Clinical Outcome of Patients with SREDA (Subclinical Rhythmic EEG Discharge of Adults)

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Abstract

Objective To clarify the clinical significance of ‘subclinical rhythmic EEG discharge of adults’ (SREDA) by analyzing characteristics of SREDA and the outcome of patients based on retrospective analysis of EEG data base.

Methods EEGs were recorded soon after the onset of patient’s initial symptoms and repeatedly recorded at various intervals of 2-3 months in all 4 patients. Neurological findings, MRI and SPECT were also investigated.

Subjects Out of 340 consecutive inpatient population who had EEGs, 4 patients (1.2%) showed SREDA. They had a diagnosis of syncope, transient global amnesia, generalized tonic-clonic seizure and right temporal lobe epilepsy for each.

Results There was no consistent abnormality in the brain MRI, CT or SPECT among the 4 patients. The acute and transient symptoms disappeared and did not recur within the follow-up period of 28 months in any patient. In 2 patients SREDA disappeared in the follow-up EEG taken 7-14 days after the first EEG showing SREDA. In the other 2 patients, the follow-up EEGs taken 5 days after the first EEG with SREDA when clinical symptoms disappeared showed less frequent occurrence of SREDA.

Conclusion Being different from the previous reports suggesting the relation with cardiogenic insults or persistent ischemic abnormality, SREDA can occur in patients with various acute brain dysfunctions followed by a favorable clinical outcome.

Key words: SREDA (subclinical rhythmic EEG discharge of adult), clinical correlates, acute brain dysfunction, outcome

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Introduction

Westmoreland and Klass (1) described a series of 65 patients who had a distinct rhythmic EEG pattern that they termed as ‘subclinical rhythmic EEG discharge of adults’ (SREDA). SREDA is an uncommon EEG finding seen in elderly people. It resembles an EEG seizure pattern, but it was considered to have little clinical significance. Based on its wave forms, SREDA is defined as “an unusual transient EEG pattern consisting of repetitive bilateral sharp contoured waveforms in the range of theta to delta frequency lasting for a few seconds to minutes, distributed mainly over the parietal and temporal regions, without subsequent evolution in frequency, distribution or morphology” (1, 2). It was described in patients with many different neurological complaints such as headache, syncope, seizures, etc., but there have been only a few reports focusing on its clinical significance and especially its relationship with epileptic seizures (3-8). The previous reports mainly focused on the EEG findings themselves (1, 2), while its correlation with MRI, SPECT and CT findings in those patients has rarely been...
Figure 1. An example of SREDA observed after transient global amnesia in Patient 2 when the patient was clinically normal. Note 1-2 Hz rhythmic sharp transient pattern, distributed diffusely, but larger over the anterior temporal–midtemporal areas. Posterior dominant rhythm of EEG is not clear before the onset of SREDA, but later it appears and is relatively well organized.

Subjects and Methods

We checked the EEG recording list in the central laboratory and found a total of 340 patients, age 15-88 years (average of 54 years), who had an EEG examination in the Department of Neurology, Kyoto University Hospital between January 1st, 2000 to April 30th, 2002 out of inpatient population. SREDA was observed in 4 out of those patients; their ages ranged from 56 to 84 years (average, 68 years).

All of the EEGs of the 340 patients were interpreted by a certified EEGer, one of the coauthors (AI), and the SREDA was diagnosed based on the initially proposed definitions (1, 2) as described in the introduction.

Patient 1 was an 84-year-old male. Left chronic subdural haematoma occurred soon after a traffic accident. One week after the first operation, the patient felt drowsy. Since the subdural haematoma recurred on MRI, the patient was admitted again for a 2nd operation, 26 days after the first operation. Seven days after the second operation, the first drop attack occurred in the toilet followed by mild left hemiparesis which partly recovered. The patient felt chest discomfort at that time and cardiogenic syncope was clinically suspected 2 days after the 1st drop attack. The patient felt faintness for a few minutes during talking with a nurse before the 1st drop attack. Three months after the 1st drop attack, the patient had a 2nd drop attack again with mild left hemiparesis. A total of four EEGs were recorded in this patient; 7, 14, 53 and 67 days after the first episode of possible cardiogenic syncope.

Patient 2 was a 56-year-old male with transient global amnesia (TGA). The patient was admitted because of two episodes of retrograde amnesia each lasting for several hours on the same day. MRI, SPECT and CT scan showed no abnormality. Neurologically no abnormal findings were detected when admitted. Two EEGs were recorded in this patient; on the first day and 7 days after the second episode of TGA, respectively.

Patient 3 was a 74-year-old male admitted to the hospital because of generalized tonic-clonic-seizure. Ten years previously the patient had episodes of vertigo and hypertension. MR angiography revealed severe basilar artery stenosis. Head MRI and CT scan showed multiple small infarctions in the bilateral subcortical areas, bilateral mesial thalamus and left cerebellar hemisphere. SPECT study showed hypoperfusion in the left cerebellum and the right thalamus. Two EEGs were recorded in this patient; 3 and 8 days after the convulsion, respectively. When the EEGs were recorded, the patient was fully alert and neurological examination was unremarkable.

Patient 4 was a 61-year-old male admitted to the hospital because of frequent episodes of loss of awareness associated with oral and hand automatons lasting for several min. Frequency of the complex partial seizure (CPSz) was once per week just before admission, although it used to occur on 4-5 occasions per year. Head MRI, CT scan, SPECT and EKG monitoring showed no abnormalities. The patient was diagnosed as temporal lobe epilepsy. Two EEGs were recorded...
in this patient; on the first day and 8 days after the episode during the hospitalization. No seizures were noticed immediately before or during the EEG recording.

### Results

A representative waveform of SREDA is shown in Fig. 1. It consists of runs of rhythmic sharp transients mainly at 1 - 2 Hz or at 4-6 Hz. The characteristics of SREDA and additional EEG abnormalities seen in the 4 patients are summarized in Table 1. Posterior dominant rhythm was 9-10 Hz and relatively well organized in all of the patients. SREDA was distributed over the parieto-occipital areas in Patient 1 and 4, and over the centroparietal areas in Patient 3. In Patient 2, SREDA appeared diffusely but predominantly over the anterior temporal–midtemporal areas. The frequency of SREDA ranged from 1 to 6 Hz and the duration of runs ranged from 5 to 100 seconds. It occurred on one to four occasions per 30 minutes. In Patient 3, SREDA was not suppressed even by eye opening or verbal call whereas the patient could follow verbal commands. For the other patients, reactivity of SREDA to eye opening or verbal call was not examined.

All 4 patients reported here suffered from at least some impairment of the central nervous system. In Patient 1, the first EEG in which SREDA was absent was obtained when left hemiparesis and cardiogenic syncope possibly occurred. The second EEG obtained one week after the development of mild left hemiparesis still showed frequent bursts of SREDA. In Patient 2, 3 and 4, EEGs taken 1-3 days after some acute episodes showed SREDA on 2-4 occasions. None of those three patients had any neurological symptoms when SREDA was seen. In the follow-up EEGs taken 5-7 days after the first EEG containing SREDA, the SREDA occurred either less frequently (Patient 2 and 3) or disappeared (Patient 4).

### Discussion

The higher prevalence rate of SREDA in our inpatient population (1.2%) as compared with the previous literature based on the inclusive patient population (0.04%) may suggest that SREDA is related to certain transient acute brain dysfunction. Westmoreland and Klass (1, 2) regarded both the typical rhythmic theta pattern and the atypical delta pattern as SREDA equally. Among our 4 patients, three showed rhythmic sharp transients of 1-2 Hz and one showed a quasi-rhythmic theta pattern (Table 1). SREDA is considered to be a benign EEG pattern of uncertain significance (8). Though there have been few studies on SREDA, Westmoreland and Klass (1) suggested that SREDA was much less frequently associated with epileptic seizures (6%) as compared with the incidence of 34% in ‘psychomotor variant’ pattern. In general, SREDA does not abolish the background alpha activity (1). Similarly in our study, the background activity was preserved while SREDA occurred.

The underlying mechanism that produces SREDA still remains unknown. Several observations by Naquet et al (9, 10) suggested that cerebrovascular disease, ischemia or transient hypoxia or a combination of those might have a prominent role in precipitation of SREDA.

Previously there has been no description of MRI, CT or SPECT findings in relation to SREDA. None of our 4 patients had clear evidence of acute stroke when the EEG showed SREDA, but all of them had acute, at least transient, brain dysfunction. In Patient 3 whose first EEG showed fre-
quent SREDA 3 days after generalized tonic-clonic seizures, the CT showed evidence of multiple old infarctions in the white matter and bilateral thalamus. In this patient, therefore, the possibility that transiently impaired global function due to generalized seizure might have played some role in the generation of SREDA could not be excluded. In the previous report (1), only 2 patients out of 65 with SREDA had TGA. One (Patient 2) of the present 4 cases had a diagnosis of TGA. Since TGA may reflect epileptic dysfunction of the hippocampus at least in some patients, the presence of SREDA in patients with TGA may suggest its pathomechanism, but a further study is needed. It is worthwhile mentioning that significant brain ischemic changes revealed by MRI/CT were present in only one (Patient 3) out of 4 patients. Thus, SREDA does not necessarily suggest persistent ischemic abnormality. SREDA is possibly a benign EEG pattern and has a little or no clinical correlation.

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References


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