A Case of Habitual Neck Compression Induced Electroencephalogram Abnormalities: Differentiating from Epileptic Seizures Using a Tc-99m HMPAO SPECT

Hongyoon Choi · Min-Seok Seo · Ho-Young Lee ·
Young-Soo Kim · Chang-Ho Yun · Sang Eun Kim ·
Sung-Ho Park

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Abstract Self-induced hypoxia has been reported particularly in adolescents, and it can result in neurological injury. Here, we present a case of electroencephalogram (EEG) abnormalities induced by habitual neck compression differentiated from epileptic seizures by Tc-99m HMPAO SPECT. A 19-year-old male was admitted for evaluation of recurrent generalized tonic-clonic seizures. No interictal EEG abnormality was detected; however, abnormal slow delta waves were found immediately after habitual right neck compression. To differentiate EEG abnormalities due to a hemodynamic deficit induced by habitual neck compression from an epileptic seizure, Tc-99m HMPAO SPECT was performed immediately after right carotid artery compression. Abnormal delta waves were triggered, and cerebral hypoperfusion in the right internal carotid artery territory was detected on Tc-99m HMPAO SPECT. The slow delta wave detected on the EEG resulted from the cerebral hypoperfusion because of the habitual neck compression.

Keywords Habitual neck compression · Tc-99m HMPAO SPECT · Electroencephalogram · Seizure

Introduction

Self-induced hypoxia has been recognized particularly in adolescents. Most cases happen because of “the choking game” played to achieve a euphoric state by cerebral hypoxia [1]. Self-induced hypoxia can result in neurological injury with variable clinical manifestations [2]. Previously, in a case of self-strangulation caused by the choking game, the patient presented recurrent seizure-like events [3]. Here, we report a case with slow delta waves on the electroencephalogram (EEG) change resulting from unintentional and habitual unilateral neck compression. The patient showed EEG abnormalities accompanied by habitual neck compression on video-electroencephalogram monitoring. To differentiate hypoxia-induced seizure-like episodes from epileptic seizures causing recurrent GTCS, Tc-99m HMPAO SPECT was performed immediately after neck compression.

Case Report

A 19-year-old male was admitted to evaluate recurrent generalized tonic-clonic seizures (GTCS), which were unresponsive to valproate. The first seizure episode had occurred 2 months before the admission, during military service. The episode was first noticed by his colleagues. The patient showed convulsive and tonic movements of all extremities for 3 min. He underwent six episodes of recurrent seizures for a month, and valproate was initiated after the first hospital visit. However, the seizure episodes persisted. Therefore, he was admitted for evaluation of his recurrent seizure events.

He had been healthy previously, and there was no familial history of neurological disease or trauma history. General physical and neurological exams were normal. However, he showed an atypical habit of right neck compression. As a further evaluation, brain MRI and video-EEG monitoring

H. Choi · M.-S. Seo · H.-Y. Lee (✉) · S. E. Kim
Department of Nuclear Medicine, Seoul National University
Bundang Hospital, 173-82 Gumi-ro, Bundang-gu, Seongnam-si,
Gyeonggi-do 463-707, Korea
e-mail: debobkr@gmail.com

Y.-S. Kim · C.-H. Yun · S.-H. Park
Department of Neurology, Seoul National University Bundang
Hospital, 173-82 Gumi-ro, Bundang-gu, Seongnam-si,
Gyeonggi-do 463-707, Korea

were performed. On the MRI scan, no structural abnormality or steno-occlusive lesion was observed. He showed habitual right hand compression of the right neck during video-EEG monitoring, followed by delta slowing activity in the right fronto-temporal area. The EEG abnormality disappeared immediately after the patient removed his right hand from his neck (Fig. 1). A further 3-day's recording was performed, and eight episodes of repetitive carotid compression-triggered EEG abnormality were found. An EEG change event occurred in the left fronto-temporal area when the patient placed his left hand on the left neck; this was combined with a right arm tingling sensation.

Two possible causes of recurrent delta waves were suspected: hemodynamic deficit due to habitual neck compression and localization-related epilepsy. To differentiate between the two possible causes, Tc-99m HMPAO SPECT was performed immediately after right carotid artery compression manipulation. Epileptic seizures would increase cerebral perfusion, while habitual neck compression inducing a hemodynamic deficit would reduce it. EEG abnormalities in the

right frontal area were found after manual right carotid artery compression. Tc-99m HMPAO (555 MBq) was injected into the right antecubital vein. An hour after injection, SPECT images were acquired using a low-energy, high-resolution parallel collimator. Forty step-and-shoot images were obtained with intervals of 3° for 20 s per step. SPECT images were reconstructed using filtered back projection with a Butterworth filter on a 128×128 matrix.

SPECT demonstrated decreased perfusion in the right internal carotid artery territory (Fig. 2). As there was no abnormality in the right internal carotid artery on the MRI, right hemispheric ictal hypoperfusion matched with EEG abnormalities suggested that habitual neck compression-induced hemodynamic abnormalities had caused the abnormal EEG findings. From those findings, it was concluded that cerebral hypoperfusion had triggered the episodic EEG changes because of habitual neck compression. Abnormal EEG findings during video-EEG monitoring were not associated with epileptic GTCS.

Fig. 1 Video-electroencephalogram monitoring results. Video-electroencephalogram was performed. **a** Interictal electroencephalogram shows normal background activity and no epileptiform discharges or pathological slowing. **b** Focal slowing is most pronounced in the right

frontal region after the patient putting his right hand on his right neck was noted; this disappeared immediately when the patient removed his right hand from his neck

Fig. 2 Tc-99m HMPAO SPECT. Tc-99m HMPAO was injected immediately after EEG abnormalities were identified. On the SPECT images, hypoperfusion was found in the right internal carotid artery territory. Considering the EEG abnormalities and semiology, cerebral hypoperfusion induced by the patient's habitual neck compression was suggested

Discussion

Carotid arterial compression can cause a cerebral hemodynamic deficit, which leads to unintentional neuronal injury. In this case, the primary point of the diagnosis of recurrent episodic EEG abnormalities was to clarify the hypoperfusion-induced event, differentiating it from an epileptic seizure. Although video-EEG monitoring revealed episodic EEG abnormalities closely associated with habitual unilateral carotid artery compression, they could not exclude epileptic seizures having caused the previous GTCS. Ictal Tc-99m HMPAO SPECT was useful in this case to help understand the patient's pathophysiology, the seizure events underlying the hemodynamic deficit.

Perfusion SPECT has an important role in the evaluation of epileptogenic foci, by measurement of the regional cerebral blood flow. In particular, because the initial HMPAO uptake in the cerebral cortex remains unchanged and fixed, ictal perfusion SPECT images can reflect the blood flow changes at the moment of the seizure event [4]. Epileptogenic lesions are identified on ictal SPECT by the increased regional cerebral blood flow.

To our knowledge, there has been no report on induced ictal SPECT differentiating hemodynamic abnormalities from epileptic seizures. There have been some reports of reflex epilepsy characterized by seizures in response to a specific stimulus diagnosed with ictal SPECT [5, 6]. Unlike previous reports of ictal hyperperfusion after seizure triggering to evaluate epileptogenic foci, our case demonstrated that Tc-99m HMPAO SPECT could be used to evaluate the cause of episodic EEG abnormalities. As perfusion SPECT is a

noninvasive diagnostic tool for evaluating regional cerebral blood perfusion, it could demonstrate whether the patient's habitual neck compression caused significant hypoperfusion or ictal hyperperfusion.

In the present case, the association between previous GTCS episodes and habitual neck compression was uncertain. Of note, as a noninvasive perfusion study, Tc-99m HMPAO SPECT only confirmed the cause of the recurrent slow delta waves, differentiating it from epileptic events. Further clinical studies are warranted in this case to determine the cause of the recurrent GTCS and whether the neck compression was associated with GTCS.

Generally, in adolescents self-induced hypoxia by compressing the carotid artery has a motive: a pleasurable sensation of euphoria [2]. However, in this case, carotid compression was an unintentional habit, not done to achieve a euphoric sensation. In order to break the patient's habit, further psychiatric evaluation is needed.

Our case showed abnormal EEG waves due to cerebral hypoperfusion. Previously, continuous EEG monitoring has been suggested to evaluate cerebral flow [7]. Furthermore, a processed EEG monitoring system, the bispectral index, could be used to detect cerebral ischemia during anesthesia or critical care [8, 9]. Thus, the slow-delta waves and cerebral hypoperfusion were correlated with each other in this case. Of note, carotid compression induced significant hypoperfusion, which could be identified on Tc-99m HMPAO SPECT, and we found a correlation with the abnormal EEG changes. Remembering the previously reported ischemia-induced EEG change and the coupling of neuronal activity and cerebral perfusion, our finding should not be specific for this case.

In summary, habitual carotid compression may result in neurological injury and recurrent EEG abnormalities. As a noninvasive functional neuroimaging technique, Tc-99m HMPAO SPECT can be used to identify underlying causes of episodic EEG changes, differentiating them from epileptic seizures.

Conflict of Interest Hongyoon Choi, Min-Seok Seo, Ho-Young Lee, Young-Soo Kim, Chang-ho Yun, Sang Eun Kim, and Sung-ho Park declare that they have no conflict of interest.

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