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Case Report

# A patient with atonic seizures mimicking transient ischemic attacks

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ABSTRACT

A focal atonic seizure is a partial seizure in which the ictal manifestation consists of paresis of the extremities or muscles on one side of the body, and this phenomenon can easily be misdiagnosed as a transient ischemic attack. An 86-year-old woman visited our hospital complaining of transient right upper extremity weakness lasting for 10 min following an unusual sensation in her chest accompanied by palpitations. On the third hospital day, she again complained of right arm weakness, which progressed to jerky movements of her right extremity accompanied by facial twitching and then generalized into a tonic–clonic seizure. The EEG displayed several interictal spikes in the contralateral temporal area, and the ictal SPECT, analyzed using the SISCOM system, showed an increased signal in both the contralateral superior parietal area and the mesial frontal area. In this case, the patient was diagnosed with focal atonic seizures as the cause of the monolimb weakness, which had been initially misdiagnosed aas transient ischemic attacks. In cases in which a patient presents with monolimb paresis, physicians should consider the possibility of an atonic seizure as the cause.



## 1. Introduction

Atonic seizures are characterized by a sudden loss or diminution of muscle tone without an apparent preceding myoclonic or tonic event [1]. This phenomenon was first described as a postepileptic paralytic phenomenon and is now well known as Todd's paralysis. Currently, such an episode of atonia during an epileptic seizure is increasingly recognized as an ictal event. Focal atonic seizures are partial seizures in which the ictal manifestation consists of paresis or paralysis of one or more parts of the body [2]. It is crucial to recognize this limb atonia as an ictal event as it may easily be misdiagnosed as a nonepileptic condition, such as a transient ischemic attack associated with severe arterial stenosis in old age.

Herein, we report the case of a patient with recurrent atonic seizures that presented as recurrent transient right upper limb paresis that was misdiagnosed as transient ischemic attacks.

## 2. Case presentation

An 86-year-old right-handed woman visited our neurology department complaining of recurrent transient right upper extremity paresis that had started 3 days previously. The patient reported that the initial symptoms occurred after an unusual sensation in her chest that was accompanied by palpitations, and she experienced weakness for 10 min without loss of consciousness. There were no abnormal movements or accompanying neurologic deficits, and similar phenomena occurred 2 more times before the hospital visit. She had been treated for hypertension for 15 years, and she had a history of cerebral infarction for more than 5 years that was treated with an antiplatelet drug, but she had no prior history of a seizure disorder. On the neurologic examination, there were no neurologic deficits at the time of admission and her electrolytes, renal function, and complete blood count were within normal ranges. Her systolic blood pressure at the time ranged between 120 and 150. The initial tentative diagnosis was recurrent transient ischemic attacks as she had obviously experienced multiple episodes of transient right-side weakness that persisted for less than 1 h with no residual deficits. The magnetic resonance imaging (MRI) of the brain performed at her admission showed no acute diffusion restriction in the diffusion restriction image (DWI), but there was a complete obstruction of the right proximal internal carotid artery and an encephalomalacia of the right temporooccipital lobe, which was probably due to an old infarction. In addition, a high-signal lesion on the left superior parietal cortical area was observed on the fluid-

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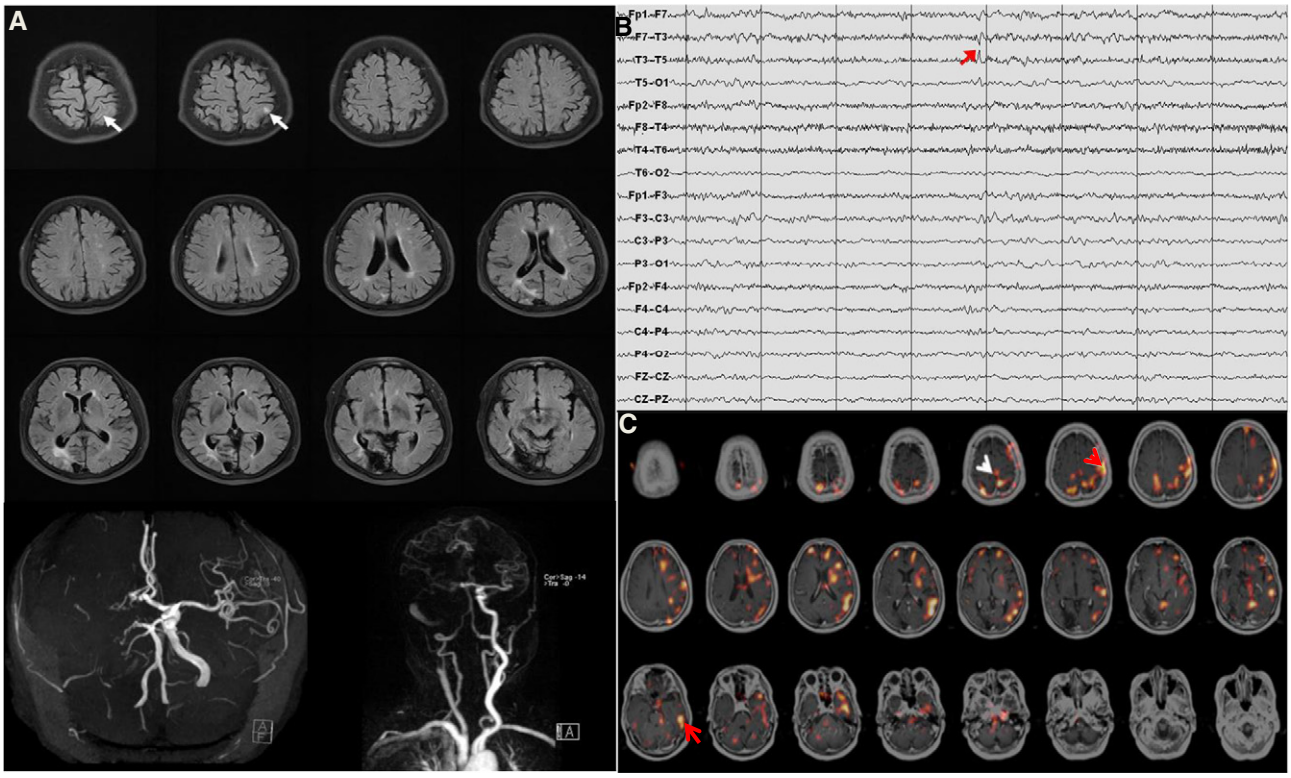


**Fig. 1.** A. The FLAIR MRI showed a high-signal lesion on the contralateral superior parietal cortex (white arrows) and an encephalomalacia of the ipsilateral occipital lobe. The MR angiography showed a complete obstruction of the right proximal internal carotid artery. B. The EEG showed several interictal spikes in the left temporal area (red arrow). C. The ictal SPECT analyzed using SISCOM imaging showed an increased signal in the contralateral temporal cortex (red arrow), the contralateral mesial frontal cortex, which lies in the supplementary negative motor area (white arrowhead), and the contralateral superior parietal cortex, which lies in the somatosensory area (red arrowhead). The arrow shows the SNMA and PNMA that possibly became activated and caused the atonia. In addition to the above SNMA and PNMA, the activation of the functionally related areas on the horizontal level can be observed, which leads to a descending activated lesion on the ipsilateral side. Moreover, a related lesion can also be observed on the contralateral side where they are connected through the commissural fiber.

attenuated inversion recovery (FLAIR) MRI (Fig. 1A). On the third day of hospitalization, the patient again complained of a sense of palpitations, agitation, and general weakness that was dominant in the right arm and very similar to the initial symptoms that she experienced before admission. However, the symptoms persisted for more than 1 h, and the patient gradually showed confusion with complete disorientation and right-side weakness with an MRC grade of II. Following these mental changes, focal repetitive jerky movements of the right extremity with right facial twitching was noted, which then progressed to generalized tonic–clonic movements accompanied by drooling and tongue biting with a duration of 1 min. The episode was controlled after an intravenous injection of 5 mg of midazolam was administered. The postictal period was characterized by confusion and memory lapses for approximately 12 h. After the secondary generalized seizure, the decision was made to evaluate the patient for an epileptic seizure disorder. Her electroencephalogram (EEG) showed several interictal spikes in the left temporal area (T3 maximum) (Fig. 1B). She was initially treated with 1000 mg of levetiracetam per day, but the episodes of right-side weakness still occurred 2 times on the following day without abnormal movements. An ictal single-photon emission CT (SPECT) combined with an MRI (SISCOM) analysis showed an increased signal in the contralateral superior parietal area, which correlated well with a high-signal lesion in the FLAIR and mesial frontal area (Fig. 1C). The dosage of levetiracetam was increased to 2000 mg per day, and no further seizure episode or vascular event occurred during the 6-month follow-up.

## 3. Discussion

The clinical semiology of the patient's seizure was characterized by the abrupt onset of monolimb paresis lasting for less than 10 min. These clinical features were remarkably similar to those of stroke, especially considering the patient's past history and the complete obstruction of the ICA.

Oestreich et al. suggested that epileptic discharge in the contralateral temporal lobe causes ictal unilateral paresis [3]. In this case, it was unlikely that the contralateral temporal area was the symptomatic zone of the ictal paresis because the ictal paresis produced from the temporal lobe is always associated with impairments of consciousness and automatism, and our patient had not lost consciousness. However, the highlighted areas in the contralateral temporal lobe on the ictal SPECT analyzed using SISCOM (the red arrow in Fig. 1C) and the interictal spikes in the contralateral temporal area on the EEG (the red arrow in Fig. 1B) suggest that the contralateral temporal area may have played an important role in the generation of the transient monolimb weakness.

The primary negative motor area (PNMA) and the supplementary negative motor area (SNMA) have been proposed as the inhibitory cortical areas responsible for atonic seizures [4,5]. The PNMA lies anterior to the primary motor face area, and the SNMA is mapped anterior to the face region of the supplementary sensorimotor area of the mesial frontal lobe. Stimulation of these areas produces not only contralateral but also, although to a lesser extent, ipsilateral atonia, predominantly of the limb muscles.

However, previous cases have reported that the primary somatosensory cortex is the possible generator of atonic seizures by activating the selective activation of the inhibitory motor system represented by the negative motor areas [6]. Our patient's unusual sensation in her chest accompanied by palpitations may be explained by the aura that originates from the somatosensory cortex. The presence of the pathology in the corresponding somatosensory area on the FLAIR MRI (the

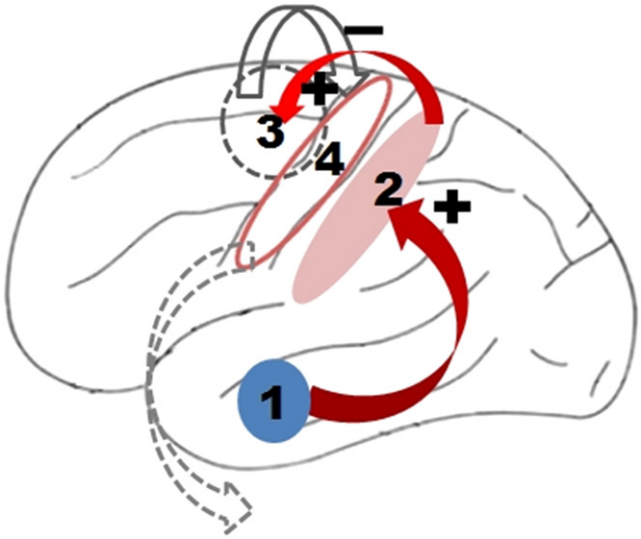


**Fig. 2.** An illustrated working model for the atonic seizure in our patient showing the contralateral temporal area (1) stimulating the contralateral superior parietal cortex (2) followed by the activation of the contralateral mesial frontal cortex (3), which elicited the ipsilateral monolimb paresis (4).

white arrows in Fig. 1A) and the increased signal in the contralateral somatosensory area in the analysis of the ictal SPECT in combination with the SISCOM analysis (the red arrowhead in Fig. 1C) suggest that the contralateral somatosensory area was responsible for the generation of the ictal monoparesis. Moreover, the ictal manifestation is consistent with a previous report that ictal monoparesis is associated with lesions in the primary somatosensory area [6].

The contralateral mesial frontal area, in which the SNMA lies, also showed increased activity in the SISCOM analysis of the patient's ictal SPECT (the white arrowhead in Fig. 1C). Activation of the mesial frontal area is well known to produce contralateral limb paresis. Therefore, we can hypothesize that the spike in the left temporal area stimulated the left superior parietal cortex, which was followed by the activation of the left mesial frontal cortex, which in turn elicited the contralateral monolimb paresis (Fig. 2).

Because focal epilepsy has been reported as a possible manifestation of a transient ischemic attack [7,8], we should consider in this case the possibilities that either the monolimb weakness was the manifestation of a negative seizure provoked by a transient ischemic attack or a newly developed transient ischemic attack provoked the excitation of the left temporal area and the cascade hypothesized above. These events may have been precipitated by the lesion in the superior parietal lobe [9], which presented as transient monolimb weakness in our case.

This case suggests that clinicians should consider the possibility of a seizure in the differential diagnosis when patients present with transient monolimb weakness in old age, and diagnostic tests for epileptic seizures, such as an EEG and ictal SPECT, may help to diagnose this treatable condition.

## Disclosure

All authors have no conflicts of interest to declare.

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