Ictal Spitting in a Patient with Dominant Temporal
Lobe Epilepsy: Supporting Evidence of Ictal
Spitting from the Nondominant Hemisphere

Sea-Mi Parka  Sang-Ahm Leea  Ji Hyun Kimb  Joong Koo Kanga

aDepartment of Neurology, Asan Medical Center, University of Ulsan College of Medicine, and
bDepartment of Neurology, Guro Hospital, University of Korea College of Medicine, Seoul, South Korea

Dear Sir,

Automatisms are traditionally defined as stereotyped nonpurposeful automatic behaviors during a 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 the 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]. There have been rare reports of ictal spitting in dominant hemispheric seizures, but in such cases it has not been well explained whether the symptomatogenic ictal spitting area originates from the dominant hemisphere.

Here, we present the case of a right-handed TLE patient with ictal spitting, whose seizures originated from the left temporal lobe. An intracarotid amobarbital procedure (IAP) demonstrated left temporal lobe. An intracarotid amobarbital atrophy on the T1-weighted image, which was consistent with hippocampal sclerosis (fig. 1a), and 18FDG-PET showed mild hypometabolism in the left medial temporal cortex (fig. 1b). Long-term video-scalp EEG monitoring showed 10 stereotyped seizures manifested by aura and motionless staring, followed by bilateral hand automatisms and repetitive spitting on the bed. Average seizure duration was 31.6 ± 8.6 s and average time of propagation to the right hemisphere was 18.8 ± 5.0 s. Ictal spitting usually occurred 19.5 ± 5.2 s after clinical seizure onset. Ictal scalp EEG with sphenoidal electrodes was of no value for either lateralization or localization. There were no identifiable interictal epileptiform discharges on scalp EEG with sphenoidal electrodes. An ictal SPECT showed mild hyperperfusion in the left temporal lobe and right thalamus. IAP demonstrated language dominance for the left hemisphere. Intracranial monitoring was performed using bilateral mesial temporal depth (1 on each side, 8 contact points per electrode) and mesial and lateral temporal subdural strip electrodes (2 on each side, 4 contact points per electrode) to determine the precise onset area of his epileptic seizures. During intracranial EEG monitoring, a total of 10 electrographic seizures were recorded. Of these 10 seizures, 9 seizures revealed that fast (15–20 Hz) ictal rhythms were recruited in the left hippocampal depth electrodes and propagated to the right hippocampus depth electrodes after which spitting automatisms were evident (fig. 2a). Ictal
Fig. 1. Neuroimaging findings. a $T_2$-weighted MR image shows left hippocampal atrophy with hyperintense $T_2$ signal (arrowhead). b $^{18}$FDG-PET shows left medial temporal hypometabolism (arrow).

Fig. 2. Intracranial EEG findings. a The seizure starts with rhythmic activity in the left depth electrodes (LD1–LD4) contacting amygdala and hippocampus (arrowhead). The patient feels aura several seconds after seizure propagation to the right medial temporal lobe (short arrow). The seizure activity propagates with fast rhythmic (10–15 Hz) recruiting in the right depth electrodes contacting amygdala and hippocampus (RD1–RD4), after which spitting automatism emerged. b One of the 10 seizures originated at the left mesial temporal lobe, but was confined to the left hemisphere, and was manifested as aura and motionless staring without spitting automatism (long arrow).
spitting occurred 21.5 ± 5 s after EEG on- 
set and EEG seizure durations were usu-
ally 31.1 ± 8.6 s. One of the 10 seizures 
originated at the left mesial temporal lobe, 
but was confined to the left hemisphere, 
and was manifested as aura and motion-
less staring without spitting automatism 
(fig. 2b). Intracranial EEG and semiologi-
cal analyses led to a conclusion that the 
epileptogenic area was the left mesial tem-
poral lobe and the symptomatic area for 
spitting automatism was the right hemi-
sphere. Left anterior temporal lobec-
tomy with amygdalohippocampectomy 
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 [4]. In 
a study on the differentiating clinical fea-
tures of right and left temporal lobe sei-
zures, ictal spitting was exclusively ob-
served in patients with right temporal lobe 
seizures [5]. Voss et al. [6] reviewed 2,500 
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 pro-
duced a seizure-free state. However, pa-
tients with ictal spitting originating from 
the left temporal lobe have been reported 
even more rarely.

Ozkara et al. [7] reported 1 patient with 
seizures originating in the left hemisphere, 
in whom the right hemisphere was domi-
nant for language. Other patients with sei-
zures arising from the left hemisphere 
have been described, but no mention was 
made of language lateralization [8, 9].

Kellinghaus et al. [4] described 12 pa-
tients with ictal spitting, including 1 pa-
tient in whom seizures were determined to 
have been caused by dominant TLE by IAP 
and invasive EEG. However, they per-
formed a unilateral invasive study in the 
left side only. In view of the possible 
discrepancy between epileptogenic and 
symptomatic areas for spitting au-
tomatism, such as in our case, unilateral 
invasive monitoring is insufficient for 
identification of the anatomical structure 
of spitting automatism whether it origi-
nated from the dominant hemisphere even 
though the epileptogenic area was the 
dominant temporal lobe.

In our case, surface EEG was not help-
ful in localizing or lateralizing the epilep-
togenic area, and the epileptogenic area 
was eventually localized to the left mesial 
temporal lobe by invasive study using bi-
lateral depth and strip electrodes. Howev-
er, the symptomatic area for ictal spit-
ting turned out to be the right hemisphere, 
because spitting automatism occurred af-
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 ter-
minated without propagating 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 arose in our patient due to 
right hemisphere involvement, though the 
seizures originated in the dominant hemi-
sphere. Also the localization for ictal spit-
ting may have been the right temporal lobe 
in this patient, because it occurred imme-
diately after ictal rhythm had propagated 
to the right temporal depth electrodes. 
However, we could not conclude on the 
precise anatomical structure of ictal spit-
ting, because the present study was con-
ducted with limited numbers of intracra-
nial electrodes that monitored only the 
bilateral mesial temporal and lateral tem-
poral lobes.

As described above, all previously pub-
lished cases with ictal spitting originating 
from the dominant hemisphere have not 
had enough evidence for identification of 
the anatomical structure of spitting automa-
tism. Thus, the present case is of value be-
cause it documents the symptomatic area 
for ictal spitting, i.e. 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 
documented by a bilateral invasive study of 
ictal spitting originating from dominant 
TLE. Moreover, this case suggests an im-
portant notion, namely, that the symptom-
atic area responsible for spitting au-
tomatism may be the nondominant hemi-
sphere, even when the epileptogenic area is 
located in the dominant hemisphere.

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