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THE ROLE OF EMERGENT SURGICAL DRAINAGE IN THE TREATMENT OF SUBACUTE SUBDURAL HEMATOMAS PRESENTING WITH TRANSIENT NEUROLOGICAL DEFICITS: CASE REPORT

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Geçici Nörolojik Defisitler ile Ortaya Çıkan Subakut Subdural Hematomun Tedavisinde Acil Cerrahi Drenajın Rolü:Olgu Sunumu

Özet

Subakut subdural hematomlarda, geçici iskemik ataklar oldukça nadir görülür. Bu klinik fenomenin patofizyolojisinde hematomun lokal ödem oluşturucu etkisi ve serebral vasküler yapılardaki değişimler yer alır. Atrial fibrilasyon yada geçici iskemik ataklara neden olabilen karotis arterinin tromboembolik hastalıkları ayırıcı tanıda dikkate alınmalıdır. Tedavide ise; oluşan kitle etkisinden kurtulmak için cerrahi drenaj yapılmalıdır. Bu çalışmada; motor afazi ve hemiparezi ile presente olan bir subakut subdural hematom vakasında hematomun boşaltılmasındaki gecikmeden dolayı oluşan nörolojik semptomların kalıcılığı tartışılmaktadır.

Anahtar Kelimeler: Subdural hematom, cerrahi tedavi, geçici iskemik atak..

Summary

Presentation with transient ischemic attacks in subacute subdural hematomas is quite rare. It is a clinical phenomeon in which hematoma mass effect with its own capability of producing local edema formation and the variations in cerebral vascular structures take place in the pathophysiology. Diseases such as atrial fibrillation or tromboembolic disorders of carotid artery, which may lead to transient ischemic attacks should be considered in the differential diagnosis. For treatment, emergent surgical drainage in order to eliminate the mass effect should be performed. In this study, a case of subdural hematoma presenting with motor aphasia and hemiparesis is reported of which neurological symptoms became permanent due to delay in the hematoma evacuation.

Key Words: Subdural hematoma, surgical treatment, transient ischemic

Introduction

Subacute subdural hematomas are almost encountered in the elderly, particularly following a minor head trauma (1,3). It is still on debate today due to controversies in the physiopathology, clinical progress and surgical treatment modalities in use (5). These hematomas may manifest with various symptoms such as headache, paresis, dementia, and epilepsy. An infrequent presentation with transient ischemic attacks has been reported (6). In this study, a case of subacute subdural hematoma in which transient ischemic attack (TIA) became permanent is reported and discussed in the context of the literature.

Case report

72-year-old-man was admitted to our department with the complaints of aphasia and right hemiparesis, which developed suddenly and continued for twenty minutes. The patient had history of trauma. He had not nausea or epileptic seizure, nor had the symptoms of hypertension, diabetes, heart failure or other types of systemic diseases. On neurological examination, Glasgow Coma Scale was found as 15 points. He had motor aphasia and right hemiparesis dominating on the lower extremity. Plantar reflexes were extensor on both sides. Other systemic and laboratory findings were unremarkable. Electrocardiographic and echocardioagraphic findings were interpreted as normal. Carotid pulsations were symmetrical bilaterally and no bruits were noted. On computerized tomography (CT), a subacute subdural hematoma in the frontoparietal region was identified (Fig. 1). A diagnosis of chronic subdural hematoma presenting with transient ischemic attack was made considering the progression of symptoms and the absence of any coexistent lesion on tomography.

Hematoma evacuation by burr-hole craniostomy and irrigation drainage in the emergent setting was planned. However, no family consent was provided, therefore no surgical intervention could be made. The patient suffered from an ischemic attack manifested with motor aphasia and right hemiparesis on the 2nd day of follow-up period. The control magnetic resonance imaging (MR), in conjunction with the subacute subdural hematoma in the left frontoparietal region with the largest diameter being 1cm, an infarct lesion on the left parietal lobe with a significant mass effect due to localized edema was identified subjacent the hematoma (Fig 2).

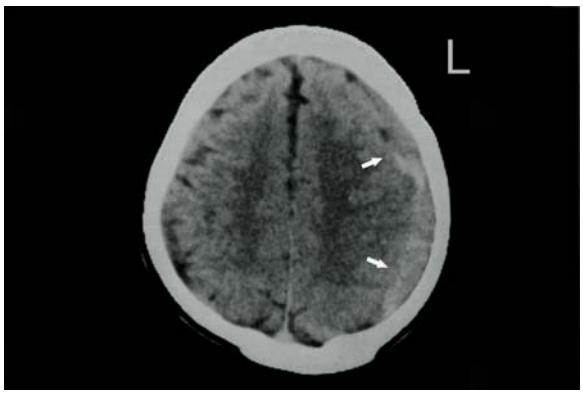

Figure 1. On computed tomography, a frontooccipital isodense subacute subdural hematoma (arrows) is delineated.

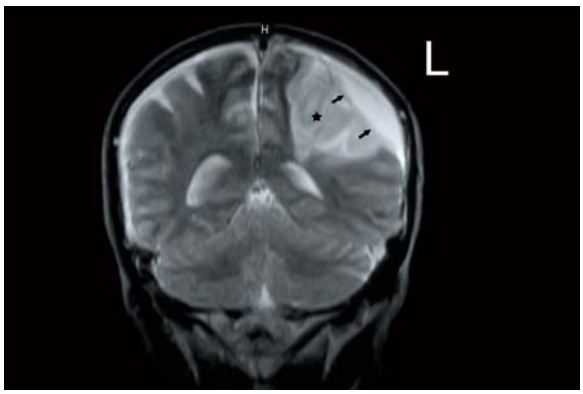

Figure 2. Localized edema and infarction area (star) are demonstrated subjacent the subacute subdural hematoma (arrows) at the level of its largest diameter.

Anti edema therapy by mannitol, and furosemide was implemented. On 24 hour follow-up examination after the beginning of the medical treatment, a partial recovery in the paresis, and a full recovery in the motor aphasia were noted. No complication developed during hospitalization and on 1st month follow-up, there was not increase thicking of subdural hemorrhage. Right hemiparesis was found to be persisting at that time.

Discussion

Coexistence of subacute subdural hematomas with TIA is seldom encountered and the real incidence is not known (8). Clinical progression of subacute subdural hematoma with the association of sudden developing transient ischemic neurological symptoms, display a different manifestation as to be a phenomenon. In this phenomenon, the most frequent symptoms are motor aphasia and paresis (4). Likewise, in our case, motor aphasia was associated with right hemiparesis.

The mechanism in the development of TIA in conjunction with chronic subdural hematomas is controversial (8). Welsh EJ reported two cases of chronic subdural hematomas presenting with transient neurological deficits in which cardiac or carotid artery bruits were identified (10). In another case of Kaminski et al, atrial fibrillation was found. Likewise, atrial fibrillation again was found in a similar study of Guptha and Puthrasingham (2). In the etiology of transient ischemic attacks, heart originated emboli are the most frequent etiological factor. Carotid athero-embolus is known as another etiological factor (4,9). Besides, development of chronic subdural hematomas has been reported following anti-aggregation therapy used in order to reduce the incidence of cerebral emboli in atrial fibrillation or carotid atheroma cases (1). Therefore, those transient ischemic attacks reported before are not due to chronic subdural hematomas, but rather due to emboli originating from heart or carotid artery, therefore it is not appropriate to mention under this phenomenon since it leads some pathophysiological controversies. Therefore, it is crucial to make differential diagnosis between these diseases causing transient ischemic attacks.

There are two basic factors in the pathophysiology of this phenomenon (2,6,8,10). 1) Mass effect of the chronic hemorrhage in the subdural space 2) Local cerebral edema caused by hematoma, without necessarily enlargement in the diameter. Furthermore, additionally mechanical stimulation to cortical mantle and even more, the changes in the head position could lead to the development of these symptoms intermittently (6,8). It is obvious that mass effect of the hematoma has a great role in the development of this phenomenon, since recovery in the attacks were achieved after the evacuation of the hematoma (4,7,10). Since no similar phenomenon is observed in most of the chronic subdural hematomas with significant mass effect, anatomical variations in the cerebral vascular structures adjacent to hematoma likely have the main effect in defining the physiopathology.

TIA is a focal neurological deficit lasting 15-20 minutes not extending beyond 24 hours. The neurologic deficit was completely resolved after the first attack in our case. However, discrete from the cases reported before, in our case, hemiparesis was partially permanent despite anti-edema therapy after the second attack. Ischemia progressing to infarction in the region adjacent to hematoma was delineated on 48 hour follow-up MR imaging. Neurological deficits in our cases were transient initially, which became permanent because of the delay in the hematoma evacuation, therefore, we recommend emergent surgical evacuation in these kind of chronic subdural hematomas associated with this phenomenon. The previous cases reported in the literature with a good recovery in TIA after evacuation of coexisting hematoma, all support our hypothesis.

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