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Case Report

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Recognition of Epileptiform K-Complexes in Generalized Epilepsy: A Case Report

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Abstract

Background: Epileptiform K-complex is spike/polyspike discharges overlap K-complex. The variable appearance of the K-complex makes the epileptiform abnormality difficult to recognize.

Objective: To recognize and raise attention for epileptiform K-complexes.

Case Report: A 35-year-old man with a history of juvenile absence epilepsy had 2 new onset generalized convulsions. EEG (Electroencephalogram) recorded 95 K-complexes periodically during sleep, 22 of which were epileptiform K-complexes. Small spike and polyspike were superimposed on either the ascending or the descending limb of the slow wave of K-complex. In addition, spike/polyspike-wave discharges were immediately preceding the K-complex to form a polyphasic slow wave with long duration of K-complex.

Summary: In generalized epilepsy, epileptiform K-complexes have superimposed spike/polyspike with sharper morphology and faster frequency than normal intra-K-complex oscillation. Epileptiform K-complexes may also have a relatively higher amplitude and longer duration when pike/polyspike-wave preceding K-complex, distorts the morphology of K-complex. The most helpful recognition of the epileptiform K-complex is the similar morphology of spike/polyspike-wave recorded during photic stimulation, hyperventilation, or awake period.

Keywords

K-complex, Sleep spindle, Spike, Polyspike, EEG, Epilepsy

Introduction

K-complex is a sharp high voltage biphasic slow wave, associated with sleep spindles during stage II and III sleep in EEG (Electroencephalogram). K-complex was initially described in 1938 by Loomis et al., as a burst of variable appearances, consisting of a high voltage diphasic slow wave frequently seen with sleep spindle, spontaneously or in response to sudden sensory stimuli [1]. Niedermeyer described overlapping polyspike discharges and K-complexes as epileptic K-complexes for patients with generalized epilepsy 50 years ago [2-4]. However, very few publications reported epileptic K-complex until recently. Seneviratns et al., found epileptiform K-complexes occurring in 65.4% their patients with genetic generalized epilepsy [5]. They described epileptiform K-complexes as spikes/polyspike, usually overlapping on the ascending limb of the surface-negative wave of K-complex. The variable appearance of the K-complex makes the epileptiform abnormality extremely difficult to recognize. There are very few published examples of the epileptic K-complex available for EEG readers to compare. This distinctive epileptiform EEG abnormality can easily be

misinterpreted as a normal variant. This recent case provides an example of such a challenge to EEG interpretation, but also provides an opportunity to compare the morphological characters of epileptic K-complexes with physiological K-complexes in generalized epilepsy.

Case Report

A 35-year-old man developed staring spells, daily multiple times, since age 13. He was diagnosed with juvenile absence epilepsy, treated with valproate acid, and did well. At the age 18, valproate acid was discontinued when he lost insurance. His staring spells, less than 30 seconds, retuned more than 10 times a day, with significantly increased frequency after sleep deprivation. He had no myoclonic jerk or convulsion until he was brought to the emergency room after he was found on the ground with loss of consciousness. He had a generalized tonic-clonic seizure less than 1 minute after arrival in the emergency room. His physical examination was normal. His head and cervical spine CT were normal, as was his brain MRI. Laboratory tests were all normal except for potassium 3.1 mmol/L and positive cannabinoids in his urine toxicology screen.

A routine EEG, performed the day after the admission, recorded a predominant sleep stage II period with a short awake period before and during photic stimulation. A normal posterior dominant alpha rhythm was observed while awake. During sleep, frequent sleep spindles and K-complexes were recorded. Twenty two of the 95 K-complexes were observed with small spike/polyspike superimposed on either the ascending or the descending limb of the surface-negative wave of the K-complex. Some time, small spike/polyspike and wave discharges immediately preceded the K-complex, distorted the morphology of K-complex from biphasic waveform to polyphasic waveform with a relatively higher amplitude and longer duration. During photic stimulation, burst generalized irregular spike/polyspike and wave discharges were observed at 2-3 Hz, 70-90 microvolt, and lasted 1-2 seconds without clinical movement. Hyperventilation was not performed. The EEG was consistent with the history of generalized epilepsy. Figure 1 shows examples of K-complexes, and interictal discharges during photic stimulation.

The patient was treated with levetiracetam 1000 mg intravenously in emergency room, and then maintained on levetiracetam 500 mg orally 2 times a day. He has been seizure free since.

Discussion

The sharply contoured K-complexes on EEG were observed the first time by the author as an adult neurologist reading EEG for 20 years. The case challenged the author as well as clinical neurophysiology colleagues with regard to whether those K-complexes were a normal variant or an epileptiform abnormality. Epileptic K-complex is not well recognized by most EEG readers. Although Niedermeyer reported epileptic K-complex in primary generalized

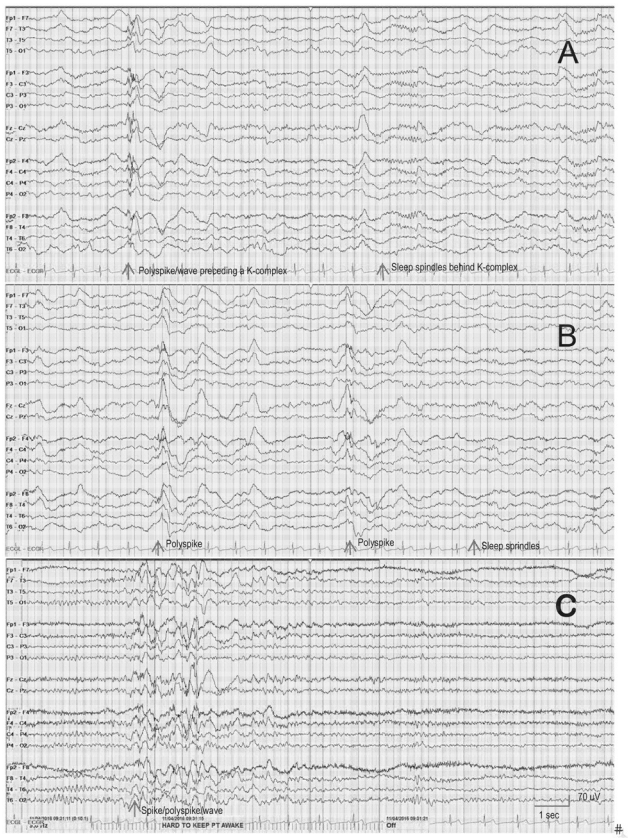

Figure 1: (A) Epileptiform K-complex with polyspike and wave preceding a K-complex is compared with a K-complex behind. Note the difference in morphology, amplitude and duration between them, as well as the difference in frequency between polyspike and sleep spindle. **(B)** Epileptiform K-complex with small polyspike overlap on either the ascending or the descending limb of the surface-negative wave of K-complex. **(C)** Spike/polyspike and wave discharges observed during photic stimulation have similar morphology as they precede K-complex in A.

epilepsy in 1965 [2-4], it is still an under recognized EEG phenomenon, and there are few examples of epileptic K-complex in the EEG atlas. It is possible that most routine EEG did not have a sufficiently long sleep period to record enough K-complexes. The largest study regarding epileptic K-complex and sleep spindles by Seneviratne et al., was a 24-hour ambulatory EEG [5]. The present case illustrates and provides additional morphological information regarding epileptic K-complexes to the existing literature, and compares qualitative morphological differences between epileptiform K-complex and physiological K-Complex. In epileptiform K-complex, polyspikes have sharper morphology and faster frequency than sleep spindles when they are superimpose on or preceding K-complexes. Polyspike and wave preceding K-complex distorts the morphology of K-complex from biphasic slow wave to polyphasic slow waves.

The K-complex is mainly a spontaneous event generated in cortical networks during sleep. As Amzica and Steriade indicated, the initial surface-positive wave is due to the synchronous excitation of cortical neurons, while the subsequent surface-negative wave represents neuronal hyperpolarization [6]. They noticed that K-complexes are periodic, and their shape and frequency are modulated by the deepening of

sleep. Niedermeyer emphasized that K-complexes are an arousal response, and more pronounced in children older than age 4 to adolescents, and decrease amplitude after age 20 [7]. Therefore, the morphology of K-complex is variable from person to person, and also variable in the same person, depending on the stage of sleep and the degree of arousal. In normal humans, Kokkinos et al., observed intra-K-complex oscillation in the 7–9 Hz range over the negative peak [8]. In contrast, generalized polyspike overlapping on K-complex in the present case, was observed in the fast beta frequency. Those polyspike may have low amplitudes, but the spiky morphology and the fast oscillation frequency are distinguishable from normal variants.

In a study of 106 patients with genetic generalized epilepsy by Seneviratns et al., none of the clinical variables, such as seizure types, duration, or treatments had any significant impact on the occurrence of epileptiform K-complexes [5]. In the present case, EEG was recorded on the second day of hospitalization. The patient had already been treated with levetiracetam intravenous loading dose the day before, and also on an oral maintenance dose. He had no convulsions or staring spells, however, epileptiform K-complexes and interictal discharges were still present during sleep and photic stimulation. Niedermeyer described epileptiform K-complexes as "dyshormia", an abnormal arousal phenomenon in several of his publications [7, 9, 10]. He suggested that arousal stimulated epileptic K-complexes are maximal in the frontal midline, but K-complexes reach maximum more posteriorly in the vertex. He considered that epileptic K-complexes are a key to understanding primary generalized epilepsy. Most patients with primary generalized epilepsy suffer from a faulty arousal which induces epileptiform discharges and clinical seizures. However, epileptiform K-complex is not unique for generalized epilepsy. Focal spikes during K-complexes in patients with focal onset epilepsy of prolonged video-EEG monitor were reported by Geyer et al. [11]. They found focal spikes within K-complex ipsilateral to the side of ictal onset in their 40 presurgical patients. In this group, fewer than 10% of K-complexes were associated with spikes. Later, Niedermeyer reported that epileptiform K-complexes also occurred in focal epilepsy, and that epileptiform K-complexes might be skewed from midline maximum to the focal discharge side [12].

Summary

Epileptiform K-complexes are present in both generalized and focal epilepsy. The distinction between epileptiform K-complex and K-complex is more difficult to recognize in generalized epilepsy than focal epilepsy because of a generalized distribution of spike/polyspike overlapping the K-complex. Spike/polyspike with spiky morphology and fast frequency are superimposed on either the ascending or the descending limb of the surface-negative wave of the K-complex. Spike/polyspike-wave may change K-complex from biphasic waveform to polyphasic waveform with a relatively higher amplitude and longer duration. These morphological features of the epileptiform K-complexes should alert EEG readers to identify them as an epileptiform abnormality due to an abnormal arousal phenomenon instead of dismissing them as normal variants of sleep architecture. The most helpful recognition of the epileptiform K-complex is the similar morphology of spike/polyspike-wave recorded during photic stimulation, hyperventilation, or awake period.

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