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Case Report

Focal Cortical Dysplasia-A Case Report

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Abstract

Focal cortical dysplasia (FCD) is a congenital abnormality of brain development where the neurons in an area of the brain failed to migrate in the proper formation in utero and is one of the important cause of intractable epilepsy in children and in adults. We are presenting a 11 years old female patient with intractable seizures since the age of 1 ½ years, Extra temporal complex partial in nature, on evaluation MRI brain showed Type IIb Focal Cortical Dysplasia (FCD), PET CT showed marked hypo metabolism in Right insular cortex and video EEG showed few epileptiform activity during wakefulness over Right fronto temporo parietal regions. Epilepsy committee meeting concluded to proceed with resection of Right frontal opercular FCD under EcoG monitoring with neuro navigation, IONM & image guidance. Patient underwent Right Pterional craniotomy and Micro surgical resection of the Right frontal opercular FCD under Monitoring. HPE suggested as Focal Cortical Dysplasia ILAE/PALMINI type IIB. Post operatively, on three months followup patient was seizure free.

Keywords: Focal Cortical Dysplasia (FCD), Intractable seizures, Microsurgical Lesionectomy.

Introduction

Focal cortical dysplasia (FCD) is a congenital abnormality of brain development where the neurons in an area of the brain failed to migrate in the proper formation in utero and most common cause of intractable epilepsy in children and in adults. There are three types of FCD with subtypes, including type 1a, 1b, 2a, 2b, 3a, 3b, and 3c, each with distinct histopathological features [1].

Type 2b FCD exhibits complete loss of laminar structure, and the presence of CDN and enlarged cells are called balloon cells (BC) for their large elliptical cell body shape, laterally displaced nucleus, and lack of dendrites.

Case Report

11 year old female patient presented with history of seizure since 1 ½ years of age. Each episode lasted for 5-10 seconds with Behavior arrest, staring look & left UL posturing suggestive of Extra temporal complex partial seizures.Initially the episodes were one per week and was managed with Sodium Valproate & Clobazam till 7 years & stopped. she remained Seizure free for 2 years. At the age of 9 years she again developed seizures and was started on Tab. Oxetol 150 mg and Tab. Lamiectal 25 mg twice dailyInspite of the medications she developed refractory seizure 2 episodes per week and subsequently almost on daily basis. The initial MRI Brain (1.5T) was done in 2011 didn't show any obvious abnormality and EEG was also normal.

In 2019 MRI Brain (3T) with seizure protocol was done showed Right frontal operculum adjacent to sylvian fissure shows focal ill define cortical thickening with mild T2 hyper intense signal, underlying indistinct gyral white matter junction and sub cortical ill defined T2 hyper intense signal consistent with Type II b focal cortical dysplasia (FCD) 1.6*1.2*1.6 cm, Fused PET with screening MRI (FLAIR) shows T2 Flair hyper intense right sided insular corti-

cal thickening with trans mantle sign confirmed the lesion to be in Right insula (Figure 1).

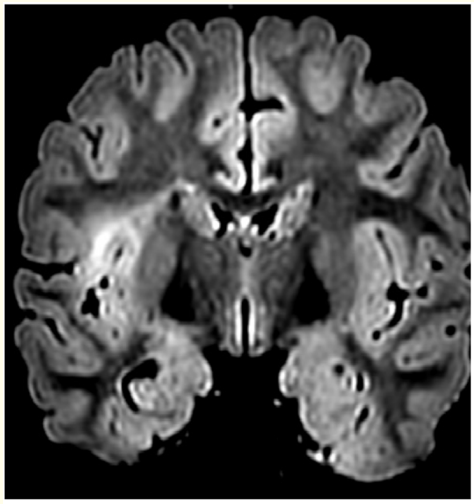

Figure 1: MRI Brain (3T) with seizure protocol was done showed Right frontal operculum adjacent to sylvian fissure shows focal ill define cortical thickening with mild T2 hyper intense signal, underlying indistinct gyral white matter junction and sub cortical ill-defined T2 hyper intense signal consistent with Type II b focal cortical dysplasia (FCD) 1.6*1.2*1.6 cm.

Functional MRI (fMRI) showed the language function in left hemisphere. Diffusion Tensor Imaging (DTI) (Figure 2) showed part of Cortico spinal tract (CST) coursing very close to mantle of FCD.Video EEG shows few epileptiform activity during wakefulness over right fronto-parieto-temporal regions and activation of multifocal epileptiform discharges during sleep over midline frontal, bifrontal and right fronto temporal region. Four complex partial seizure of probable right hemispheric origin was recorded.

PET CT Brain shows Asymmetrical marked Hypo-metabolism in right insular cortex (asymmetrical index (AI) of – 54.57%. Neuro psycologial assessment showed impaired visuo conceptual and visuo perceptive memory suggestive of Right hemisphere abnormality.Epilepsy committee meeting concluded to proceed with Resection Right Frontal Opercular FCD under Ecog monitoring with Neuro Navigation, IONM & Image guidance.

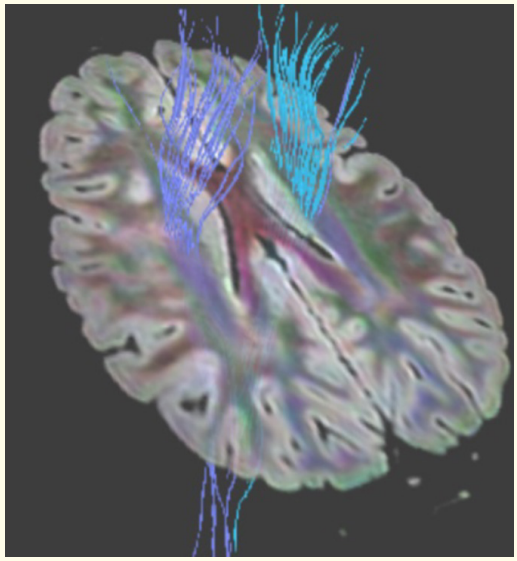

Figure 2: Diffuse tensor Imaging.

Surgery

Right Pterional craniotomy and Resection the Right Frontal Opercular FCD under Ecog monitoring with Navigation, IONM & image guidance.

- Right pterional craniotomy was done with navigation assistance (Medronic Stealth Station S8). Neuro monitoring was done by placing scalp electrodes. The modalities tested were EcoG, sub cortical motor fibres mapping, Motor evoked potential (MEP) and Somato sensory evoked potentials (SSEPs).
- After opening the sylvian fissure, the frontal operculum was exposed and ill defined abnormal gyri was seen and with navigation assistance, was confirmed to be the site of FCD. The lesion was resected with EcoG guidance. Following resection of insular lesion, the mantle was localised with neuro navigation and the same also excised carefully monitoring the subcortical motor fibres.
- Then the patient underwent intra operative MRI (3T) which showed residual FCD in the inferior lip of Right frontal operculum. She was brought back to operating room and the residual FCD was excised. EcoG remained silent after the Lesionectomy. MEP and SSEPs were intact throughout the procedure.
- Postoperatively patient developed transient weakness of left upperlimb and lower limb (4-/5) which recovered after a week. She remained seizure free and she was discharged on 5th POD. She remains seizure free on 3 months followup.

- HPE shows dysmorphic neurons,"balloon cell" (formerly known as Taylor cells),Mitotic activity is virtually negative and Increased astroglial cell density suggested as FOCAL CORTICAL DYSPLASIA, ILAE / PALMINI TYPE IIB.

Discussion

- Focal cortical dysplasia (FCD) is a congenital abnormality of brain development where the neurons in an area of the brain failed to migrate in the proper formation in utero and most common cause of intractable epilepsy in children and in adults. Gene mutations associated with FCD2a and FCD 2b include *MTOR, PI3KCA, AKT3,* and *DEPDC5*.Mutations in these genes lead to enhanced mTOR pathway signaling at critical periods in brain development [2].
- Medication is used to treat the seizures that may arise due to cortical dysplasia. If anticonvulsants fails to control seizure activity, Lesionectomy should be an option for FCD. Care has to be taken to remove the mantle to prevent status epilepticus in post op period.

Conclusion

Micro surgical lesionectomy to be considered in drug resistance refractory seizure in patients with FCD. Adequate pre operative work up with optimal utilization of new adjuvants like IONM and Image guidance will greatly help in improving the surgical results and thereby achieving seizure freedom.

Bibliography

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