

A Rare Case Presentation of Somatosensory Reflex Epilepsy

Krupa Torne* and Nita Sutay

Department of Paediatrics, Division of Paediatric Neurology, Sir JJ Hospital and Grant Government Medical College, India

***Corresponding Author:** Krupa Torne, Department of Paediatrics, Division of Paediatric Neurology, Sir JJ Hospital and Grant Government Medical College, India.

Received: September 11, 2017; **Published:** October 09, 2017

Abstract

Objective: 10 year old boy presented with 2 years of multiple episodes of seizures precipitated by stimulus with weakness of left upper and lower limb since 1 year of age. He was started on Carbamazepine to which he responded completely in 3 - 4 weeks.

Material: He was born at 35 weeks of gestational age with birth weight 1.75 kilograms. His perinatal course was uneventful. He had first seizure at age of 15 days which was associated with fever. He had a second afebrile seizure at 7 month of age. No further episodes were noted until 8 years of age. He was treated with Sodium Valproate, Clobazam, Levetiracetam with poor response. On examination he had microcephaly, global developmental delay. His power on the left side was less than right side with tone more in left than right side.

Methods: MRI Brain showed significant volume loss of entire right hemisphere with ex vacuo dilatation of right lateral ventricle with cystic lesion in right fronto temporal lobe. EEG showed significant low amplitude response from right anterior area. His IQ noted to be (70) indicating mild intellectual disability.

Result: After appropriate investigations he was diagnosed with somatosensory reflex epilepsy. He was given appropriate trial of antiepileptic (Carbamazepine) in adequate dosages to which he responded completely over next 3 to 4 weeks.

Discussion: We describe a rare case of somatosensory reflex epilepsy triggered by touch manifested in the form of tonic seizures which responded completely to Carbamazepine. The epilepsy can be provoked by external (light, touch, sound) or internal stimulus (reading, thinking). They are triggered by activation of primary sensory cortex. Reflex epilepsies can be acquired or inherited. Acquired reflex seizures may be a result of acquired cerebral lesions like stroke, encephalitis, or cortical dysplasia. Clinically it can present as generalised, partial, absence or myoclonic seizures. Treatment hinges on limiting exposure to the provoking stimulus and using appropriate medications.

Conclusion: Somatosensory reflex epilepsy is a rare condition and can be treated successfully with appropriate antiepileptics and by avoiding trigger factors.

Keywords: Somatosensory Reflex Epilepsy; Carbamazepine; Seizures

Objective

10 year male child born of non-consanguineous marriage presented with weakness of left upper and lower limb since 1 year of age and multiple paroxysmal episodes precipitated by giving stimulus since last 2 years prior to presentation. He was started on Carbamazepine to which he responded completely in 3 - 4 weeks.

Material

He was born at 35 weeks of gestational age with birth weight of 1.75 kilograms. His perinatal course was uneventful. He had first generalised tonic clonic seizure on day 15 of life which was associated with fever. He had second afebrile seizure at 7 month of age. No further episodes were noted until 8 years of age. Weakness of left upper and lower limbs noticed by mother at 1 year of age when child started to sit which was non-progressive.

Child was apparently alright 2 years back before presentation to our hospital, when he had multiple paroxysmal episodes characterized by tonic movements of both upper and lower limbs, more on left side lasting for 1 - 2 min. It was associated with deviation of eyes to left side with bladder incontinence. Multiple such episodes occurred during a day and were provoked by sensory stimulus. No history of post ictal drowsiness, head injury, altered sensorium, headache giddiness or loss of consciousness. Before coming to our hospital He was treated with Sodium Valproate, Clobazam, Levetiracetam then with poor response.

On examination noted to have microcephaly with global developmental delay. No evidence of neuro cutaneous markers was seen. His power of the left side was less than right side with tone noted to be more in left side than right side. He was started on Tablet Carbamazepine and Tablet Sodium Valproate, Levetiracetam and Clobazam were tapered gradually. Child responded well to the treatment. He was discharged on Tablet Carbamazepine at 30 mg/kg/day as he had no further repeat episodes of seizures.

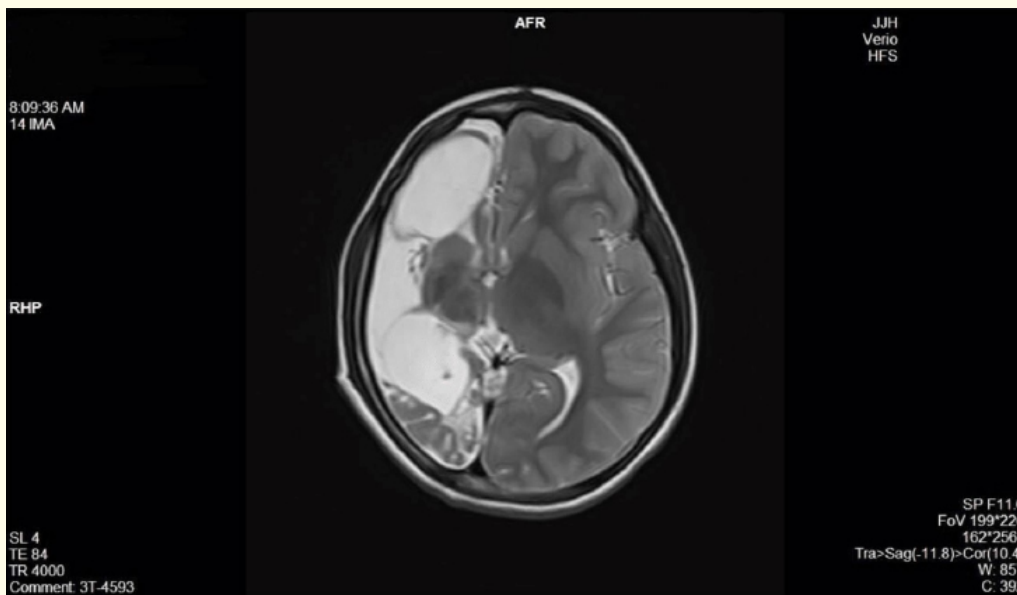


Figure 1

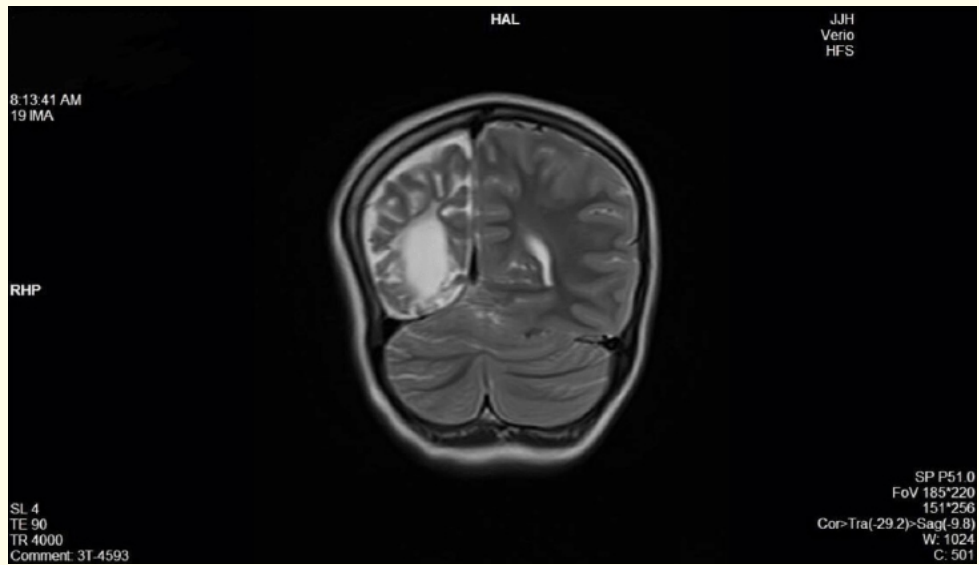


Figure 2

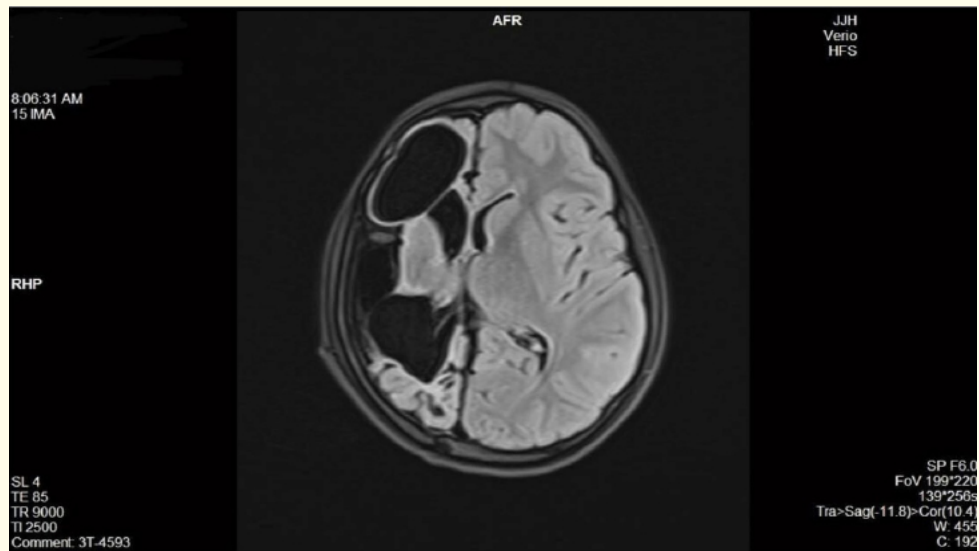


Figure 3

Methods

MRI Brain showed significant volume loss of entire right hemisphere with ex vacuo dilatation of right lateral ventricle with cystic lesion in right fronto temporal lobe. Few well defined large cystic lesions occupying entire right frontal and temporal lobe with surrounding

gliotic changes. Significant volume loss of entire right hemisphere with ex-vacuo dilatation of right lateral ventricles. EEG showed focus from right anterior hemispheric area with significant low amplitude response from right hemispheric area. His IQ noted to be (70) indicating mild intellectual disability.



Figure 4

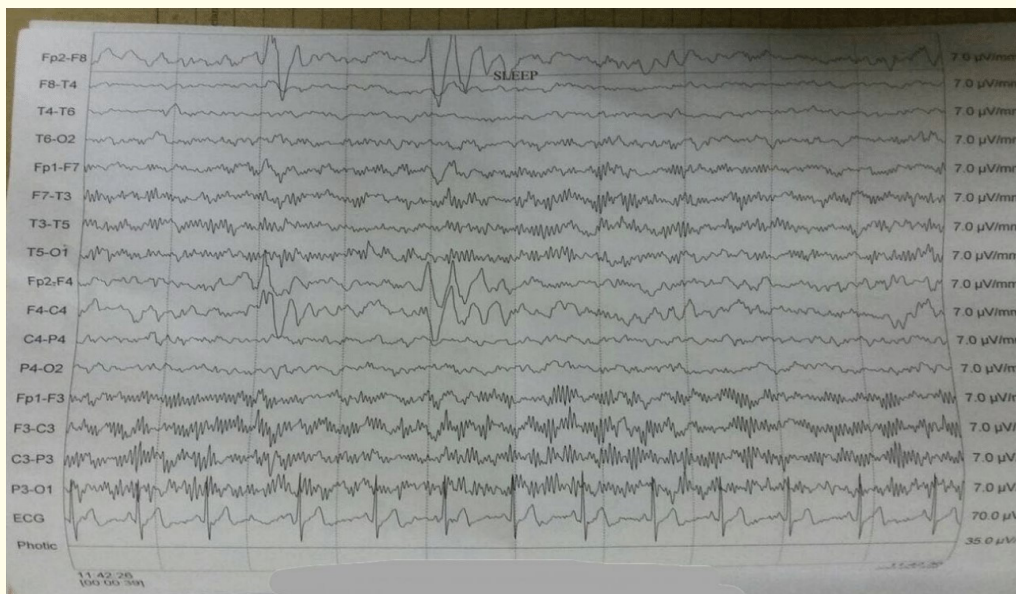


Figure 5

Result

After appropriate investigations he was diagnosed with somatosensory reflex epilepsy. He was given appropriate trial of antiepileptic (Carbamazepine in adequate dosages to which he responded completely over next 3 to 4 weeks.

Discussion

Reflex epilepsy is a condition in which seizures can be provoked by either external stimulus (light, touch, sound) or internal stimulus (reading, thinking). Clinically can present as generalised, partial, absence or myoclonic seizures. Most common triggers are visual somatosensory, auditory, and olfactory stimuli. It is relatively rare condition, occurring in only 5% of all epilepsies. Reflex epilepsies can be acquired or inherited. Acquired reflex seizures may be a result of acquired cerebral lesions. The most common etiologies are stroke, encephalitis, or cortical dysplasia. Common manifestations of acquired reflex epilepsy are startle seizures, expressed as sudden myoclonic or tonic contraction of truncal and extremity musculature.

Inherited reflex seizures are a result of several genetic factors those may be channelopathies or those affecting brain development like neurodegenerative disorders.

Following triggers may cause reflex epilepsy. Visual stimuli such as flickering of light, removal of visual fixation or light intensity, complex visual patterns. Somatosensory stimuli like light touch, tapping or immersion in hot water and Auditory stimuli like complex actions or mental processes like reading, eating, micturition, tooth brushing etc. also can cause reflex epilepsy.

Reflex seizures are triggered by activation of primary sensory cortex, such as primary auditory or somatosensory cortex and also by activation of pre motor, paracingulate and parietal lobe association cortices. Triggering factors induce epileptic discharges from these areas which lead to seizures.

Diagnosis can be made by EEG, Video EEG and neuro imaging.

The treatment of reflex seizures hinges on limiting exposure to the provoking stimulus and using standard antiepileptic drugs. The choice of particular antiepileptic drug is guided by many considerations like electro clinical seizure type, prior treatment history, patient's age, co-morbidities, and adverse effects. Valproic acid monotherapy has a success rate of 73 - 86% in patients with photosensitive epilepsies. Levetiracetam, Lamotrigine, Ethosuximide, and Topiramate are recommended as 2nd choice. Patients with partial seizures benefit from Carbamazepine or Oxcarbazepine [1-5].

Conclusion

Somatosensory reflex epilepsy is a rare condition and can be treated successfully with appropriate antiepileptics and by avoiding trigger factors.

Bibliography

1. Rudolf G., *et al.* "Genetic reflex epilepsies". Orphanet Encyclopedia (2004).
2. Andermann E and Straszak M. "Family studies of epileptiform EEG abnormalities and photosensitivity in focal epilepsy". In: Seino M, Kazamatsuri H, Ward A (eds) *Advances in Epileptology: 1st Epilepsy International Symposium*, New York, Raven Press (1982): 105-112.
3. "Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised classification of epilepsies and epileptic syndromes". *Epilepsia* 30.4 (1989): 842-849.

4. Gastaut H and Tassinari CA. "Triggering mechanisms in epilepsy: the electroclinical point of view". *Epilepsia* 7.2 (1966): 85-138.
5. Matsuoka H, *et al.* "Neuropsychological EEG activation in patients with epilepsy". *Brain* 123.2 (2000): 318-330.

Volume 8 Issue 3 October 2017

©All rights reserved by Krupa Torne and Nita Sutay.