Ictal Spitting in a Patient with Dominant Temporal Lobe Epilepsy: Discrepancy between Epileptogenic and Symptomatogenic Areas for Spitting Automatism

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Ictal spitting is an unusual manifestation that originates from the non-dominant hemisphere, but rarely from the dominant hemisphere. In the latter case, it has not been well defined as to whether symptomatogenic area for ictal spitting originates from the dominant hemisphere. We present a patient with ictal spitting. Intracranial EEG demonstrated a left hippocampal onset with propagation to the right hemisphere, and subsequent ictal spitting development. Even in dominant hemispheric seizures, the non-dominant hemisphere is a symptomatogenic area for ictal spitting.


Key Words: Spitting automatism, Non-dominant temporal lobe epilepsy, Lateralizing sign.

Automatisms are traditionally defined as stereotyped non-purposeful automatic behaviors during complex partial seizure, and the automatisms known to have a lateralizing value in temporal lobe epilepsy (TLE). Spitting automatisms, though rarely reported, have been predominantly associated with nondominant temporal lobe lateralization. Although pathophysiological mechanism of spitting automatism has not been fully defined, it has been hypothesized that the right hemispheric dominance of spitting automatism is due to the functional asymmetry of the central autonomic network,\(^1\) such as ictal vomiting or ictal urinary urge.\(^2\)\(^-\)\(^3\) Ictal spitting has also been rarely reported in dominant hemispheric seizures, but in such cases it has not been well defined as to whether the symptomatogenic ictal spitting area originates from the dominant hemisphere.

We present here a right-handed TLE patient with ictal spitting, whose seizures originated from the left temporal lobe. An intracarotid amobarbital procedure (IAP) demonstrated left hippocampal onset with propagation to the right hemisphere. The symptomatogenic area of ictal spitting was located to the right hemisphere, most likely temporal lobe.

CASE REPORT

A 28-year-old right-handed man with an 11-year history of intractable seizures underwent a compre-
우성 측두엽 간질환자에서 침뱉기 자동증의 증상발생 영역의 측위는 어느 쪽인가?

Figure 1. Neuroimaging findings. (A) T2 weighted MR image shows left hippocampal atrophy with hyperintense T2 signal (arrow head) (B) 18FDG-PET shows left medial temporal hypometabolism (arrow)
spitting automatism (Fig. 2–B). Intracranial EEG and semiological analyses led to a conclusion that the epileptogenic area was the left mesial temporal lobe and the symptomatogenic area for spitting automatism was the right hemisphere. Left anterior temporal lobectomy with amygdalo–hippocampectomy was performed, and at the time of writing this patient had remained seizure–free without antiepileptic medication for 2 years after surgery.

**DISCUSSION**

Ictal spitting is a rare epileptic event and only occurs in approximately 0.3% of the monitored epilepsy population. In a study on the differentiating clinical features of right and left temporal lobe seizures, ictal spitting was exclusively observed in patients with right temporal lobe seizures. Voss et al., reviewed 2500 epilepsy patients and found only 5 ictal spitting cases with a lesion in the right temporal lobe. Moreover, in all patients, resection of the right temporal lobe produced a seizure–free state. However, even more rarely patients with ictal spitting originating from the left temporal lobe have been reported. In 25% of cases with ictal spitting, seizures may arise from the left, language–dominant hemisphere. But the symptomatogenic area for ictal spitting in seizures originating from dominant hemisphere has not been well documented.

Ozkara et al., reported one patient with seizures originating in the left hemisphere, in whom the right hemisphere was dominant for language. Other patients with seizures arising from the left hemisphere have been described, but no mention was made of language lateralization. Kellinghaus et al., described 12 patients with ictal spitting, including one patient in whom seizures were determined to have been caused by dominant temporal lobe epilepsy by IAP and invasive EEG. However, they performed an unilateral invasive study in the left side only. In view of the possible discrepancy between epileptogenic and symptomatogenic areas for spitting automatism, such as in our case, unilateral invasive monitoring is insufficient for identification of anatomical structure of spitting automatism whether it originated from the dominant hemisphere even though the epileptogenic area was the dominant temporal lobe.

In our case, surface EEG was not helpful at localizing or lateralizing the epileptogenic area, and the epileptogenic area was eventually localized to the left mesial temporal lobe by invasive study using bilateral depth and strip electrodes. However, the symptomatogenic area for ictal spitting turned out to be right hemisphere, because spitting automatism occurred after ictal rhythms propagated to the right temporal depth electrodes. One seizure without spitting automatism was recorded in our patient, during which the ictal rhythm originated from the left mesial temporal lobe as previous seizures and terminated without propagation to the right hemisphere. Moreover, the corresponding sequence of semiology was identical to the previous nine seizure events, except for the ictal spitting. These findings suggest that ictal spitting in our patient arose due to right hemisphere involvement, though the seizures originated in the dominant hemisphere.

The location of structures responsible for spitting automatism has not been well known. The localizing value of this symptom appears to arise from temporal lobe, because ictal spitting requires activation of the limbic structures of at least one side. Considering the functional asymmetry of the central automatic network or functional disturbances of area controlling emotional behavior on the nondominant hemisphere, the right temporal lobe may be important for ictal spitting. In our case, the localization for ictal spitting may have been the right temporal lobe, because it occurred immediately after ictal rhythm had propagated to the right temporal depth electrodes. However, we could not conclude with precise anatomical structure of ictal spitting, because the present study was conducted with limited numbers of intracranial electrodes that monitored only the bilateral mesial temporal and lateral temporal lobes.

As described above, all previously available published cases with ictal spitting originated from dominant hemisphere have had not enough evidence for identification of
anatomic structure for spitting automatism. We documented in the present case the symptomatic area for ictal spitting as the nondominant hemisphere, using bilateral depth and strip electrodes, even in a case of dominant temporal lobe seizure.

To our knowledge, this is the first case report issued in Korea regarding ictal spitting originating from dominant temporal lobe epilepsy. Moreover, this case suggests an important notion, namely, that the symptomatic area responsible for spitting automatism may be the non-dominant hemisphere, even when the epileptogenic area is located in the dominant hemisphere.

REFERENCES