Intern Emerg Med (2013) 8:425–426
DOI 10.1007/s11739-013-0904-3

CASE RECORD

Nothing as it seems

Todd's paralysis

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Received: 17 December 2012 / Accepted: 23 January 2013 / Published online: 12 February 2013

Case presentation

Dr. Tobaldini: An 82-year-old woman was brought by her daughter to the Emergency Department (ED) because she was found on the ground, confused, with right ocular deviation, dysarthria, left hemisoma hyposthenia, attention and executive functions' deficit.

The past medical history was significant for Lewy bodies dementia and systemic hypertension. Her medications included quetiapina, selegina, zolpidem, escitalopram, acetylsalicylic acid, atenolol, enalapril and amlodipina.

On admission to ED, the patient was confused and apyretic; blood pressure was 235/135 mmHg, $SatO_2$ 97 % in ambient air, heart rate 95 beats/min, respiratory rate 26/min and plasma glucose 101 mg/dl. General physical examination revealed pulmonary bibasilar crackles. Neurological examination showed a left sided inattention, right ocular deviation, left partial vision paralysis, deficiency of the VII left cranial nerve, left hemisoma hyposthenia, positive Babinsky reflex on the left side, deep pain stimulus extinction on the left side; the NIH Stroke Scale (NIHSS) score was 11.

Laboratory blood tests, ECG and a chest X-ray study were normal; the first brain CT-scan performed showed a past lacunar ischemia of the left thalamic region but it was negative for acute lesions.

In the ED, the patient was treated with captopril and urapidil for blood pressure control; due to the patient's agitation, iv delorazepam was administered. The patient was admitted to internal medicine service.

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The patient was sleepy but arousable, the physical examination remained unchanged.

Differential diagnosis

Dr. Prado, *Dr. Fiorelli and Dr. Wu*: The patient's neurological features were compatible with an acute stroke; the severe hyposthenia of the left hemisoma suggested an injury in the right cerebral hemisphere. A cerebral hemorrhage was excluded by the first CT-scan performed in the ED that was negative for acute lesions.

A second brain CT-scan after 48 h was mandatory to exclude an ischemic event.

To investigate a possible vascular or cardiac origin of the neurological manifestation, a carotid color doppler ultrasound and an echocardiography should be performed. The execution of transesophageal echocardiogram, to identify possible sources of thrombi, should be carefully evaluated, taking into account the patient's age and clinical condition.

The impairment of consciousness on admission could be interpreted as a consequence of the administration of benzodiazepines in ED.

Preliminary diagnosis

Dr. Prado, *Dr. Fiorelli and Dr. Wu*: Carotid color doppler ultrasound and transthoracic echocardiography were negative. A continuous ECG monitoring did not reveal any episode of atrial fibrillation or other dysrhythmias; transesophageal echocardiogram was not performed due to patient's age and clinical condition.

Surprisingly, the brain CT-scan performed 48 h after the admission to hospital was negative for an acute ischemic

lesion; this result was in contrast with the hypothesis of an acute ischemic stroke causing the neurological features.

Intravenous hydration was administered to facilitate benzodiazepines elimination.

Within 48 h from the admission, there was a complete remission of the neurological findings.

Further investigation

Dr. Sandrone and Dr. Tobaldini: At this time, the diagnosis of acute ischemic stroke was wavering. In fact, the two CT-scans being negative for acute ischemic lesion and with a complete remission of the neurological clinical presentation within 48 h from the admission the presentation was not consistent with the hypothesis of acute cerebral stroke.

Nonetheless, to definitely exclude the presence of acute cerebral lesions not detectable with CT-scan, a brain magnetic resonance (MRI) was planned. The day the MRI was scheduled, the patient's right arm became paretic. The MRI revealed an acute ischemic lesion in left thalamic-capsular region, compatible with the new neurological finding.

Diagnosis and therapy

Dr. Sandrone and Dr. Tobaldini: The MRI finding of an acute injury in left thalamic-capsular region was consistent with the right arm paresis of recent onset, but not compatible with the previous neurological features.

Hence, an EEG was performed, revealing slow temporal abnormalities in the left hemisphere in a context of mild diffuse encephalopathy.

Considering the acute onset of symptoms, the absence of compatible findings in brain-CT and MRI, and the rapid resolution of the neurologic signs, with complete neurological recovery within 48 days, the most likely diagnosis was post-seizure paralysis (Todd's paralysis), and a new left thalamic-capsular ischemia. The second neurological event was the "real" stroke, detectable with brain imaging as left thalamic-capsular ischemia. The patient, completely recovered from the first neurological clinical presentation, showed slow but gradual improvement of the right arm hyposthenia and was referred to a Rehabilitation Centre.

Anticonvulsant therapy was withheld because antiepileptic treatments are generally started after the second convulsive episode.

Discussion

Dr. Prado, *Dr. Sandrone and Dr. Tobaldini:* Seizures and strokes have various clinical manifestations, and their differential diagnosis may be challenging. Features of non-convulsive status epilepticus or post-seizure Todd paralysis may be similar to ischemic stroke. Otherwise, seizures can also be a presenting manifestation of acute cerebral ischemia.

Todd's paralysis is a post-seizure event described for the first time in the nineteenth century. It is defined as a transient weakness and a depression of motor ability, that usually lasts hours to days. Todd's paralysis usually affects one or more limbs and its onset follows a focal seizure. Nevertheless, other neurological manifestations such as neglect, aphasia and psychosis can occur according to the focal area involved [1, 2].

The pathophysiology of Todd's paralysis is not fully understood. Different hypotheses about its pathogenesis have been suggested: neuronal desensitization, neurotransmitter depletion, altered local cerebral blood flow, active suppression and exhaustive neuronal firing [3, 4].

In our patient, the neurological clinical presentation, age and comorbidities (i.e., systemic hypertension) suggested a diagnosis of acute ischemic stroke.

On the other hand, the negativity of two brain CT-scans within 48 h from the onset of symptoms, and the rapid neurological improvement with a complete recovery within 48 h excluded our first hypothesis. It is worth noting that carotid color doppler ultrasound, echocardiography and ECG monitoring did not reveal any possible source of thrombi.

Therefore, these data suggested a possible different cause of the neurological manifestation, such as a Todd's paralysis that followed an unobserved focal seizure; the EEG partially confirmed this diagnosis.

In addition, the cause of the right arm plegia was detected by the brain MRI as an acute ischemic lesion in left thalamic-capsular region.

In conclusion, our diagnoses were Todd's paralysis followed by acute left thalamic-capsular stroke.

Acknowledgments This case record was selected at the 2012 SIMI Annual Congress in Rome.

Conflict of interest None.

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