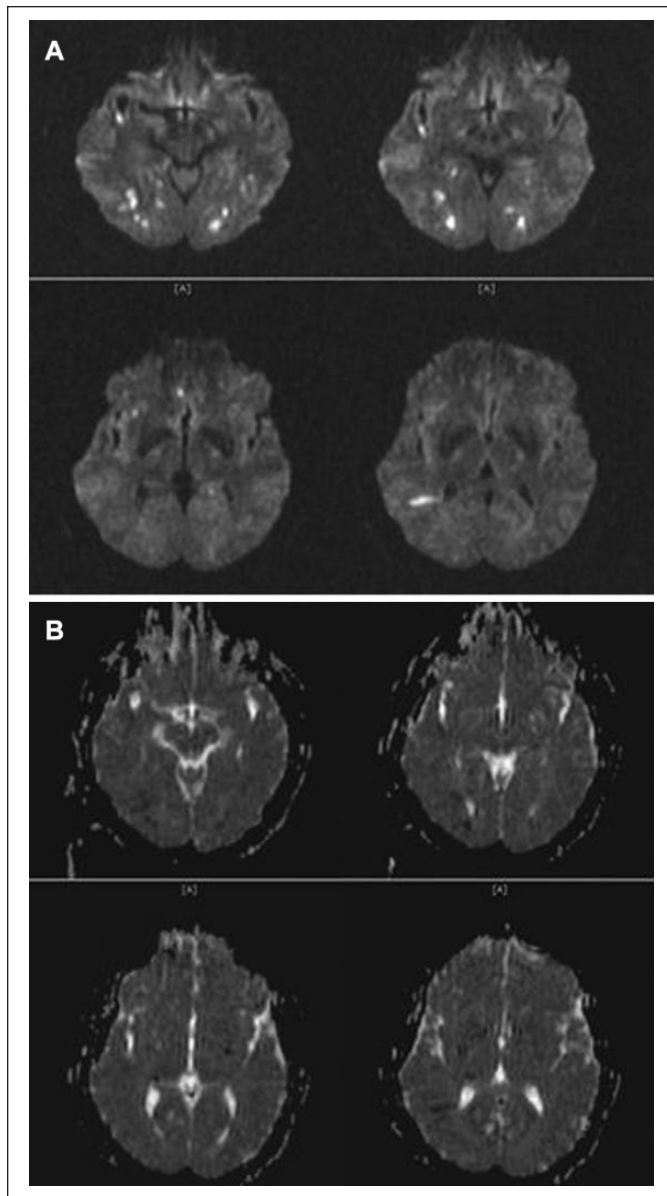


Figure 2. Axial brain magnetic resonance image showing acute infarcts in the posterior regions bilaterally. (A) Diffusion-weighted imaging (DWI) sequences and (B) apparent diffusion coefficient (ADC) sequences.

next day that demonstrated similar clinical and electrographic findings. Photoc stimulation or other provocative maneuvers were not performed during these routine records. The patient was maintained on oral levetiracetam with no further clinical seizures.

A nonenhanced brain magnetic resonance imaging (MRI) was obtained on the same day as when the patient was extubated and it demonstrated multiple foci of T2 and FLAIR (fluid-attenuated inversion recovery) signal hyperintensities scattered throughout the white matter, most prominently in the parietal and occipital lobes with diffusion restriction suggesting embolic infarcts in the setting of recent aortic valve replacement surgery (Figure 2).

Ten weeks after the patient was discharged from the hospital, an outpatient routine EEG was completed that revealed a normal awake and sleep background without interictal or ictal abnormalities at resting state with eye opening or eye closure. The patient reported no residual deficits and no seizure recurrence. He was able to return to full employment without restrictions or accommodations.

Discussion

FOS and scotosensitivity, phenomena that are elicited by the elimination of central vision, can be differentiated from eye closure sensitivity (ECS). ECS refers to the emergence of epileptiform discharges attributed to the interruption of light due to the physical mechanism of eye closure.⁸ In contrast to FOS and scotosensitivity where continuous epileptiform discharges consistently occur within 1 to 3 seconds of eye closure, persist throughout eye closure, and disappear immediately with eye opening, epileptiform paroxysms from ECS occur more gradually after eye closure, are self-limited and brief typically lasting 1 to 4 seconds, and do not persist for the entire duration of the eye closure period.^{1,9}

Testing with modified Ganzfeld stimulation or Frenzel lenses was not performed, which would have helped differentiate FOS from scotosensitivity. But as asserted by others, scotosensitivity overall is extremely rare. Furthermore, most cases in the literature described as scotosensitivity are suspected to represent FOS.¹

The electroclinical finding of FOS or scotosensitivity as an acute and transient phenomenon after acute stroke has not been previously reported. The advanced age of onset of our patient is novel as FOS is typically observed in children with idiopathic epilepsy syndromes with occipital paroxysms such as Gastaut syndrome, Panayiotopoulos syndrome, eyelid myoclonia with absences and atypical features, and idiopathic generalized epilepsy with photosensitivity.^{3,4} When observed in the adult population, FOS has been most frequently associated with symptomatic epilepsy syndromes representing a continuation of the same remote pathology of childhood. The interictal and ictal abnormalities recorded in adults have predominantly been in the posterior regions (temporo-occipital, occipital, temporal, temporo-parietal region)^{6,10} consistent with the electroclinical findings in our case.

The etiology of FOS or scotosensitivity in our case also distinguishes this case from the other causative pathologies identified in existing literature. There are reports in children with cryptogenic focal and symptomatic epilepsies in association with Sturge-Weber syndrome, celiac disease, cortical dysplasia, and bilateral occipital gliosis.² Hassan et al⁷ reported of a 19-year old male who had habitual seizures from the age of 3 years exhibiting persistent left-sided FOS following remote perinatal insult. His brain MRI revealed gliosis in the bilateral parieto-occipital regions and ulegyria over the left posterior occipital cortex.⁷ This report is the closest approximation of structural pathology associated with FOS or

scotosensitivity to an acute multifocal ischemic process in the occipital regions as demonstrated in our case, though the timing and exact nature of the insults differ. These cases also diverge in the lack of persistence of FOS or scotosensitivity in our patient whose EEG 10 weeks after initial insult was interpreted as normal. This is an observation consistent with FOS as it has been thought to be a condition with relatively low epileptogenicity.⁴

FOS is thought to be modulated by α -rhythm generators and influenced by cortical hyperexcitability.⁴ The exact location and extent of hyperexcitable cortex needed to elicit FOS though remains unclear. A magnetoencephalography study revealed magnetic sources of FOS discharges involving parieto-occipital areas along with deeper brain structures such as the cingulate gyrus and the basomesial occipitotemporal region.¹¹ Functional MRI studies have also demonstrated similar widespread functional involvement with occipital FOS paroxysms correlating with activation of parieto-occipital and frontal lobe regions.¹² It is nonetheless postulated that in cases of symptomatic epilepsy, FOS occurs as a consequence of occipital hyperexcitability resulting from a structural brain lesion or from cortical deafferentation.⁶

Conclusion

This is a novel case where FOS or scotosensitivity was acutely observed in an adult patient with new-onset seizures and posterior cerebral infarcts likely reflecting a shared mechanism of posterior cortical hyperexcitability and deafferentation.

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Declaration of Conflicting Interests

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