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# Todd's Paresis as A Post Ictal 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 history of post-traumatic epilepsy presented with right-sided hemiparesis and facial weakness after a general motor tonic-clonic seizure. The location of the hemiparesis was contralateral to the post-trauma lesion in left frontal lobe which was suspected to be the focus of the seizure. 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. The patient was diagnosed as Todd's paresis following a post ictal condition in patient with post-traumatic epilepsy. **Conclusion:** Todd's paresis should be considered as a diagnosis in patients with seizure and stroke-like syndrome such as hemiparesis due to its similar manifestations, especially if it resolves within hours. Several mechanisms were suggested to be 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 which 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, paralytic state remains after the epileptic convulsion that affected only one side or one limb. The paralysis stays for some hours or days after the convulsion but will perfectly recover.[1] Todd reported nine cases of children to elderly with epileptic hemiplegia. The clinical manifestation of the seizures varied from unilateral seizures lasting for several minutes, sudden fall with foaming mouth, or described only as fits and numbness. The postictal states were usually weakness in accordance with the site of the seizure. When the seizure presented in patient's whole left side, the postictal state was left hemiplegia including facial paralysis. The durations of postictal state varied from twenty minutes up to a few days.[2]

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

A study by Rolak et al. in 1992 evaluated 229 patients with generalized tonic-clonic seizures, which fourteen were identified of having transient focal neurological deficits suspected as Todd's paresis. This study explained the clinical manifestations of the weakness, duration of epilepsy and paralysis, and the causes of seizures.[3] Gallmetzer *et al*. described the frequency and duration of postictal paresis in patients during video-EEG monitoring.[4] In 2014, Yang *et al*. explored the clinical features of post-epileptic dysfunction among seven patients, including Todd's paresis.[5]

Todd's paresis is a syndrome associated with significant regional limb weakness following seizure activity in the contralateral motor cortex. This condition usually lasts from minutes to hours to days before complete resolution of the symptoms. Its manifestation can affect one limb or half of the body or another 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 were neuronal exhaustion and anoxia, active inhibition, and hypoperfusion.[2,8,9]

Todd's paresis is one of the most common post-epileptic dysfunctions.[5] The prevalence of Todd's paresis was relatively low in older studies, however there were no novel research exploring regarding this. A study by Gallmetzer *et al*., reported that the incidence of Todd's paresis was 13.4% or 44 patients among 328 samples, and 34.1% of those patients or 15 patients were having recurrent postictal paresis.[4] In an older study by Rolak *et al*., Todd's paresis was found in 6.1% or 14 patients out of 229 samples.[3]

Our case presented a manifestation of Todd's paresis as postictal phenomenon of post-traumatic epilepsy. A review regarding the from the history and pathomechanisms of Todd's paresis were also provided in this study.

## CASE

A male patient, 48 years old, was consulted to the Neurology Department due to seizure, with a pattern of stiffness and spasms throughout the extremities or general motor tonic-clonic seizure that lasted for 5 minutes then stopped. After the seizure, the patient seemed to have difficulty picking up the spoon with his right hand. On physical examination, right sided supranuclear facial nerve paresis and hemiparesis with grade of 2 were shown. There was positive right Babinski plantar reflex.

The patient had a history of previous head trauma, namely epidural hematoma (EDH) and subdural hematoma (SDH) in the left frontal region, 9 months before, however he refused the operative procedure. Since then, the patient started having seizures. The last seizure was 2 months ago, where he had a similar seizure at home. Therefore, he was diagnosed with post-traumatic epilepsy, however he had not started antiseizure medication.

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

Based on the current head computerized tomography (CT) scan, a hypodense lesion was found in the left frontal lobe. The location of the current hypodense lesion was in accordance with the site of lesion from the previous trauma. Therefore, this lesion was suspected to be the focus of the seizure.

Acute EDH with 23cc volume was spotted in the right occipital region, due to the blunt trauma in the back of his head when falling during the seizure. This lesion led to the decrease of consciousness at the hospital. Therefore, he underwent craniotomy procedure.

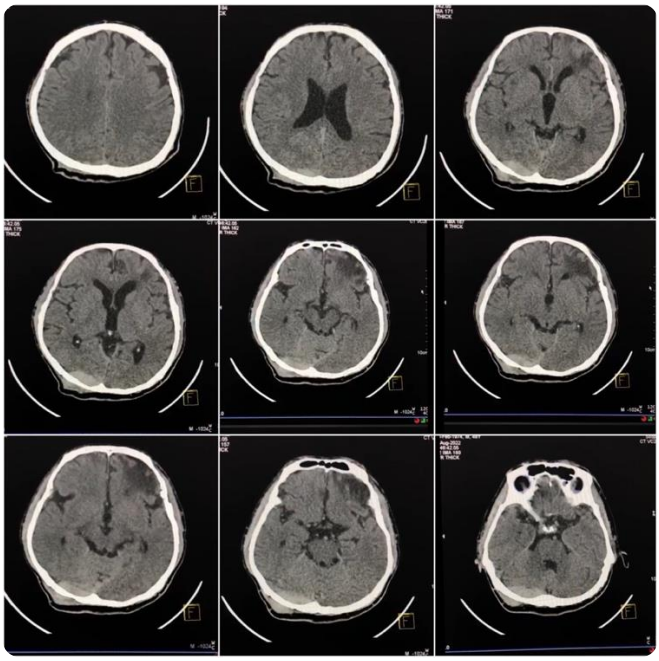

Figure 1. Head CT scan showed a chronic hypodense lesion in the left frontal region and acute EDH in the right occipital region.

Thirty hours later, the patient regained his consciousness. He started to get restless and moved all his extremities, showing the right hemiparesis had resolved. On physical examination, no neurologic deficits were observed. There were no recurrent seizures. Therefore, this patient was diagnosed with Todd's paresis due to post-traumatic epilepsy. He was given maintenance dose of phenytoin for the epilepsy.

## DISCUSSION

The clinical manifestations of Todd's paresis were highly variable.[5] Todd's paresis affected both the upper and lower limbs in 50% of the cases, compared to 21.4% that affected the face and upper and lower limbs. The manifestations on the upper limbs, lower limbs, or face only was less common.[1,4] This finding was supported by the other studies which mostly found hemiparesis or hemiplegia.[3]

A study by Yang et al. focused on exploring another clinical manifestation of postictal states which accompanied Todd's paresis, such as aphasia, cognitive disorder, gaze palsy, and hemianopsia.[5] Dague et al. and Qureshi et al. reported a case opthalmoparesis and gaze palsy after unprovoked seizure which returned to normal several days and weeks later.[10,11] Cranial nerves deficits such as slurred speech and facial dropping were also reported as Todd's paresis in recurrent generalized seizures.[12]

A case reported an incident of right-sided hemiplegia lasting for 24 hours after a generalized tonic-clinic seizure following an acute head trauma.[13] This case was similar with our case report where there were post-traumatic epilepsy and Todd's paresis. The patient's postictal state were right-sided hemiparesis and facial nerve weakness.

The duration of postictal paresis depends on the type of seizure, structural damage, or other symptoms. General tonic-clonic type of seizure usually had longer postictal durations from 30 minutes to 36 hours with an average of 15 hours after the seizure. There is a prolonged duration in patients with brain damage, especially those with stroke, brain tumors, or metabolic disorders due to impaired synaptic transmissions.[1,14] In this case report, it was natural that the duration of the postictal paresis was above the average, which was 30 hours, due to the structural damage from the trauma.

Structural damage such as cerebral infarction or hemorrhage, meningioma, and embolism played an important role in the causation of Todd's paresis. Chronic alcoholism and alcohol withdrawal were also noted as the cause of seizure before the postictal paresis.[5] Untreated epilepsy might contribute to the incidence of Todd's paresis. In a case report, patient with Todd's paresis had a history of epilepsy since the age of 10 years but was not compliant in taking the antiepileptic drug.[15] This study reported an untreated post-traumatic epilepsy due to brain trauma. The brain lesion manifested as structural damage, hence increasing the risk of Todd's paresis. The paresis was on the right side, contralateral from the damage on the left frontal lobe. This is in accordance with the study where postictal paresis in 44 patients was always contralateral to the seizure focus.[4]

There were many developing theories regarding the pathomechanism. The first and oldest theory was neuronal exhaustion and anoxia described by Todd himself. A lesion in the brain, such as tumor or foreign body, caused instability of cells near it and started a discharging lesion with abundant energy during the seizure. The excessive discharge of neuron lead to the exhaustion of nerve fibers. After the discharge had ceased, the spasm reduced and postictal weakness appeared.[2] Metabolic phenomenon as in the increased lactic acid levels could also lead to cortical anoxia and cerebrovascular dysfunction.[4,16] However, this theory was said to be outdated.

Active inhibition after seizures might contribute to the transient weakness. Local electrical inhibition worked to relieve the overactive neurons and ceased the seizure. This inhibition affected the brain area that controlled postictal movement, hence causing the paresis.[15,17] Selective hyperpolarization during refractory period led to inhibition which prevented effective neuronal coupling and causing postictal symptoms. Increase of extracellular kalium also prevented depolarization and suppressed the neuronal activity.[8]

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

Anatomic alterations following epilepsy contributed to the postictal vascular hypoperfusion and symptoms. The alterations involving changes in blood-brain barrier (BBB) permeability, formation of new capillaries, central inflammation, increased astrocyte count, axonal sprouting, and neuronal loss. These changes occurred in epilepsy and stroke, hence the similar manifestation in stroke and postictal weakness. Traumatic brain injury also caused the insult in BBB tight junction integrity, followed by immune cells activation and pro-inflammatory cytokines release. Inflammation activated the proliferation of glial cells, which is a common feature in epilepsy. Impaired astrocytes due to damage in brain structure contributed to the mismatch of metabolism and perfusion, thus leading to hypoxia.[7,8]

There are no standardized examinations to diagnose Todd's paresis. The diagnosis is made by history taking and symptomatology, as well as the involvement of structural and functional damage from imaging and electroencephalography (EEG). Detailed history taking regarding history of seizures or epilepsy, the patient's baseline condition, and risk factors are important in analyzing the probability of Todd's paresis.[1]

CT perfusion (CTP) or MRP and magnetic resonance imaging (MRI) contribute to finding the etiology of hypoperfusion and hypoxia. Reversibility of hypoperfusion in the perfusion imaging is usually shown in Todd's paresis.[9,14,15] EEG in Todd's paresis commonly presents with focal or generalized slowing activity which will return to normal after the resolution of paresis. Previous cases reported ictal activity or focal slowing without epileptiform activity which resolved in the 2 weeks follow-up.[9,19]

Laboratory examinations are essential to evaluate the risk factor. In patient with neurologic deficits mimicking stroke, the presence of electrolyt disturbances such as hypomagnesemia, hypokalemia, or hypocalcemia tend to be associated with seizures which might lead the diagnosis to Todd's paresis instead of stroke.[12] Referring to the hypoxia theory in Todd's paresis, evaluating the partial pressure of oxygen ($pO_2$) levels is important to assess the severity of hypoxic condition which impact the brain structure and the duration of paralysis. Production of proinflammatory biomarkers such as tumor necrosis factor alpha (TNF-α) and Interleukin-1 beta (IL-1β) might contribute to recurrent episodes of postictal hypoxia.[7]

Due to its similar presentation with stroke, Todd's paresis was commonly misdiagnosed as acute ischemic stroke. The presenting symptoms of Todd's paresis depend on the seizure foci. A seizure in the motor cortex will cause hemiparesis. Meanwhile, acute ischemic stroke depends on the infarct location, size, and duration. Stroke symptoms varied and can presents as sudden headache, unilateral weakness, confusion, aphasia, or visual deficits which may worsen as time progresses.[20]

From 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 common in the MTT which indicated the hypoperfusion was might not be caused by focal lesion of ischemic stroke, and it usually resolved after the Todd's paresis period was over.[1,9,15] MRI examination in a case study of prolonged Todd's paresis mimicking stroke demonstrated chronic signal intensity alterations in pons and midbrain due to the recurrent seizures since childhood.[14] Another case showed a female with right side paralysis and history of right-sided seizure. Her MRI revealed a widespread cortical diffusion restriction in the left hemisphere which did not match any vascular territory for stroke, hence diagnosed with Todd's paresis.[15]

Treatment of Todd's paresis is mainly supportive and symptomatic as it resolves without any intervention. Intubation is required in patients who have problem protecting their airway. Usual antiepileptic medication is needed for maintenance of the epilepsy to prevent recurrent episodes of seizure 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 studies in clinical trials are yet to be done.[7,18]

This study reported a case of Todd's paresis in post-traumatic epilepsy which is often underdiagnosed or even misdiagnosed as stroke. We also reported a concise review regarding its history, clinical

manifestations, pathomechanism, and diagnosis. Lack of follow-up examinations such as advance imaging and EEG became the limitation of this case report due to limited resource. Brain perfusion imaging should be evaluated to compare the cerebral blood flow during Todd's paresis and after its resolution.

## CONCLUSION

Todd's paresis is important to be considered as a postictal phenomenon in the setting of stroke-like symptoms with history of seizures. Todd's paresis commonly presents as hemiparesis and might also presents with cranial nerves neurocognitive deficits. The duration and clinical manifestation of postictal Todd's paresis are varied depending on the structures involved. There have been many developing theories regarding the pathomechanism of Todd's paresis, where postictal hypoperfusion is one of the mechanisms which was recently proposed. There were no specific treatments for Todd's paresis, however future researches regarding management of hypoperfusion and hypoxia after seizures are still needed to prevent the occurence of Todd's paresis.

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### 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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