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Cervical epidural hematoma mimicking a transient ischemic attack: A report of two cases

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Abstract

Spontaneous cervical epidural hematoma (SCEH) is a rare condition that causes paraparesis or quadriparesis. As spontaneous resolution is seldom expected, it usually requires surgical treatment for relieve symptoms. Even if spontaneous resolution occurs, relief from symptoms usually requires several hours to days. In contrast, hemiparesis is the most common symptom of a transient ischemic attack (TIA), and usually resolves over minutes to hours. We report here two patients with SCEH who presented with hemiparesis with severe neck pain. Both patients were taking antiplatelet drugs. Their neurological symptoms recovered spontaneously over a very short time. They were initially misdiagnosed as TIA. These cases show that patients with transient hemiparesis may have SCEH if there is severe neck pain and no cranial nerve involvement.

INTRODUCTION

Spontaneous cervical epidural hematoma (SCEH) is a rare cause of cord compression that usually requires surgical evacuation to relieve symptoms. Spontaneous resolution has seldom been reported. In these cases, transient neurological abnormalities usually resolve over a period of several hours to days. Here, we present two cases of cervical epidural hematoma mimicking transient ischemic attack (TIA) because the symptoms of TIA last for only a short time.

CASE REPORTS

Patient 1

A 78-year-old man with hypertension taking antihypertensive medication and aspirin developed sudden onset of posterior neck pain and weakness in his left limbs. The symptoms were relieved over time (30 min), and the weakness recovered upon arrival at our hospital. There were no obvious abnormalities on neurological examination. Brain magnetic resonance imaging (MRI) and angiography (MRA) showed no relevant abnormalities. An antiplatelet agent and hydration were administered with a diagnosis of TIA. On the second day, he complained again of severe neck pain without weakness. Cervical MRI performed that day revealed a posterior epidural hematoma on the left side of C2 – C6 and severe spinal canal narrowing (Figure. 1). As the patient did not develop neurological abnormalities, his condition was treated conservatively and pain was relieved.

Patient 2

A 67-year old woman taking cilostazol for right pontine infarction one year ago and with a 2-year history of hypertension came to our hospital for sudden severe posterior neck pain and right side weakness that developed 3 hours before the visit. The weakness was improving on arrival. Neurological examinations revealed no abnormalities. Brain MRI and MRA were also normal. An antiplatelet agent and hydration were administered with a diagnosis of TIA. Cervical MRI was performed because she complained of continuous severe posterior neck and shoulder pain after admission, and showed an epidural hematoma at the C3 – 4 level (Figure 2). We discontinued the antiplatelet drugs and treated the patient conservatively. Fourteen days later, a repeat cervical MRI showed no residual hematoma (Figure 3).

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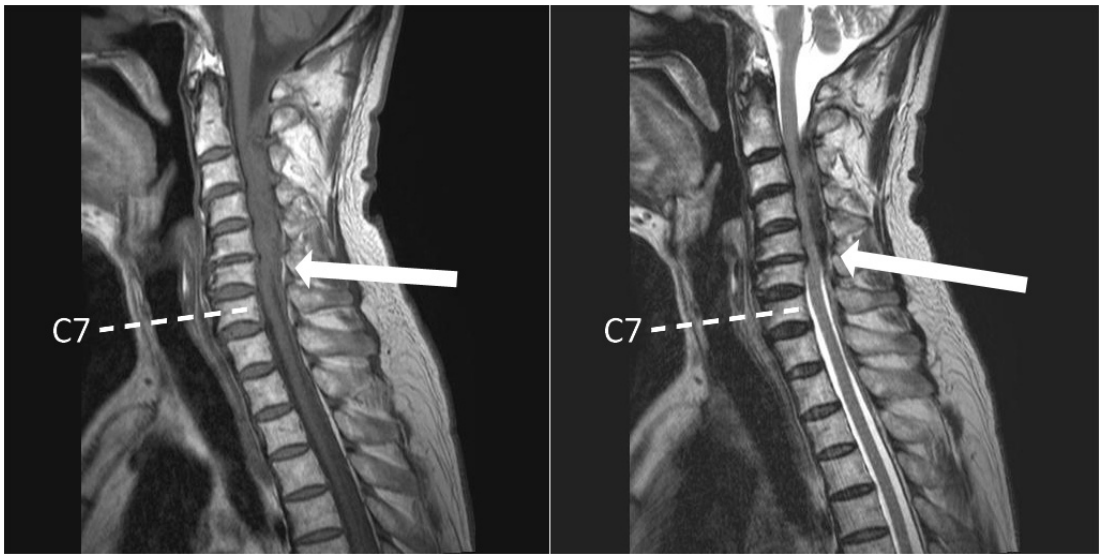

Figure 1: T1-weighted MRI of Patient 1 showing an isointensity space-occupying lesion (left, arrow), and T2-weighted MRI showing a low-intensity space-occupying lesion (right, arrow) at the C2 – C6 level.

DISCUSSION

SCEH is an infrequent condition with an estimated incidence of 0.1 per 100,000 patients per year.[1] Patients with SCEH usually present with sudden severe posterior neck or interscapular pain and neurological deficits localized to the level of the hematoma. Paraparesis and tetraparesis are common.[2,3] Hemiparesis, such as the clinical manifestations in our cases, is an extremely rare presentation and is sometimes misdiagnosed as cerebrovascular disease.[4-6] Factors that may contribute to the development of SCEH include an underlying hematological disorder (coagulopathy), ingestion of anticoagulants, straining, sneezing, lifting, and some spinal vascular anomalies.[7,8] Among these, the most common causative factor is ingestion of anticoagulants. It has been reported that 25% – 70% of patients with SCEH have a history of anticoagulant treatment.[7] In addition, there have been several reports of antiplatelet-related SCEH.[9-12] One of our patients was taking aspirin and the other was taking cilostazol. Both reported no recent trauma, and the laboratory

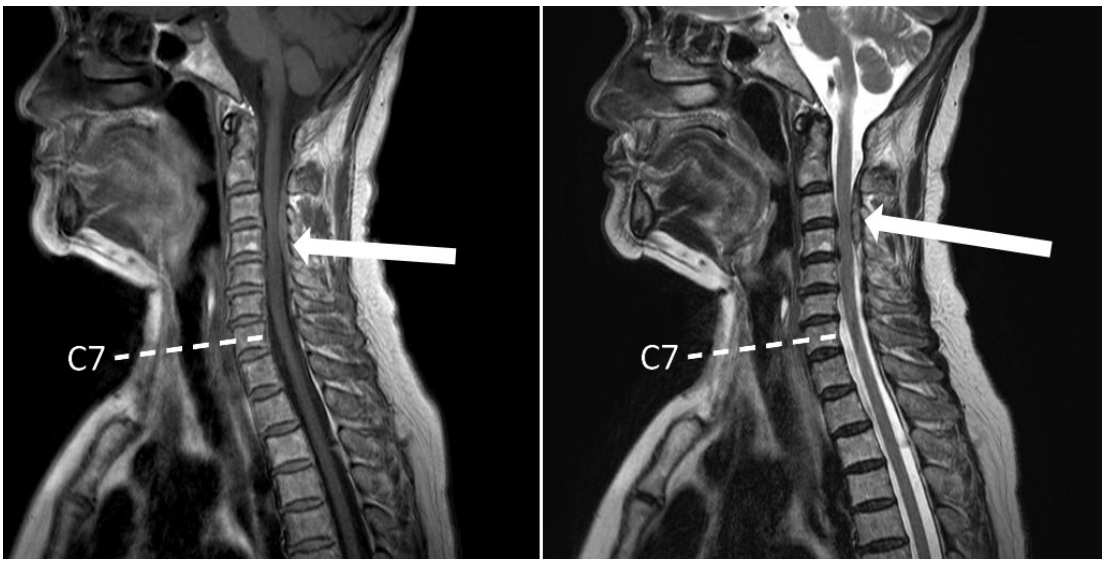

Figure 2: Initial T1-weighted MRI of Patient 2 showing an isointensity space-occupying lesion (left, arrow) and T2-weighted MRI showing a low- to isointensity space-occupying lesion (right, arrow) at the C3 – C4 level.

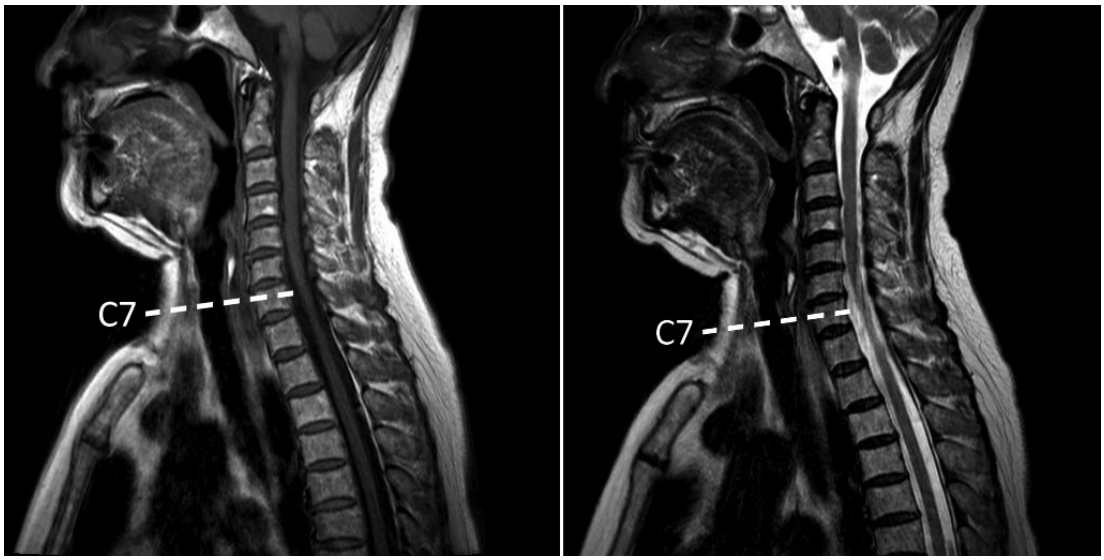

Figure 3: Follow-up cervical T1(left) and T2-weighted(right) MRI of the same patient (Patient 2) 14 days later revealed no signal changes or space-occupying lesion at the C3 – C4 level.

data did not reveal coagulopathy in either case. Therefore, the SCEH was thought to have been due to the ingestion of antiplatelet agents, although we could not completely exclude the possibility of a coincidence of SCEH with a medical history of antiplatelet agent use. However, considering the mechanism of action, the probability of a causal association seems high.

The clinical presentations in these two patients were unusual in that the hemiparesis was transient and continued for only a very short time. Transient hemiparesis in SCEH is very rare. The usual symptom duration in patients with SCEH, including classic symptoms such as paraparesis, is at least several hours to weeks.[13-16] The precise mechanisms underlying the spontaneous recovery in these two cases remain unknown. However, several hypotheses have been proposed to explain the spontaneous resolution of neurological signs and symptoms in cases of SCEH. The first is leakage of the hematoma through the intervertebral foramen, leading to spontaneous decompression of the spinal neural structures.[17] In addition, spreading of the hematoma within the spinal canal along the spinal epidural space has been proposed as a plausible explanation for spontaneous recovery.[18-20] We encountered two cases of spontaneously resolving epidural hematoma that presented with sudden unilateral limb weakness. The weakness in these patients improved more rapidly than in previously reported cases, and so they were initially misdiagnosed as TIA. The major difference between our cases and TIA was the severe posterior neck pain and absence of symptoms due to cranial nerve involvement. Severe posterior neck pain and an absence of cranial nerve abnormalities should be taken as indicators of cervical epidural hematoma in cases of suggested TIA.

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DISCLOSURE

Conflict of interest: none

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