

**IJCRR**

Vol 06 issue 08

Section: Healthcare

Category: Case Report

Received on: 17/02/14

Revised on: 13/03/14

Accepted on: 05/04/14

## CHRONIC HEMIFACIAL SPASM - A MASQUERADER OF EPILEPSIA PARTIALIS CONTINUA

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### ABSTRACT

Hemi Facial Spasm (HFS) is commonly caused by compression of seventh cranial nerve by dolichoectatic arteries of posterior circulation though genetic and idiopathic causes to be considered<sup>1</sup>. Here we report a case of chronic HFS, A 55 years old female previously diagnosed to be HFS due to aberrant vascular loop compressing the facial nerve, on treatment for the past five years with no improvement in the symptoms. Patient EEG showed electrical activity in the right frontal lobe suggesting each spasm was due to partial seizure and diagnosis of Epilepsia Partialis Continua (EPC) was made. Patient symptoms resolved completely after starting newer anti convulsant medication suggesting that the facial spasms are due to EPC. Even though EPC is arising from cerebral cortex or sub cortical region it may present and to be mistaken as HFS - a peripheral movement disorder when it affects the face alone as in this case.

**Keywords:** Hemifacial spasm, Epilepsiapartialis continua

### INTRODUCTION

Hemi facial spasm represents myoclonus of the muscles innervated by seventh cranial nerve. As the name suggests it occur unilaterally but bilateral involvement can also occur rarely. Though the ephaptic transmission theory<sup>2</sup> and kindling theory<sup>3</sup> suggests facial nerve dysfunction as the cause of hemifacial spasm the compression of facial nerve by posterior circulation vessels is considered to be the wide accepted cause of hemifacial spasm. Here we report a case of hemifacial spasm which on evaluation showed epileptic discharge from right cerebral cortex as its cause which is unusual and reported very few in literature<sup>4-6</sup>.

### CASE REPORT

A 55-year-old-female was admitted and evaluated for an acute onset of Generalized tonic clonic seizure (GTCS) with post ictal confusion and 5 years history of facial twitching in left side (fig-1) with discomfort without pain. She had no prior

illnesses. No history of head injury, meningitis / encephalitis or other precipitating events. There were no other abnormalities on the neurological or general examination. Brain MRI performed before 5 years at the onset of spasms and showed aberrant vascular loop from posterior circulation causing compression over the exit of the facial nerve (fig-2) and started on clonazepam as patient was allergic to carbamazepine. In spite of therapy for one year patient symptoms didn't resolve and she underwent botulinum toxin injection. Patient did not improve and advised for micro surgical decompression of facial nerve at the site of compression but patient was not willing for surgery and continued on clonazepam and baclofen. Patient now presented with GTCS and evaluated for the same and repeat MRI brain did not reveal any new finding and EEG was taken which showed frequent epileptiform discharges from right frontal and temporal lobe (fig-3, 4) for every facial twitching which the patient had

experienced and discharges from both the cortex when patient had GTCS suggesting secondary generalized seizure. Patient was allergic to carbamazepine, newer antiepileptics like levetiracetam and lacosamide provided almost complete resolution of facial twitching. When she temporarily discontinued the drugs due to sleepiness there was subsequent recurrence of these abnormal movements. The diagnosis of Epilepsia partialis continua was confirmed by the finding of irregular epileptiform discharges from right cerebral hemisphere and rhythmic / semirhythmic focal slowing during prolonged video / EEG monitoring while the patient was experiencing Hemi facial spasm.

## DISCUSSION

Epilepsia partialis continua manifest as focal motor clonic seizures, which remain localized to the part of the body in which they originate (face / limbs) and the motor activity is persistent lasting for minute, hours, days, weeks or even years together<sup>7</sup>. EPC can be considered the status epilepticus equivalent of simple partial motor seizures<sup>7</sup>. In most cases of EPC the seizure focus lie on cerebral cortex eventhough subcortical foci have also been reported<sup>8</sup>. Hemi facial spasm (HFS) is defined as unilateral, involuntary, irregular, clonic or tonic movement of facial muscles innervated by seventh cranial nerve.. Although most cases of hemifacial spasm are idiopathic and probably caused by vascular compression of facial nerve other etiologies like bells palsy, facial nerve injury by demyelination, vascular insult and hemifacial spasm mimickers should be considered. The hemifacial mimickers are classified as psychogenic, tics, dystonia, myoclonus and hemi masticatory spasm<sup>9</sup>. In our case the mimicker has been identified as EPC.

The ictal etiology for hemifacial spasm is evident from our case by the presence of electrical activity from the right frontal lobe for each facial twitching witnessed when EEG was taken and the prompt response to the antiepileptic drugs. It is further

substantiated by the recurrence of twitching on withdrawal of the drug. The presence of aberrant vascular loop causing compression of the facial nerve made the previous clinician to think and treat as hemifacial spasm caused by vascular compression of facial nerve as it is the most widely accepted cause of hemifacial spasm. But the presentation of hemifacial spasm with GTCS made us to evaluate for seizure and found that epileptic discharge from the right frontal lobe (partial seizure) present as facial twitching of the left side of face which is mistaken as hemifacial spasm due to clinical similarity and by the anatomic finding of aberrant vascular loop causing compression of the facial nerve. Since this partial seizure in our patient was persistent for 4 years it is considered as Epilepsia partialis continua.

The EPC nature of HFC reported already in literature<sup>10</sup> has a normal facial nerve course. In our case the patient had vascular compression of facial nerve and the cause for the facial twitching was not because of the compression but because of the Seizure activity is evidenced by the response from anticonvulsant medication and failure to respond for botulinumtoxin injection which is the commonest treatment for HFS of vascular compression of facial nerve<sup>11</sup>. Hence we hypothesize and emphasize that the prompt response of this patient with chronic HFS to newer antiepileptic drugs possibly indicate that HFS can occur sometimes as a manifestation of EPC with focal epileptiform discharge from the brain rather than due to facial nerve hyperexcitability.

## CONCLUSION

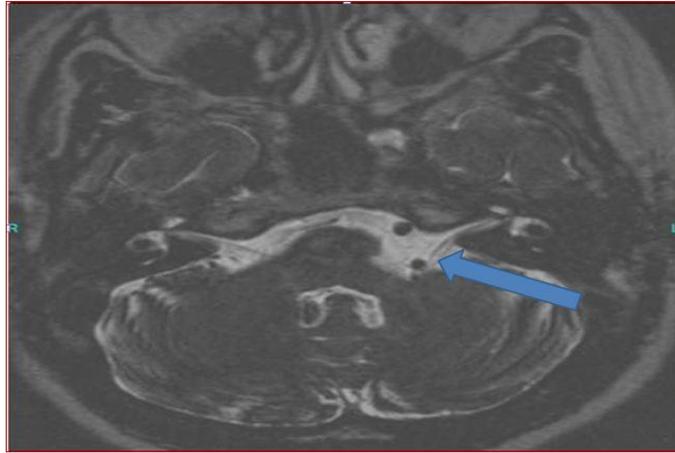
We conclude that, Even though Hemifacial spasm is of neuropathic or idiopathic of origin, this case report should alert the physicians about the seizure activity from the cerebral cortex presenting as hemifacial spasm and EEG should be advised for all the cases of HFS irrespective of the MRI finding.

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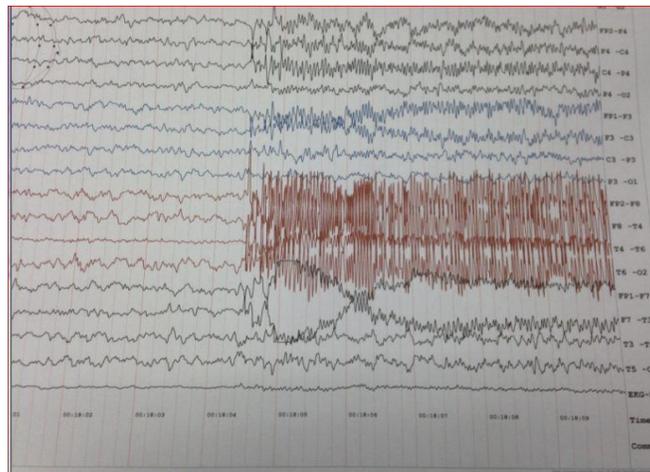
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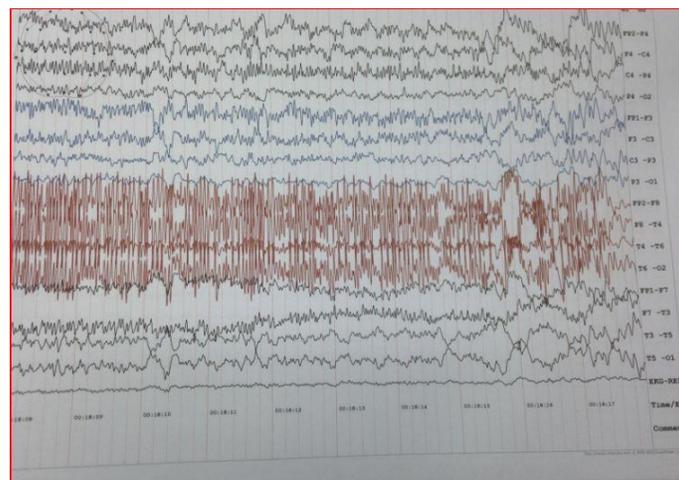
**Figure 1 Showing hemifacial twitching on left side**



**Figure 2 showing an aberrant vascular loop from posterior circulatoron causing compression over the exit of the facial nerve on left side**



**Figure 3: EEG showing epileptiform discharges from right frontal and temporal lobe for every hemifacial twitching the patient experienced.**



**Figure 4: Another EEG tracing showing epileptiform discharges from right frontal and temporal lobe for every hemifacial twitching the patient experienced**