Subdural Hematoma Simulating a Capsular Warning Syndrome

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Abstract

Capsular warning syndrome (CWS) leads to early capsular stroke in a high proportion of cases. We present a 73 year-old female who suffered from a right hemiparesis with hypoesthesia. Urgent cranial CT, CT-angiography and perfusion CT were normal. A spontaneous clinical improvement was seen. She was admitted with the diagnosis of left sensory-motor lacunar syndrome. Hours later the patient suffered from a clinical worsening. A control cranial CT was performed and was normal. IV sodium heparin was initiated with an improvement (NIHSS 0). In the following days, several clinical fluctuations were seen. A cranial MRI at day 5, showed a laminar left subdural hematoma. CWS is a differential entity which requires a prompt and accurate diagnosis. A cranial MRI should be performed as quickly as possible in all cases of suspected CWS in order to exclude CWS mimics such as, in this case, a subdural hematoma.

ABBREVIATIONS

ASA: Acetyl Salicylic Acid; CT: Computed Tomography; CWS: Capsular Warning Syndrome; ED: Emergency Department; IV: Intravenous; MRI: Magnetic Resonance Imaging

INTRODUCTION

Capsular warning syndrome (CWS) has been described as a distinct form of TIA which leads to early lacunar in a high proportion of cases [1]. However, some CWS mimics have been described [2].

CASE PRESENTATION

A 73 year-old female attended the Emergency Department (ED) due to right paresis. She had high blood pressure without any other cerebrovascular risk factors. The patient had suffered a cold with a frequent and heavy cough over the previous days. She attended the Health Center, where she reported a right hemibody tingling which had begun at 12:00 am. The patient was sent to the hospital where she arrived at 13:15. Neurological status in the ED showed dysarthria, right facial paresis, and a right hemiparesis (4/5) with hypoesthesia (NIHSS 6). Urgent cranial CT, intra and extracranial CT and perfusion CT were found to be normal. A clinical improvement was seen after all the studies were performed in the ED, with the persistence of a slight right leg paresis (NIHSS 1). Intravenous (IV) thrombolysis was discarded. She was admitted to the Stroke Unit with the diagnosis of left sensory-motor lacunar syndrome. Acid acetyl salicylic (ASA) 100 mg/d and atorvastatin 80 mg/d were prescribed. Hours later, in the Stroke Unit, the patient suffered from a clinical worsening and reached the previous neurological status (NIHSS 6). A control cranial CT was performed and was found to be normal (Figure 1A,B). With the suspicion of CWS, IV sodium heparin was initiated with a posterior clinical improvement (NIHSS 0). Two days after admission, the patient suffered from a clinical fluctuation while she was sitting. IV levetiracetam (500 mg/d) was initiated. She remained clinically stable over the next 24 hours. IV sodium heparin and IV levetiracetam was stopped, and ASA 100 mg/d was began. In the following 24 hours she again suffered from a clinical worsening while she was in a sitting position for eating. Neurological status at this time showed dysarthria, right hemiparesis (3-4/5) and right hypoesthesia. Hemodynamic measures were begun and improvement was seen after one hour (NIHSS 0). In the afternoon, the patient suffered from several episodes of paresis and paresthesias. IV levetiracetam was reinitated. During the following 48 hours the episodes were less frequent. Five days after admission, a cranial MRI was performed. It showed a laminar left subdural hematoma (Figure 1C,D). ASA and atorvastatin were stopped. Up to the present the patient remains asymptomatic with levetiracetam 1000 mg c/12 hours.

DISCUSSION

Capsular warning syndrome (CWS) has been described as a distinct form of transient ischemic attack 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]. Several etiologies have been suggested for...
CWS such as cardioembolism, hemodynamic failure and arterial microemboli [3-4]. For this reason, treatment of this syndrome has not been well defined; however, antiplatelet treatment seems to play the main role [4], even with a combination [4] or loading dose [5] of antiplatelet drugs. In the acute phase of CWS, fibrinolytic treatment may be safe, without considering CWS as exclusion criteria for IV thrombolysis [6]. Despite the high infarct incidence in CWS, few recurrences of stroke and cardiovascular events have been described in these patients, this suggesting a favorable outcome in CWS [6].

Our patient accomplished the criteria for a CWS. A cranial CT misdiagnosis of the hemorrhage made this patient a candidate for IV thrombolysis, which was finally discarded because of clinical improvement. Later IV sodium heparin was initiated due to persistent clinical fluctuation, with posterior antithrombotic treatment with ASA. Thus, an increased risk of an intracranial hemorrhage existed. The patient only improved with anticonvulsive treatment as the lesion was cortical and provoked an irritating phenomenon.

In conclusion, CWS is a differential entity which requires a prompt and accurate diagnosis. A cranial MRI should be performed as quickly as possible in all cases of suspected CWS in order to exclude CWS mimics such as, in this case, a subdural hematoma.

REFERENCES


Figure 1 A,B: Cranial CT was found to be normal. C,D: Cranial MRI (FLAIR T2 sequences) showed a right parietal subdural hematoma (white arrows).