# Focal occipital seizures with cerebral polyopia

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**ABSTRACT** – Polyopia is the visual perception of multiple images of a single visual stimulus. Cerebral polyopia has previously been described as an ictal phenomenon associated with temporal lobe seizures. We report the case of a man with multiple cavernous angiomas and occipital lobe seizures manifesting as cerebral polyopia.

Key words: cerebral polyopia, occipital lobe seizures, cavernous angioma

Occipital ictal events are often characterized by elementary visual hallucinations, blurring of vision or rarely ictal blindness (Sveinbjornsdottir and Duncan 1994). We report the case of a patient with cerebral polyopia as the primary manifestation of a simple partial occipital lobe seizure secondary to multiple cavernous angiomas.

## Case study

A 59-year-old man developed generalized tonic-clonic seizures at the age of 56 years. A brain MRI performed at that time showed multiple cerebral cavernous angiomas. The seizures were controlled with a combination of valproic acid and levetiracetam. The patient was then instructed to gradually decrease his levetiracetam dose with the goal of eventually continuing on valproic acid monotherapy.

Subsequently, the patient developed episodes of severe, left temporal headache, nausea, vomiting and polyopia. The episodes of polyopia lasted seconds to several minutes at a time and were characterized by up to six multicolored copies of the normal image in

his right hemi-field, which rotated along a vertical axis. There was no improvement upon closing either eye. His neurological examination was significant for right upper extremity dysmetria that coincided with the episodes of polyopia. The dysmetria resolved with eye closure. There was no associated nystagmus. However, he reported several bouts of emesis associated with these events. After the episodes, the patient complained of transient loss of vision in various parts of his right temporal visual field, lasting up to one hour. He had several, identical episodes during the day and returned to baseline in between events.

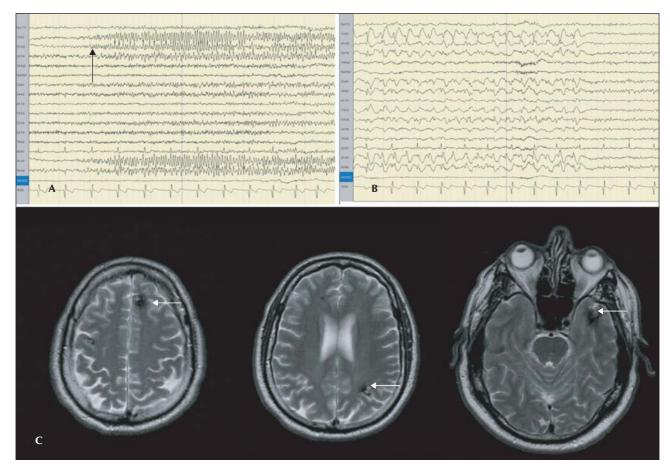
MRI of the brain revealed multiple cavernous angiomas without evidence of acute hemorrhage. Ictal EEG showed two electrographic seizure discharges in the left occipital region lasting 115 and 155 seconds respectively (figure 1) which were coincident with his experiencing polyopia.

The patient was restarted on his prior outpatient dose of levetiracetam in combination with valproic acid. He remained seizure-free at the 6 month follow-up.

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**Figure 1.** Ictal EEG showing two electrographic seizure discharges in the left occipital region. **A)** Electrographic seizure discharge originating in the left occipital region and characterized by high voltage rhythmic beta activity. **B)** Evolution of the seizure to a focal spike and wave discharge localized to the left occipital region. **C)** Axial, non-contrast T2 weighted brain MRI images. Arrows in each image point to the cavernous angiomas.

# **Discussion**

Typically, the most prominent symptoms in occipital lobe seizures involve elementary visual hallucinations (Sveinbjornsdottir and Duncan 1994, Lee et al. 2005). These may have either positive phenomena such as colors, shapes or flashes of light in the affected visual field, or may involve negative phenomena such as transient loss of vision. More complex visual hallucinations involving formed objects, movement, and vivid, complex visual experiences have also been described. Associated nonvisual symptoms can include forced eye blinking or postictal headache and vomiting with the latter symptoms often misdiagnosed as a migraine aura (Panayiotopoulos 1999). However, most reports of epileptic events involving the occipital cortex describe an initial visual event followed by spread to the visual association areas, the temporal lobe, as well as the vestibular cortices resulting in

skewed eye deviation (Galimberti *et al.* 1998), and the motor cortices with complex partial phenomena or generalized motor activity.

Polyopia is the visual perception of multiple images of a single visual stimulus (Miller et al. 1998). Cerebral polyopia is associated with parieto-occipital lesions, e.g. trauma, strokes, tumors, plaques (Bender 1945) and occipital lobe seizures (Sveinbjornsdottir and Duncan 1994). Cerebral polyopia is distinguished from palinopsia, which is the illusion of an image that persists following removal of the stimulus (Miller et al. 1998). Recently, polyopia was described as an ictal event occurring in relation to a neocortical temporal lobe lesion and more rarely as a manifestation of a mesial temporal lobe focus (Okada et al. 2004). Although the exact mechanism of this phenomenon is unknown, the authors postulated that the visual illusions may occur due to a dysfunction of memory storage and processing in the hippocampal and parahippocampal regions.

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To our knowledge, our patient is the first reported case of multiple cerebral angiomas associated with focal seizures and cerebral polyopia. However, our patient also had a cavernous angioma in the left temporal lobe, which could theoretically account for his symptoms. He also had a cavernous angioma in the left occipital lobe and the ictal EEG while the patient was experiencing polyopia revealed a discrete focal seizure with onset in the left occipital region. The cessation of events with administration of anti-epileptic medications further establishes polyopia as an ictal phenomenon in this patient. Hence, it is reasonable to postulate that multiple images of a single object may be generated by a dispersion of sensory input by epileptic activity occurring in the adjacent visual and association areas. The subsequent temporary visual field loss would then be analogous to a Todd's paralysis from neuronal exhaustion in the occipital cortex.

Oculo-vestibular stimulation can precipitate nausea and emesis in ways similar to chemical emetics and by direct alteration of gastro-intestinal motility (Zhang *et al.* 2001, Lang *et al.* 1999). It is possible that the illusion of multiple rotating objects may result in motion sickness by the same mechanism. Conversely, a delayed, slow spread of occipital epileptic discharges to the mesio-temporal limbic structures resulting in ictal emesis has been described (Guerrini *et al.* 1994).

Our patient's right-sided dysmetria, in association with the epileptic symptoms, may well have been due to an inability to distinguish between the true and false images on examination. A true ataxia was ruled out when the patient was able to perform the test adequately with his eyes closed.

In conclusion, this is the first clinical and electrophysiological description of a partial seizure, manifested as pure cerebral polyopia caused by a discrete occipital cerebral angioma. We suggest that occipital lobe seizures be considered in the differential diagnosis of cerebral polyopia.  $\square$ 

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