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Todd's Paresis as A Postictal Phenomenon in Post-Traumatic Epilepsy: A Case Report

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ABSTRACT

Introduction: Todd's paresis is defined as a paralysis state after an epileptic seizure that lasts for several hours to days and recovers completely afterwards. It usually manifests as a transient hemiparesis after a focal or generalized seizure. Todd's paresis cases and its mechanisms were still widely discussed. **Case:** A 48-year-old man with a history of post-traumatic epilepsy presented with right-sided hemiparesis and facial weakness after a general motor tonic-clonic seizure. The hemiparesis was located contralateral to the post-traumatic lesion in the left frontal lobe, which was suspected to be the seizure focus. The postictal weakness resolved completely after 30 hours without any specific intervention. It was revealed that the patient had been having several seizures beforehand after the epidural and subdural hematoma due to head trauma. He was diagnosed with Todd's paresis as a postictal condition in the context of post-traumatic epilepsy. **Conclusion:** Todd's paresis should be considered a diagnosis in patients with seizure and stroke-like syndromes, such as hemiparesis, due to its similar manifestations, especially if the symptoms resolve within hours. Several mechanisms have been suggested as the pathophysiology of Todd's paresis, including neuronal exhaustion, active inhibition, and postictal hypoperfusion.

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INTRODUCTION

In 1849, Robert Todd initially described a case of hemiplegia following seizures that lasted for several hours or days. This condition came to be called "Todd's paresis" or "Todd's paralysis." Todd's paresis was originally described as postictal paralysis, a paralytic state that remains after an epileptic convulsion affecting only one side or one limb. The paralysis lasts for several hours or days after the convulsion, but full recovery is expected.[1] Todd reported nine cases of epileptic hemiplegia in patients ranging from children to the elderly. The clinical manifestations of the seizures varied, ranging from unilateral seizures lasting several minutes to sudden falls with foaming at the mouth, or were described only as fits and numbness. The postictal states usually involved weakness in accordance with the site of the seizure. When the seizure occurred on the patient's entire left side, the postictal state was left hemiplegia including facial paralysis. The durations of postictal state varied from twenty minutes to a few days.[2]

The studies on Todd's paresis were continued by several neurologists. In 1869, Alexander Robertson reported four cases of paralysis lasting for a few hours or days due to severe convulsions. John Hughlings Jackson also acknowledged epileptic hemiplegia following a convulsion. However, in the first half of the 20th century, there were only a few significant studies on postictal paralysis.[1]

A study by Rolak *et al*. evaluated 229 patients with generalized tonic-clonic seizures, of whom fourteen were identified as having transient focal neurological deficits suspected to be Todd's paresis. This study explained the clinical manifestations of the weakness, the duration of both epilepsy and paralysis, and the causes of seizures.[3] Gallmetzer *et al*. reported on the frequency and duration of postictal paresis in patients undergoing video-EEG monitoring.[4] In 2014, Yang *et al*. examined the clinical features of post-epileptic dysfunction in seven patients, including Todd's paresis.[5]

Todd's paresis is a syndrome characterized by significant regional limb weakness following seizure activity in the contralateral motor cortex. This condition typically lasts from minutes to several days before complete resolution of symptoms. Its manifestations may involve one limb, one side of the body, or a wide range of manifestations.[6,7] The mechanism of Todd's paresis is still widely discussed and suspected to be multifactorial event. Several theories regarding patomechanism of Todd's paresis include neuronal exhaustion, anoxia, active inhibition, and hypoperfusion.[2,8,9]

Todd's paresis is one of the most common post-epileptic dysfunctions.[5] Although older studies reported a relatively low prevalence, no recent studies has further explored this topic. Gallmetzer *et al*. reported an incidence of 13.4% (44 patients) among 328 samples, with 34.1% of those patients (15 patients) experiencing recurrent postictal paresis.[4] An older study by Rolak *et al*. found Todd's paresis in 6.1% (14 patients) out of 229 samples.[3]

Our case describes a manifestation of Todd's paresis as a postictal phenomenon of post-traumatic epilepsy. This study also provides a review of the history and pathomechanisms of Todd's paresis.

CASE

A 48-year-old male patient was referred to the Neurology Department due to seizures characterized by stiffness and spasms of all extremities, or by a generalized tonic-clonic seizure lasting approximately five minutes and then stopped. Following the seizure, the patient seemed to have difficulty picking up a spoon with his right hand. On physical examination, right-sided supranuclear facial nerve paresis and hemiparesis with a grade of 2 were shown, along with a positive right Babinski plantar reflex.

The patient had a history of head trauma 9 prior, resulting in epidural hematoma (EDH) and a subdural hematoma (SDH) in the left frontal region; however, he refused surgery. Since then, the patient started having seizures. The last seizure occured two months ago, when he experienced a similar seizure at home. Therefore, he was diagnosed with post-traumatic epilepsy but had not started any antiseizure medication.

The complete hematologic examination was within normal limits, with only a slight increase in lymphocytes (14.36 x 10^3/µL). The blood glucose level was 244 mg/dL, and the electrolyte measurements were 138 mmol/L of sodium, 4.9 mmol/L of potassium, and 108 mmol/L of chloride. The serum lactate concentration was 0.9 mmol/L. These laboratory results did not meet the threshold to induce an acute symptomatic seizure.

The current head computed omography (CT) scan revealed a hypodense lesion in the left frontal lobe. The current hypodense lesion was found in the same area as the previous trauma's lesion. As a result, this lesion was suspected to be the focus of the seizure.

An acute EDH with a volume of 23 cc was spotted in the right occipital region, caused by blunt trauma to the back of the head when falling during the seizure. This lesion caused a decrease in consciousness during hospitalization. Therefore, he underwent a craniotomy procedure.

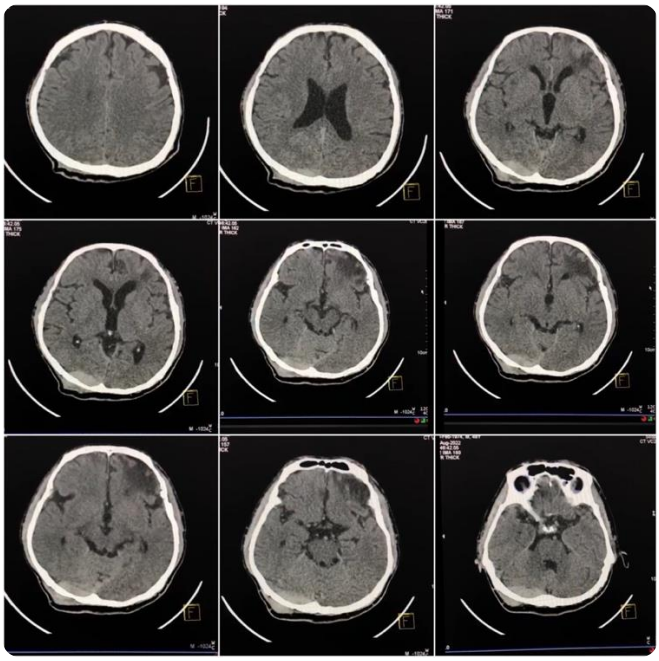

Figure 1. Head CT scan showed a chronic hypodense lesion in the left frontal region and acute EDH in the right occipital region.

The patient regained consciousness thirty hours. He became restless and moved every limb, showing the right hemiparesis had resolved. There were no neurologic deficits found during the physical examination. No recurrent seizures were observed. Therefore, the patient was diagnosed with Todd's paresis due to post-traumatic epilepsy and was prescribed a maintenance dose of phenytoin.

DISCUSSION

The clinical manifestations of Todd's paresis are highly variable.[5] Todd's paresis affects both the upper and lower limbs in approximately 50% of the cases, compared with 21.4% of cases that affect the face as well as the upper and lower limbs. Manifestations involving only the upper limbs, lower limbs, or face are less common.[1,4] This finding is supported by other studies, which mostly report hemiparesis or hemiplegia.[3]

A study by Yang *et al*. explored other clinical manifestations of postictal states accompanying Todd's paresis, such as aphasia, cognitive disorder, gaze palsy, and hemianopsia.[5] Dague *et al*. and Qureshi *et al*. reported cases of opthalmoparesis and gaze palsy after unprovoked seizure, which returned to normal within several days to weeks.[10,11] Cranial nerve deficits, such as slurred speech and facial dropping, have also been reported as Todd's paresis in recurrent generalized seizures.[12]

A case was reported of right-sided hemiplegia lasting for 24 hours after a generalized tonic-clinic seizure following an acute head trauma.[13] This case was similar to ours, in which the patient had post-traumatic epilepsy accompanied by Todd's paresis. The patient's postictal state included right-sided hemiparesis and facial nerve weakness.

The duration of postictal paresis depends on the type of seizure, the presence of structural damage, and other symptoms. Generalized tonic-clonic seizures usually have longer postictal durations, ranging from 30 minutes to 36 hours, with an average of 15 hours after the seizure. The duration is prolonged in patients with brain damage, especially those with stroke, brain tumors, or metabolic disorders, due to impaired synaptic transmissions.[1,14] In this case, it was normal that the duration of the postictal paresis would exceed the average, which was 30 hours, due to the structural damage from the trauma.

Todd's paresis was caused mostly by structural damage, such as cerebral infarction or hemorrhage, meningioma, and embolism. Chronic alcoholism and alcohol withdrawal have also been noted as causes of seizures prior to postictal paresis.[5] Untreated epilepsy may contribute to the incidence of Todd's paresis. In a case report, a patient with Todd's paresis had a history of epilepsy since the age of 10 but was noncompliant with antiepileptic drug therapy.[15] This study reported an untreated case of post-traumatic epilepsy due to brain trauma. The brain lesion manifested as structural damage, thereby increasing the risk of Todd's paresis. The paresis was on the right side, contralateral to the damage on the left frontal lobe. This finding is consistent with a previous study in which postictal paresis in 44 patients was always contralateral to the seizure focus.[4]

Many theories have been developed regarding the pathomechanism. The first and oldest, described by Todd himself, is the theory of neuronal exhaustion and anoxia. A lesion in the brain, such as a tumor or foreign body, was thought to cause instability of nearby cells and started a discharging lesion with abundant energy during the seizure. The excessive neuronal discharge led to exhaustion of nerve fibers. After the discharge ceased, the spasm reduced and postictal weakness appeared.[2] A metabolic phenomenon, such as increased lactic acid levels, could also lead to cortical anoxia and cerebrovascular dysfunction.[4,16] However, this theory is now considered outdated.

Active inhibition after seizures may contribute to transient weakness. Local electrical inhibition works to relieve the overactive neurons and cease the seizure. This inhibition affects the brain area responsible for controlling postictal movement, resulting in paresis.[15,17] Selective hyperpolarization during the refractory resulted in inhibition, which prevented effective neuronal coupling and causing postictal symptoms. Additionally, elevated extracellular

potassium levels also prevented depolarization and suppressed the neuronal activity.[8]

Postictal hypoperfusion had been proposed as an underlying mechanism of brain abnormalities and behavioral dysfunction following a seizure. Magnetic resonance perfusion (MRP) demonstrated a reversible decrease in regional cerebral blood volume and flow, along with an increase in mean transient time (MTT), in the right frontal lobe after an incident of transient left hemiparesis following a seizure. However, the 24-hour follow-up MRP showed total resolution of the abnormalities.[9] Farrell *et al*. reported about a 50% drop in local blood flow following evoked hippocampal seizures in rats, lasting for more than an hour, mediated by local vasoconstriction of hippocampal arterioles. They observed forelimb weakness after seizures elicited in the motor cortex, which recovered once the rats were no longer hypoxic. Clinically, the confirmed these findings by using MRI to measure postictal perfusion within one hour following spontaneous seizures in 10 patients, and found a decrease in cerebral blood flow, with severity correlating with seizure duration.[7,18]

Anatomic alterations following epilepsy contributed to the postictal vascular hypoperfusion and symptoms. These alterations involve changes in blood-brain barrier (BBB) permeability, formation of new capillaries, central inflammation, increased astrocyte count, axonal sprouting, and neuronal loss. These changes occur in both epilepsy and stroke, which explains the similar manifestations observed in stroke and postictal weakness. Traumatic brain injury also disrupts BBB tight junction integrity, followed by immune cell activation and pro-inflammatory cytokine release. Inflammation activates the proliferation of glial cells, a common feature in epilepsy. Impaired astrocytes due to damage in brain structure contributed to a mismatch of metabolism and perfusion, resulting in hypoxia.[7,8]

There are no standardized examinations for diagnosing Todd's paresis. The diagnosis is made by history taking and symptomatology, supported by findings from imaging and electroencephalograpy (EEG) that may reveal structural and functional damage. Obtaining a detailed history of seizures or epilepsy, the patient's baseline neurological condition, and risk factors is important in analyzing the probability of Todd's paresis.[1]

CT perfusion (CTP), magnetic resonance perfusion (MRP), and magnetic resonance imaging (MRI) contribute to determining the etiology of hypoperfusion and hypoxia. Reversibility of hypoperfusion on perfusion imaging is usually observed in Todd's paresis.[9,14,15] EEG in Todd's paresis commonly presents with focal or generalized slowing activity, which returns to normal after the resolution of paresis. Previous cases have reported ictal activity or focal slowing without epileptiform activity, both of which resolved within two weeks on follow-up.[9,19]

Laboratory examinations are essential to evaluate risk factor. In patients with neurologic deficits mimicking stroke, the presence of electrolyte disturbances such as hypomagnesemia, hypokalemia, or hypocalcemia is often associated with seizures, which may lead to a diagnosis of Todd's paresis rather than stroke.[12] According to the hypoxia theory in Todd's paresis, evaluating partial pressure of oxygen (pO_2) levels is important for assessing the severity of hypoxia, which affects brain structure and the duration of paralysis. Furthermore, the production of proinflammatory biomarkers, including tumor necrosis factor alpha (TNF-α) and interleukin-1 beta (IL-1β), may contribute to recurrent episodes of postictal hypoxia.[7]

Todd's paresis was commonly misdiagnosed as acute ischemic stroke because of its similar presentation to stroke. The symptoms of Todd's paresis vary depending on the location of seizure focus; for example, a seizure originating in the motor cortex typically causes hemiparesis. In contrast, the presentation of acute ischemic stroke depends on the infarct location, size, and duration. Stroke symptoms vary and present as sudden headache, unilateral weakness, confusion, aphasia, or visual deficits, which may worsen as time progresses.[20]

From the diagnostic test, MRP and CTP showed mean transit time (MTT) as a sensitive indicator of acute cerebral ischemia due to large-vessel occlusion or stenosis. In Todd's paresis, symmetric hypoperfusion was commonly observed in the MTT, which indicated that the hypoperfusion might not be caused by focal lesion of ischemic stroke, and it usually resolved after the Todd's paresis period has ended.[1,9,15] MRI examination in a case study of prolonged Todd's paresis mimicking stroke demonstrated chronic signal intensity alterations in the pons and midbrain, caused by recurrent seizures since childhood.[14] Another case involved a female patient with right-sided paralysis and a history of right-sided seizures. Her MRI revealed a widespread cortical diffusion restriction in the left hemisphere, which did not match any vascular territory for stroke, and was therefore diagnosed with Todd's paresis.[15]

Treatment of Todd's paresis is mainly supportive and symptomatic, as it typically resolves without intervention. Intubation may be required in patients who are unable to protect their airway. The usual antiepileptic medications are needed for maintenance of epilepsy and prevent recurrent seizures or postictal Todd's paresis.[1,6,8] Farrell *et al*. discovered that drugs targeting cyclooxygenase-2 (COX-2) and L-type calcium channels were able to prevent postictal hypoxia without altering the seizure duration;

however, further clinical trials are yet to be done.[7,18]

This study presents a case of Todd's paresis in post-traumatic epilepsy, a condition that is often underdiagnosed or misdiagnosed as stroke. We also provide a concise review of its history, clinical manifestations, pathomechanism, and diagnosis. The lack of follow-up examinations, such as advanced imaging and EEG, was a limitation of this case report due to limited resource. Brain perfusion imaging should be performed to compare cerebral blood flow during Todd's paresis and after its resolution.

CONCLUSION

Todd's paresis should be considered a postictal phenomenon in patients presenting with stroke-like symptoms and a history of seizures. It most commonly presents as hemiparesis but may also manifest with cranial nerve or neurocognitive deficits. The duration and clinical manifestation of postictal Todd's paresis vary depending on the structures involved. Several theories have been developed regarding its pathomechanism, with postictal hypoperfusion being one of the more recently proposed mechanisms. There is no specific treatments for Todd's paresis; however, future research on the management of hypoperfusion and hypoxia after seizures is still needed to prevent the occurrence of Todd's paresis.

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Conflict of Interest
There were no conflict of interest.

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Author Contributions
NMS initiated the concept and idea for paper. IGAAAY drafted the manuscript, which was revised and finalized by NPAPM and NMS.

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