

## Case Report

# An Atypical Presentation of Epilepsy; What a Headache!

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## Abstract

The relationship between headache and seizure is a poorly understood and controversial topic; however, the literature has recently suggested that the two conditions may be related. The interplay between these conditions seems to be even more complex in a group of patients with epilepsy related headaches. It has been proposed that the association could be classified into preictal, ictal, postictal, or interictal headaches. Here we present a case report of a 62 year old male who presented with a chief complaint of new onset severe headache and subsequently underwent multiple diagnostic testing modalities before he was finally diagnosed and treated for epilepsy, which lead to the resolution of his headache. We conclude with a short discussion of how to subcategorize seizure related headaches based on their temporal relationship and why they can pose such a difficult diagnostic challenge.

## Keywords

- Epilepsy
- Headache
- Ictal epileptic headache

## ABBREVIATIONS

EEG: Electroencephalogram; CBC: Complete Blood Count; CMP: Complete Metabolic Panel; CT scan: Computerized Tomography Scan; CTA: Computed Tomographic Angiography; MRI: Magnetic Resonance Imaging

## INTRODUCTION

Headache and epilepsy are two very common neurological disorders that account for nearly 20% of all annual visits to neurologists [1]. Both are complex chronic disorders and tend to occur together more frequently than one would expect [2]. The classification of seizure related headaches has recently been a source of controversy in the literature; however, two methods have emerged for describing the association and they can be based on either the temporal relationship of the headache and seizure, or the type of headache associated with the seizure [3]. In terms of the temporal relationship, most recent studies have subcategorized these epileptic headaches into preictal, ictal, postictal, and interictal [3]. Here we illustrate a case report of a patient who presented with a chief complaint of headache and subsequently underwent multiple diagnostic testing modalities before he was finally treated for epilepsy, which lead to the resolution of his symptoms. We conclude with a short discussion of seizure related headaches and why they pose such a difficult diagnostic challenge.

## CASE PRESENTATION

The patient is a 62 year old male who presented to the emergency department with a new onset severe headache. He described it as the worst headache of his life and that it had been intermittent in nature since its onset the previous evening. He also reported accompanying nausea and confusion, but was

afebrile with a normal CBC and CMP. On arrival to the hospital he underwent a non-contrast head CT scan; however, no evidence of acute intracranial abnormality was seen. Additionally, a lumbar puncture was performed which was negative for xanthochromia and showed a protein count of 27, glucose 230, and 2 white blood cells. Upon completion of the initial diagnostic tests he was discharged home with a presumed diagnosis of cephalalgia and a prescription for acetaminophen.

Three days later the patient came back to the emergency department with the same complaints. He was having another severe headache, which was located only on the right side of the head. He stated that this headache was pulsatile and throbbing in nature, and associated with nausea. His head pain had never completely resolved from the previous visit and the acetaminophen prescribed to him helped minimally, if at all. There were no aggravating or relieving factors. Family members were present during this visit and stated that at times he seemed to be more forgetful and confused, especially after the pain peaked. They also stated that he urinated on the floor the previous day during an episode of severe pain. The patient denied any recent head trauma, fever, neck stiffness, rash, photophobia, or phonophobia. He denied any recent travel, exposure to animals, or ill contacts. Past medical history was positive for hypertension, hyperlipidemia, chronic back pain, and diabetes. His current medications included carvedilol, hydrocodone-acetaminophen, metformin, atorvastatin, and cyclobenzaprine. He has no past psychiatric history. Family history is positive for a cerebrovascular accident in his father at age 50, but negative for migraines or other neurological disorders. He has smoked a pack of cigarettes per day for the last 50 years.

During his second hospital visit neurology was consulted during an episode of severe head pain and examination revealed

decreased deep tendon reflexes, decreased movements on the right side of the body, and intermittent patchy visual field deficits more pronounced on the right than the left. With concern for posterior circulation ischemia the patient again underwent multiple diagnostic tests, including a CTA of the head which again showed no intracranial hemorrhage, and no evidence of a brain aneurysm or arteriovenous malformation. Carotid duplex ultrasound had shown no significant stenotic or occlusive disease and CTA of the neck showed no focal flow narrowing. MRI with and without contrast was also unremarkable. The infectious disease team was also involved during this visit due to concerns over viral encephalitis and he was prophylactically started on acyclovir.

Later on during the course of hospitalization, the patient complained of another severe headache and was witnessed having a fixed right gaze with nystagmus along with difficulty holding objects in the right hand. After the headache subsided he had intermittent confusion, echolalia, and emotional lability. EEG was ordered showing bifrontal organized slow waves and a presumptive diagnosis of partial complex seizures was made. A day later, continuous EEG then showed asymmetric 2-3Hz slow waves over the left frontotemporal region; however, the patient was confused and agitated and EEG was terminated early due to patient non-compliance.

Following the EEG study, acyclovir was discontinued and patient was started on IV levetiracetam. Unfortunately, he continued to have headaches associated with right eye deviation so oral phenytoin was added to his regimen. He was maintained on oral oxcarbazepine and phenytoin until his phenytoin level was therapeutic at which point no more neurological episodes were seen and the patient's headache resolved. He was then discharged with instructions to follow up with a neurologist, and he has not had a recurrence of his symptoms in over two years.

## DISCUSSION

The relationship between seizure and headache is a poorly understood and controversial topic; however, the literature has recently suggested that the two conditions may be related [4]. Membrane channel abnormalities and the imbalance of excitatory and inhibitory factors is the proposed pathogenesis, and multiple studies support the hypothesis that increased neocortical cellular excitability is the major underlying mechanism for both epileptic seizures and migraines [5]. It has been reported that the frequency of epilepsy in patients with migraine is about 17% higher than the frequency of epilepsy in the general population [6]. Moreover, the prevalence of migraine is 23% higher among patients with epilepsy compared to healthy individuals [6]. The interplay between the two conditions seems to be even more complex in the group of patients with epilepsy related headaches. It has been proposed that the association between these two neurological conditions can be classified into preictal, ictal, postictal, and interictal headaches [7]. Preictal headaches occur in 5-15% of cases, ictal in 3-5%, postictal in 10-50% of cases, and interictal in 25-60% [8].

When the headache precedes seizure activity it is referred to as a preictal epileptic headache. According to Cianchetti et al., the presence of a time interval of less than one hour between the

headache cessation and the onset of a seizure suggests a true preictal headache. A headache occurring within 3 hours after the cessation of a seizure is referred to as a postictal epileptic headache, which is the most frequent headache associated with seizures [2]. Inter-ictal headaches, especially migraines, are also very common in epileptic patients compared to the general population. Interictal headaches can present as various types of headache; however, their occurrence must be independent of a seizure, meaning that it does not fit the timing criteria presented for preictal or postictal headaches [9].

Ictal Epileptic headache represents a rare type of painful epileptic seizure in which there is no interval between headache onset and presence of epileptic discharges on EEG [2]. Recent case reports have documented that this type of headache can manifest with a plethora of symptoms, such as a sensation of bifrontal pressure, vague ache in the head, sharp stabbing retro-orbital pains or a sensation of electricity passing through the head of varying intensity, or just general discomfort [10]. Although headache is the predominant symptom, this type of seizure can occasionally be accompanied by other minor symptoms, which are typically vegetative [11]. This poses a diagnostic challenge, because only an EEG done during the headache can reveal the diagnosis, and epileptic form discharges are not always detectable by scalp electrodes. The current literature contains very few cases which have been successfully documented by EEG [2]. Parisi et al., have suggested four criteria for the diagnosis of this rare form of epilepsy that need to be simultaneously fulfilled for a diagnosis of ictal epileptic headache to be made. These include, as outlined in Table 1, duration of headache from seconds to hours, epileptic form discharges during the electroencephalogram with ipsilateral or contralateral focus with respect to the headaches location, epileptic form discharges on scalp EEG during a headache attack, and termination of the headache after intravenous administration of an anticonvulsant therapy [12-14].

Although the patient previously presented does not meet the diagnostic criteria set forth by Parisi et al., for ictal epileptic headache, we had strong electro-clinical evidence that our patient was suffering from seizures with the predominant presenting symptom being a headache. In a scalp EEG, ictal activity often stops abruptly, followed by postictal EEG depression or slowing [15]. In a study of focal onset seizures that included patients with temporal lobe epilepsy, postictal disturbances such as slow waves were found in 69 to 81% of seizures, with no changes

**Table 1:** Proposed criteria for Ictal Epileptic Headache as per Parisi et al., in Cephalalgia 2012 [12].

Diagnostic Criteria A-D must all be fulfilled to make a diagnosis of ictal epileptic headache
A. Headache lasting minutes, hours, or days.
B. Headache that is ipsilateral or contralateral to lateralized ictal epileptiform EEG discharges
C. Evidence of epileptiform discharges on scalp EEG concomitantly with headache; different types of EEG anomalies may be observed with or without photoparoxysmal response.
D. Headache resolves immediately after IV antiepileptic therapy.
<b>Abbreviations:</b> EEG: Electroencephalogram; IV: Intravenous

found in the remainder of the EEG [16]. Furthermore, ictal epileptic headaches have previously been found to have a variety of different EEG presentations, with some cases having none at all [17]. Our patient also responded to anticonvulsant therapy once therapeutic levels were achieved and his symptoms have been in remission since the onset of therapy. Although an epileptic etiology was inferred by our patients response to anticonvulsant therapy, recent studies have shown that migraines can also be responsive to certain types of anticonvulsant therapy [18]. We propose that further research is needed in order to clarify the relationships between headache and epilepsy, and how anticonvulsive therapy plays a role in treatment when the two are intertwined. Additionally, due to the technical difficulties involved in EEG testing as well as the fact that not all epileptiform discharges are detectable by scalp electrodes, perhaps the absolute requirement for EEG abnormalities during the episodes needs to be reconsidered if other evidence of seizure is present.

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