

Surgical treatment of occipital epilepsy: Basic and clinical approach

Tatsuya TANAKA, Masato SAITO, Masao SATO, Ryogo ANEI, Yoshimitsu HAYASHI, Satoru HIROSHIMA, Ryosuke ORIMOTO, Akira HODODUKA, Kiyotaka HASHIZUME, Kyousuke KAMADA

Department of Neurosurgery, Asahikawa Medical College, Asahikawa, Japan

Abstract

A kainic acid microinjection into unilateral occipital cortex induced an experimental model of occipital lobe epilepsy in cats and rats. Elicited focal seizures in the occipital cortex promptly propagated to the bilateral cortices and also to the subcortical structures. Behavioral and EEG observations were well correlated to the human occipital lobe epilepsy. Metabolic study using ¹⁴C-deoxyglucose autoradiography in rats demonstrated a rapid propagation of the hypermetabolic area in the parietal, frontal, temporal and contralateral occipital cortices and also to the thalamus, basal ganglia, MRF and lateral geniculate body. The result shows that not only Meyer's loop but also subcortical fasciculus between occipital lobe and other lobules may have an important role in the mechanism of seizure evolution and propagation of the occipital lobe epilepsy.

INTRODUCTION

The occipital lobe is the main center of the visual system. Occipital lobe epilepsy accounts for about 5-10% of all epilepsy. This kind of epilepsy can be either idiopathic (of unknown, presumed genetic cause) or symptomatic (associated with a known or suspected underlying lesion). In patients with medically refractory occipital lobe epilepsy, surgical indication is often impossible due to the risk of visual field impairment. In this study, experimental occipital lobe epilepsy is induced by a microinjection of kainic acid (KA) in the unilateral occipital cortex of cats and rats. Behavior and EEG were observed, and a glucose metabolism using ¹⁴C-deoxyglucose was analyzed in rats.

METHODS

In the present study, chronic cats and rats models of occipital lobe seizures were used. KA was delivered to the target occipital cortex via implanted stainless steel pipe.

Experiment I

Seven cats and seven male Wistar rats were used in this study. Stereotaxic surgery was performed and a cannula for KA injection was implanted into the left occipital cortex. Depth electrodes were inserted into bilateral lateral geniculate

body (LGB) in cats. Exact electrode tip location in the LGB was confirmed by an intraoperative EEG recordings during intermittent photic stimulations. Screw electrodes were fixed to the bilateral occipital and frontal bones. Electrodes were connected to the head socket for EEG recordings. Seven days after the surgery, KA solution (2 µg as KA) was injected using injection needle. About 30 minutes after the KA injection, focal occipital seizure status was elicited in all cats and rats on EEG. Seizures recurred every 5-6 minutes. These seizures were correlated to the contralateral focal motor seizures on the right side, initially. These seizures rapidly developed to secondarily generalized seizures within 60 minutes and subsided within 8 hours. At the end of the experiments, all animals were perfused with 10% formalin solution and processed for the pathological study.

Experiment II

Seven Wistar rats were used in this study. Stereotaxic surgery was performed and a cannula for KA injection was implanted into a unilateral occipital cortex. Seven days after the surgery, they are again anesthetized with halothane and the right femoral vein and artery were cannulated. KA solution (2 µg as KA) was injected via cannula using injection needle. About 30 min after the KA injection, all rats presented

occipital lobe seizure status. Autographic process was then performed 30 and 60 minutes after intravenous administration of $100 \mu\text{Ci}/100\text{gr}$ of ^{14}C -deoxyglucose. Coronal section of the frozen brain was prepared and exposed to the X-ray films. Glucose utilization of the brain during occipital seizure was investigated.

RESULTS

In Experiment I, KA injection into unilateral occipital cortex produced focal occipital lobe seizure status about 30 minutes after the injection without any exception. Each seizure recurred every 5-6 minutes. Initially, seizures were focal motor seizure of the contralateral limbs. Seizures developed rapidly and animals began to present secondarily generalized seizures within 1 hour after KA injection. On EEG, remarkable epileptic excitations were induced in the both LGBs, more on the focus side. These tonic clonic generalized seizures were bilateral tonic seizure with short term clonic convulsions. There was no clear laterality in generalized seizures. These generalized seizures gradually disappeared and subsided within 8 hours after KA injection.

In Experiment II, autoradiograms were made during generalized seizure periods (2 hours after the injection). A remarkable hypermetabolic area was noted in the injected site of the occipital cortex. This hypermetabolic area rapidly propagated to various sites of the brain. These hypermetabolic sites were observed at bilateral motor cortex, temporal cortex, thalamus, basal ganglia and LGB.

DISCUSSION

In occipital lobe epilepsy, seizures arise from the occipital lobe of the brain. Occipital lobe has a rich connection to frontal, parietal and temporal lobes. They are superior occipito-frontal fasciculus, superior longitudinal fasciculus and inferior occipito-frontal fasciculus between frontal and occipital lobes. Arcuate fibers are between parietal and occipital cortices. Inferior longitudinal fasciculus is a connection between temporal and occipital lobes. Moreover, optic tract comes from retina via lateral geniculate body to the occipital cortex (Meyer's loop). Bilateral occipital cortices are connected by callosal fibers by way of splenium of the corpus callosum.¹ The seizure of the occipital cortex demonstrates very rapid propagation to the various sites of the ipsilateral and contralateral cortices comparing to the other models of KA induced seizures.^{2,3} The

characteristics of the anatomical connections may play an important role in the rapid seizure propagation not only to the various ipsilateral brain sites but also to the contralateral hemisphere of the brain. In the present study, KA-induced focal occipital seizure initially demonstrated pure focal occipital seizures on EEG. Then, rapid evolution of seizure propagation to the both hemisphere were characteristic phenomenon. Behavioral generalized seizures are also bilateral tonic-clonic seizures. Autoradiograms also demonstrated rapid generalization of the seizures. The present results is not only important to understand a seizure mechanism of the occipital epilepsy but also useful to make a surgical strategy to treat intractable occipital lobe epilepsy.⁴ Resective epilepsy surgery of the occipital lobe has a risk of visual impairment. In the present study, both LGBs demonstrated remarkable seizure activities during occipital seizures. As LGB has an important connection to the thalamus, deep brain stimulation may be a possible option in the treatment of intractable occipital lobe epilepsy in order to avoid visual problem.^{5,6}

REFERENCES

- Penfield W, Boldrey E. Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. *Brain* 1937; 60:389-443.
- Takano K, Tanaka T, Fujita T, Nakai H, Yonemasu Y. Zonisamide: Electrophysiological and metabolic changes in kainic acid-induced limbic seizures in rats. *Epilepsia* 1995; 36:644-8.
- Tanaka T, Tanaka S, Fujita T, Takano K, Fukuda H, Sako K, Yonemasu Y. Experimental complex partial seizures induced by a microinjection of kainic acid into limbic structures. *Prog in Neurobiol* 1992; 38:317-34.
- Tanaka T, Tsuda H, Hashizume K, Sakurai J, Hodozuka A, Nakai H. Clinical application of experimental cortical dysplasia in rats. *J Child Neurol* 2005; 20: 351-6.
- Takebayashi S, Hashizume K, Tanaka T, Hodozuka A. The effect of electrical stimulation and lesioning of the anterior thalamic nucleus on kainic acid-induced focal cortical seizure status in rats. *Epilepsia* 2007; 48:348-58.
- Takebayashi S, Hashizume K, Tanaka T, Hodozuka A. Anti-convulsant effect of electrical stimulation and lesioning of the anterior thalamic nucleus on kainic acid-induced focal limbic seizure in rats. *Epilepsy Research* 2007; 74:163-70.