Delayed Capsular Warning Syndrome after a Recurrent Capsulothalamic TIA

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Abstract

We present a 57-year-old male who presented a right arm paresthesia lasting for 5 minutes. Two hours later he suffered from a right faciobrachial paresthesia lasting for 10 minutes. Cranial MRI didn’t reveal ischemic lesions. The patient was discharged home with acetyl salicylic acid and atorvastatin. Three months later the patient presented two episodes of a right faciobrachial paresthesia lasting for 10 minutes, separated by a few minutes. The patient came to the Emergency Department (ED), where he presented a new episode without a complete recovery. While the patient was in the ED, he presented a clinical fluctuation on two occasions this extending to the leg hypoesthesia and a slight right hemiparesis. Neurological status at the time of admission showed a right faciobrachial hypoesthesia. Cranial MRI at this time showed a left capsulothalamic stroke. Antithrombotic treatment was changed to clopidogrel. Up to the present, he remains clinically stable. This case shows that capsular warning syndrome (CWS) is a broader syndrome than initially described. The clinical evolution of our patient (a CWS that resulted in a left capsulothalamic stroke three months after a recurrent capsulothalamic TIA) may points toward an unstable hemodynamic process with an underlying high degree atherosclerotic disease in small intracranial vessels, such as may occur in large artery disease, such as carotid artery high degree stenosis.

ABBREVIATIONS

ASA: Acetyl Salicylic Acid; CWS: Capsular Warning Syndrome; EEG: Electro Encephalography; ED: Emergency Department; MRI: Magnetic Resonance Imaging; TIA: Transient Ischemic Attack

INTRODUCTION

Capsular warning syndrome (CWS) has been described as a distinct form of TIA that may be clinically located in the region of the internal capsule and which leads to early capsular infarction in a high proportion of cases (most often within 72 hours) [1]. However, the accurate etiology and optimal treatment of the CWS remain unknown.

CASE PRESENTATION

A 57-year-old male, previous smoker was admitted on the 23rd of April 2014 due to a neurological deficit. His father has died due to massive myocardial attack at the age of 60. At 9:30 am the patient suddenly presented, while shaving, a right arm paresthesia lasting for 5 minutes. Two hours later, while driving, he suffered from a right faciobrachial paresthesia lasting for 10 minutes. The patient consulted in another hospital where neurological status in the Emergency Department (ED) was normal. Cranial CT and cardiological studies were all normal (holter and transthoracic echocardiography). Cranial MRI showed a small right thalamic cavernoma and did not reveal ischemic lesions (Figure 1 A). The patient was discharged home with acetyl salicylic acid (ASA) 100 mg per day and atorvastatin 40 mg per day. On the 20th of July 2014, the patient present date 2:00 pm, while at home, two episodes of a right faciobrachial paresthesia lasting for 10 minutes, separated by a few minutes. The patient came to the ED in our hospital, where he presented a new episode without a complete recovery. While the patient was in the ED, he presented a clinical fluctuation on two occasions this extending to the leg hypoesthesia and a slight right hemiparesis. Neurological status at the time of admission showed a right faciobrachial hypoesthesia. Cranial MRI at this time showed a left capsulothalamic stroke. Antithrombotic treatment was changed to clopidogrel. Up to the present, he remains clinically stable. This case shows that capsular warning syndrome (CWS) is a broader syndrome than initially described. The clinical evolution of our patient (a CWS that resulted in a left capsulothalamic stroke three months after a recurrent capsulothalamic TIA) may points toward an unstable hemodynamic process with an underlying high degree atherosclerotic disease in small intracranial vessels, such as may occur in large artery disease, such as carotid artery high degree stenosis.

DISCUSSION

CWS is classically considered as at least three episodes of neurological dysfunction and the time frame during which these
events occur is usually quite brief (most often within 72 hours) [1]. But from the time of its first description, the definition of CWS has become broader, with other territories being affected or lasting longer than 72 hours. In this sense, some authors supported a 7-day stroke risk of 60% in patients with CWS with no strokes at 8-90 days [2]. However, a case of CWS with a number of TIA episodes extended to over fifty times over the course of 219 days has been recently described [3]. Several hypotheses about the etiology of CWS had been described such as cardioembolic [2] hemodynamic or artery-to-artery microemboli within the penetrating vessels themselves [4].

In our case, the first episode did not accomplish the classical definition of CWS, as there were only two neurological transient episodes, which could be considered as left capsulothalamic TIAs. However, a classical CWS that resulted in a left capsulothalamic stroke was seen three months later. This clinical evolution may points toward an unstable hemodynamic process with an underlying high degree atherosclerotic disease in small intracranial vessels, such as may occur in large artery disease, such as carotid artery high degree stenosis. We initiated ASA and statins after the first ischemic event, and it was changed to clopidogrel after the second one, without a new ischemic cerebral event up to the present. We do not know whether the treatment was efficacious or it was the natural course of the disease.

In conclusion, CWS is a broader syndrome than initially described. This case, a CWS that resulted in a left capsulothalamic stroke three months after a recurrent capsulothalamic TIA, may support the pathophysiological mechanism of unstable hemodynamic process with an underlying high degree atherosclerosis of intracranial small vessel disease.

REFERENCES