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# POSTICTAL TODD'S PARALYSIS AS A MANIFESTATION OF POSTTRAUMATIC ENCEPHALOPATHY AND A "MASK" OF STROKE: CLINICAL OBSERVATION

***Alexey Gorbunov*[1], *Dmitry Parshin*[2] ✉,**
***Maria Kalugina*[3], *Zhanna Shishkina*[3],**
***Alexey Loktev*[3], *Svetlana Shirokova*[4]**

[1]*Tambov State Technical University, Tambov;*
[2]*Astrakhan State Medical University, Astrakhan;*
[3]*Derzhavin Tambov State University, Tambov;*
[4]*Medical center "Medlab", Tambov, Russia*

✉ ***parshin.doc@gmail.com***



## ABSTRACT

Todd's paralysis is rare phenomenon, but its identification is of key diagnostic and therapeutic value. The authors present a clinical observation of post-traumatic encephalopathy accompanied by Todd's paralysis. The main differential diagnostic criteria for post-traumatic encephalopathy with cerebral paroxysms in the form of Todd's paralysis and stroke syndromes have been identified: closed craniocerebral injury in history; epileptiform paroxysms; a clear connection between epileptic seizures and trauma; characteristic "scarcity" of the clinical picture; stereotype paroxysms and rapid regression of symptoms; electroencephalographic study; absence of significant pathological changes on magnetic resonance tomo- and angiography; dissociation between the presence of a neurological deficit and magnetic resonance neuroimaging data; cumulated clinical analysis.

***Keywords:*** *post-traumatic encephalopathy, Todd's paralysis, postictal paralysis, acute cerebrovascular accident, stroke, differential diagnosis, craniocerebral injury*

## INTRODUCTION

Chronic traumatic encephalopathy is a neurodegenerative disease associated with repetitive head trauma and characterized by perivascular accumulation of hyperphosphorylated tau protein deep in the cortical sulci [1–3]. This pathology can be definitely diagnosed only post mortem, and the cellular mechanisms of the disease onset have yet to be clarified. Understanding the full range of pathological changes currently identified in post-traumatic encephalopathy is necessary to identify new areas for further research [4-7].

Although postictal neurological deficit has long been known, its underlying causes are not fully understood. A wide range of neurological and psychiatric symptoms have been described, which is an important diagnostic problem, especially for emergency physicians [8-10]. At the same time, the possibility of acute hemiparesis in the form of Todd's paralysis in patients with anamnestic signs of a closed craniocerebral injury is described by isolated observations. At the same time, Todd's phenomenon being a relatively rare, its correct identification is of key diagnostic and therapeutic importance as a stroke mimics [11-14].

**The purpose of the study** was to describe a clinical case reflecting the complexity of assessing

etiopathogenesis and the clinical features of the Todd's phenomenon.

## CLINICAL CASE

Patient Sh., 39 years old, professional track and field athlete. He sharply got sick on 27.12.2001, when "among full health" he felt a moderate weakness of the right limbs. On 28.12.2001, he was examined and then sent to an emergency hospital with a diagnosis of acute cerebrovascular accident. From 30.12.2001 to 21.01.2002 he received inpatient treatment in the neurological department with a diagnosis of "Acute ischemic cerebrovascular accident in the left hemisphere". On 10.01.2002, he underwent magnetic resonance imaging (MRI) of the brain. Conclusion: no focal changes in the cerebral cortex, brainstem and cerebellum. Median structures were not displaced. The ventricles were not dilated, symmetrical. The fourth ventricle was in the midline. The pituitary gland was not enlarged. Electroencephalography (EEG) was not recorded. At the same time, right-sided hemiparesis "almost regressed" on the day 10-14 from the onset of the disease.

From 14.03.2002 to 01.04.2002, he received a planned course of inpatient treatment with a diagnosis of "Consequences of acute ischemic cerebrovascular accident in the left hemisphere". An EEG was performed: "Mild cerebral changes with mild diffuse dysrhythmia of cortical activity". From 20.11.2002 to 04.12.2002 he was treated in the neurological department of the day hospital with a diagnosis of "Dyscirculatory encephalopathy stage 2". On 06.12.2002, he had a MR-angiography of intracranial vessels: "The diameter of the vessels of the arterial circle is within the normal range: vertebral arteries - 2.2 mm (right), 3.1 mm (left) - slight asymmetry; basilar artery - 2.7 mm; internal carotid arteries in the siphon area - 5.2 mm (right), 4.8 mm (left); middle cerebral arteries - 1.9 mm (right), 1.8 mm (left); posterior cerebral arteries - 2.0 mm (right), 2.1 mm (left); anterior cerebral arteries - 1.6 mm each. Pathological constrictions and dilations of the main vessels, as well as additional vascular formations, are not detected". Further, he did not turn to neurologists - he returned to his former normal life.

On 29.05.2003, weakness of the right limbs developed again for several hours, but didn't last more than one day. From 29.05.2003 to 05.06.2003 he was hospitalized in the neurological department with a diagnosis of "Cerebral vascular crisis associated with the consequences of acute cerebrovascular accident in the left hemisphere". EEG and MRI of the brain were not performed. Once again, he didn't have a follow up by neurologists and doctors, and continued his professional sports activities.

On 17.12.2004, another episode of weakness of the right limbs "again for 3-5 days". From 17.12.2004 to 14.01.2005: inpatient treatment in the neurological department of Tambov with a diagnosis of "Acute ischemic cerebrovascular accident in the left hemisphere with right-sided pyramidal insufficiency". On 12.01.2005, an MRI and magnetic resonance angiography (MRA) of the brain were performed: "No dislocation of the midline structures of the brain. Slight dilatation of the lateral ventricles of the brain. Single small dyscirculatory foci in the subcortical nuclei. Sylviian fissures and cortical furrows are unevenly soldered, partly dilated in a cystic form. Sellar area appears unremarkable. Cerebellar tonsils are on the Chamberlain line. The blood flow in the vessels of the circle of Willis is preserved and symmetrical. Slight tortuosity of the supraclinoid sections of the internal carotid arteries. There is venous vascular congestion, dilatation of the right transverse venous sinus against the background of partial obliteration of the lumen of the left transverse venous sinus." EEG was not performed. From 18.01.2005 to 01.02.2005, he underwent a rehabilitation course at the Khimik sanatorium, where on 01.02.2005, he got "hit in his head by a volleyball during a game", and felt a severe weakness of the right limbs "for 2-3 days". In this connection, from 02.02.2005 to 15.02.2005, he had an inpatient treatment in the neurological department of Tambov with a diagnosis "Discirculatory encephalopathy stage 2 with circulatory failure in the vertebrobasilar system". Then from 15.02.2005 to 23.02.2005, he underwent a repeated course of rehabilitation in a sanatorium. He was referred for sociomedical assessment that defined a disability group 3.

**Past medical history**: at the age of 17 he was beaten by unknown people with a short-term loss of consciousness, he was treated in the neurosurgery department with a diagnosis of "Closed craniocerebral injury. Concussion"; at the age of 19, during military service, "fainting with convulsions twice within several days", after which he was transferred to the reserve; Since then, he has completely quitted consuming alcohol and smoking.

**Neurological picture**: clear consciousness, but cognitive functions slightly reduced, no meningosigns, pupils D = S, weakness of the left facial muscles, no paresis of the extremities and sensory disorders detected, tendon reflexes - D>S, pathological foot signs on the right, stable in the Romberg test, coordinating tests within normal, pelvic functions controlled. Duplex ultrasound scan of the arteries of the aortic arch and their branches (15.03.2005): "The carotid arteries are patent. The carotid arteries pathway without significant deformations. Vessel diameter and wall mobility are within normal limits. The walls of the vessels are even, IMC 0.5 mm, normal. Speed and spectral characteristics unaltered. Vertebral arteries: $V_1$ - $V_2$ segments of the right vertebral artery – 2.5 mm (normal), left vertebral artery – 4.1 mm (normal). The arteries pathway is unaltered; the left vertebral artery is not clearly located at the mouth. No local changes in the spectrum of blood flow were found in the areas accessible for visualization." EEG (05.04.2005): "The

background EEG is dominated by the α-rhythm, frequency 10 Hz, amplitude 67–79 μV. The shape of the waves is pointed. Amplitude modulation is random. Zonal differences are smoothed out due to the presence of α-like fluctuations in the central temporal leads. β-activity is moderate, with a predominance in the frontal areas. Irregular polymorphic slow waves. Sharp fluctuations are recorded in all leads, more pronounced in the left anterior-temporal and central-parietal regions. When opening the eyes the cortical rhythm is depressed. Photostimulation and hyperventilation result in increased disorganization of the cortical rhythm, bilaterally synchronous flashes of pointed α- and θ-waves, sharp fluctuations in the fronto-central and occipital-parietal regions lasting up to one second."

Based on complaints, life history, course of the disease, analysis of discharge epicrises, data from neuroimaging methods and EEG, the patient was diagnosed with: Post-traumatic partial epilepsy due to mild closed craniocerebral injury with concussion: rare simple partial seizures with postictal Todd's paralysis in the right extremities and rare secondary generalized tonic-clonic seizures.

The patient was prescribed Depakine Chrono 300, one tablet twice a day. In the future (within 18 months), with ongoing therapy, there are no complaints, there are no attacks of "transient and regular weakness of the right limbs". The level of valproic acid in the blood: 95.8 mcg/ml (05.07.2005) and 60.8 mcg/ml (23.11.2005) (norm: 50 - 100 mcg/ml). EEG: "Mild cerebral changes with mild diffuse dysrhythmia of cortical activity, preserved regionality and activation reaction."

## CONCLUSION

Thus, the crucial differential diagnostic criteria for post-traumatic encephalopathy with cerebral paroxysms in the form of Todd's paralysis and stroke syndromes are:

1. history of closed craniocerebral injury;
2. a clear connection between epileptic seizures and trauma;
3. history of epileptiform paroxysms;
4. characteristic "scarcity" of the clinical picture;
5. stereotype paroxysms and rapid regression of symptoms;
6. electroencephalographic study;
7. absence of significant pathological changes on MRT and MRA;
8. dissociation between the presence of a neurological deficit and magnetic resonance neuroimaging data;
9. cumulated clinical analysis.

However, a diagnosis of Todd's postictal paralysis is quite possible.

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