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Dissociative Head and Eye Movement during Seizure: A Case Report

Soheila Rezakhani [1*], MahmoodMotamedi[2]

1. Department of Neurology, Kerman Medical University, Kerman, Iran
2. Department of Neurology, Tehran Medical University, Tehran, Iran

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***Corresponding author:**

Soheila Rezakhani. Department of Neurology, Kerman Medical University, Kerman, Iran

Phone: +983432810917

Fax: +983432810917

Email: drrezakhani@gmail.com

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Abstract

Introduction: Deviation of the eyes may occur with seizures involving any cerebral lobe. Most notably, a lesion of the FEF that causes excess neural activity, like a focal seizure, or spreading of ictal discharges to the FEF will drive the eyes contralaterally during the period of the seizure. The head also may turn contralateral to the seizure focus during the ictus. In the post-ictal state, when there may be lingering hypoactivity of the FEF neurons, the eyes may deviate ipsilateral to the side of the lesion because of a relative increase in the input from the unaffected FEF on the opposite side of the brain.

Methods: A 14- year old right handed boy with refractory epilepsy was admitted for video EEG monitoring in an attempt to better define his seizures and to evaluate him for possible epilepsy surgery. He had thirteen clinical seizures with different semiology. In one of these, he had disconjugated head and eye movement that is an unusual finding. In this investigation, we noted his brain MRI with and without contrast that reported focal signal abnormality in the right prerolandic region indicative of AV malformation and also abnormal intraaxial infratentorial cystic mass located in the left cerebellar peduncle.

Results: In the present case, disconjugated head and eye movement occurred during the seizure, probably; due to brain stem cystic lesion that is mainly located in mesencephalic area that may interrupt the connections between FEF and superior colliculus.

Conclusion: The patient studied in the present study experienced Rt. Side (ipsilateral) eye gaze with dis-conjugated left sided (contralateral) head version during his seizure. While long term video-EEG monitoring and imaging studies pointed to epileptogenic zone of right centroparietal region. This is probably due to a cystic lesion located in the mesencephalic region mainly in the left cerebellar peduncle.

Keywords: Disconjugated Head and Eye Movement, Seizure, Frontal Eye Field

Introduction

Deviation of the eyes and head is regularly detected in epileptic seizures. There has been a controversial discussion on the lateralizing and localizing values. Head turning related to seizure- can manifest as forced unnatural tonic head version (پversionپ), mostly followed by ipsilateral turning of body and eyes or as smooth head turning like voluntary movements (پhead deviationپ) (1). Versive seizure, defined as extreme and sustained conjugate movements of eye with lateral movements of body and head can happen in partial epileptic seizures. پversive seizureپ, Contraversive epileptic eye deviation, is a typical frontal lobe seizure involving frontal eye field by epileptic stimulation (1, 2). Additionally, it is one of the precious semiological symptoms for epileptogenic area lateralization (3, 4). The localizing and lateralizing importance of deviation of ictal eye, even the versive type, in partial epileptic seizures has been discussed by medical

observations from some focal epilepsy papers concentrating on the lateralization, especially because the deviation of epileptic eye may be contralateral or ipsilateral to the electroencephalography (EEG) focus, and has been correlated with EEG focal manifestations or neuroimaging evidence from parietal, central, frontal, temporal, and occipital zones (5-7). Since the basic causes of these variations may be highly complex, various pathophysiologies should be considered during seizures in eye movement variations; however, the head and eye versions conjugated in epileptic seizures. The FEF affects saccade generation via four main pathways: (1) a pathway via the basal ganglia through the ipsilateral striatum, (2) a projection to the ipsilateral superior colliculus concentrated in the intermediate layers, (3) projection to the cerebellum through the pontine nuclei, and (4) a weaker projection to pontine and mesencephalic nuclei making up the saccade generator circuit. FEF is innervated by the thalamus nuclei, bordering the internal medullary lamina, largely the medial part of the ventroanterior nucleus and the lateral part of the mediodorsal nucleus. The thalamic areas connected to the FEF are innervated by oculomotor afferents from the middle layer of the substantia nigra pars reticulata, the superior colliculus, and the dentate nucleus of the cerebellum (8).

Methods

A 14-year-old right handed boy was admitted for video EEG monitoring in an attempt to better define his seizures and to evaluate him for doing possible epilepsy surgery. The patient's first seizure occurred at the age of four. Initial seizures for the first couple of years were characterized by Lt. sided tonic posturing, lasting about 40 seconds with obvious impairment of consciousness. Now, his seizures are characterized by sudden awakening, tonic posturing of upper limbs, and flushing of the face with obvious impairment of consciousness. At times, these seizures are followed by generalized tonic clonic activity. Frequency of these minor motor seizures is about 1 to 2 per week, but major motor seizures occur rarely. These seizures often get worse by stress and anger and are more common during sleep especially at nights. The patient is the product of a cesarean gestation. Birth was spontaneous at 9 months gestation without any complication. The early development was normal; he walked by the age of 1 and could talk fluently at the age of two. He had no history of febrile seizures, CNS infection, or CNS trauma and no family history of epilepsy. He had also no history of tobacco, alcohol, or illicit drug use. The patient lives with his parents and siblings. At the moment, he studies at an intermediate school, and he has no job. Previous brain MRI reported abnormal signal in the right Para hippocampal gyrus and VR space in the cerebral peduncle measuring 10*10*13.5mm. Brain MRS from brain stem lesion showed no increased peak of Cholin, but normal peak of NAA and increased lactic acid and lipid mostly VR space. There was no evidence of neoplasm. The latest brain MRA and MRV (Haghighat medical imaging center) were normal. Latest brain MRI with and without contrast reported focal signal abnormality in the right prerolandic region. Itshowed tubular signal void configuration in the T2W and SWI sequences and represented signal void abnormality. In postcontrast images, abnormal enhancement was observed in the mentioned focus with vascular tortuse configuration typical of AV malformation accompanying peripheral brain parenchymal gliosis and hemosiderin deposition. There was abnormal intraaxial infratentorial cystic mass located in the left cerebellar peduncle (its size was about 15*17 mm in diameter). In the corresponding postcontrast images, associated enhancement or solid component was not seen. His prior medication trials were Carbamazepine, Depakine, Lamotrigine, Topiramate, Levebel, and Phenobarbital. He has been currently taking Tegretol 400 mg TDS, Phenobarbital 100 mg QHS, and Acetazolamide 250 mg BD. Routine labs, including FBS, Ca, P, alkaline

phosphatase, SGPT, SGOT, and U/A, were within normal limits. VDRL was negative. The patient was alert and oriented to time, place, and person. His mental state was within normal limits. Visual fields were intact to confrontation. Pupils were equal and reactive to light with extraocular movements intact. There was no facial sensory or motor asymmetry. Palate and uvula rose symmetrically, and the tongue protruded on midline. Force and tone assessments were normal. Reflexes were 2+ and symmetric. Cutaneous plantar reflexes were in flexion. Cerebellar functions including finger-to-nose and heel-to-sheen testing and gait and tandem walking were normal. A video EEG monitoring session was scheduled using modified international 10-20 system. Silverman's true anterior temporal electrodes (T1, T2), and T9, T10 were also applied. Recorded EEGs were reviewed in bipolar and referential montages using reformatting.

Computerized spike and seizure detection were employed. Acetazolamide was discontinued, and Tegretol was slowly tapered. To provoke his habitual seizures, sleep deprivation was also performed from the third night of his admission.

Results

Interictal findings

The basic rhythm of his resting records consisted of medium amplitude 9 CPS alpha activity attenuated to eye-opening. Abnormal interictal findings are as following:

1. Intermittent theta and delta activity of Central region (C4>C3)
2. Intermittent theta and delta activity of left temporal region (T3, T5)
3. Sharp Lt. temporal region (T1, T3)
4. Sharp Lt. anterior temporal region (F7)
5. Bifrontocentral spike (F3, C3, F4, C4)
6. Sharp frontal region (F4>F3)

Frequent slow waves were noticed on right centroparietal leads (C4, P4). Bursts of diffuse spike-wave and sharp and slow waves were noticed on his resting records, but they were very prominent on right centroparietal leads. Discrete sharp and slow wave complexes were also observed on right centroparietal leads (C4, P4). The epileptiform discharges were also demonstrated on his sleep records.

Ictal findings

The patient had a total of 13 clinical seizures with different semiology consisting of startle seizure, bilateral asymmetric tonic seizure, Gelastic seizure, and Lt. arm tonic seizure (LOA) with postictal Lt arm paresis. In one of his seizures, three seconds after the beginning of EEG ictal pattern and two seconds after startle seizure (at 15:19:23), his head turned to the left side and about one second later he had Rt. eye gaze. At 15:19:27, he had full left head turn, and right eye gaze. After one second, he had bilateral asymmetric tonic seizure. Finally, his seizure ended at 15:19:37. This seizure lasted about 14 seconds. Right arm and leg postictal Todd's paresis was noticed in this seizure.

Discussion

Orienting the movements of the head and eyes, the superior colliculus (SC) is connected reciprocally to the mesencephalic reticular formation (MRF), demonstrating that the latter contributes to gaze control. The MRF has been subdivided provisionally to stimulate a rostral and caudal portion, subserving vertical and horizontal gaze, respectively. Although there is notable knowledge on the circuitry controlling movements of eye during gaze variations, our perception of the circuits monitoring the components of head is not comprehensive (9- 11). Thesuperior colliculus (SC) is the main brainstem center for measuring a gaze change vector. Using preparations in which the head is unrestrained, SC gaze signals orient head and eyes movements (12). The major gaze related outflow of the SC reaches the upper cervical cord through the crossed tectobulbospinal or predorsal bundle pathway (13). However, the major control of head movements related to gaze is within the rostromedial medullary reticular formation (MdRF). The electrical

stimulation of the paramedian part of the gigantocellular medullary reticular formation between the abducens and hypoglossal nuclei levels generates movements of head similar to those observed with gaze variations (14). This zone has reticulospinal neurons which receive collicular inputs and fire before movements of head in connection with gaze variations. Although the collicular pathways projecting indirectly and directly to the upper cervical spinal cord render an essential substrate for tectally-initiated movements of head, the eye and head interactions are complicated and can change by a gaze change context (15). In addition, the steps needed to change the spatial code for the movement of gaze in the SC to the temporal code through which motor neurons orient the musculature have to be completely identified (13). Even at the neck musculature, the activity of different muscles is monitored in a complicated manner, owing to the plenty of joints and the large mass of head (10). The mesencephalic reticular formation (MRF) is endowed well with tectal terminals among structures which receive collicular input. MRF has been subdivided into a rostral portion near (peri) the interstitial nucleus of Cajal (piMRF), involved in the vertical component of gaze variations, and a caudal portion, the central mesencephalic reticular formation (cMRF), contributing to the changes of horizontal gaze. Many neurons in the MRF demonstrate firing features similar to those of collicular or paramedian pontine reticular formation (PPRF) neurons. Collaterals of the predorsal bundle axons extensively terminate in the ipsilateral MRF.Lesions in or chemical inactivation of the MRF damages the precision of gaze variations and changes the position of head (16). Accordingly, probable cause of this disconjugated head and eye movement is possibly a cystic lesion located in mesencephalic region mainly in left cerebellar peduncle.

Conclusion

The patient studied in the present study experienced Rt. Side (ipsilateral) eye gaze with dis-conjugated left sided (contralateral) head version during his seizure. While long term video-EEG monitoring and imaging studies pointed to epileptogenic zone of right centroparietal region. This is probably due to a cystic lesion located in the mesencephalic region mainly in the left cerebellar peduncle.

Ethical issues

Not applicable.

Authors' contributions

All authors equally contributed to the writing and revision of this paper.

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