

Cerebral Contusion as a Rare Cause of Foot Drop: Case Report

Nadir Bir Düşük Ayak Nedeni Olarak Serebral Kontüzyon: Olgu Sunumu

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ABSTRACT

A 74-year-old woman, taking anticoagulant therapy for chronic heart failure, presented to our emergency room with left dorsiflexion weakness 8 hours from after multitrauma. A detailed neurological examination revealed only 0/5 strength in the left foot dorsiflexion without any upper motor neuron signs. While there was no spinal cord pathology detected, cranial computed tomography demonstrated a lesion in the right parasagittal localization consistent with hemorrhagic contusion. Clinical follow-ups showed an improvement in neurological findings with muscle power of 3/5 in day 5 and 5/5 in day 45 of admission. The parasagittal region has a foot localization in the homonculus and lesions in this area can rarely present with the foot drop sign. Thus, parasagittal region lesions should always be kept in mind in foot drop cases.

KEYWORDS: Foot drop, Parasagittal region, Craniocerebral trauma

ÖZ

Yetmiş dört yaşında kronik kalp yetmezliği nedeniyle antikoagülan tedavi alan kadın hasta, multitravmadan 8 saat sonra sol ayağını kaldıramama şikayeti ile acil servise başvurdu. Hastanın yapılan nörolojik muayenesinde hiçbir üst motor nöron bulgusu olmadan sol ayak dorsifleksiyonunun 0/5 olması dışında özellik yoktu. Spinal incelemede spinal kord patolojisi saptanmazken, kraniyal bilgisayarlı tomografide sağ parasagittal lokalizasyonda hemorajik kontüzyon ile uyumlu lezyon tespit edildi. Klinik takibinde nörolojik iyileşme görülen hastanın 5. günde 3/5 olan kas gücü, 45. günde 5/5 idi. Hastanın kontrol kraniyal tomografisinde sağ parasagittaldeki kontüzyonun tamamen rezorbe olduğu görüldü. Parasagittal bölge, homonkulusun ayak lokalizasyonuna uyar ve bu bölge lezyonları nadiren düşük ayak bulgusu ile prezente olabilir. Bu nedenle, düşük ayaklı hastalarda parasagittal bölge lezyonları da akla gelmelidir.

ANAHTAR SÖZCÜKLER: Düşük ayak, Parasagittal bölge, Kranioserebral travma

INTRODUCTION

Foot drop is described as significant weakness of foot and ankle dorsiflexion due to motor function loss. Acute foot drop due to a central reason has been reported before (1,2, 4, 5, 7, 9, 11,12). Foot drop can occur as a result of peripheral and spinal neuron lesions or muscular atrophy. The most common cause is L4-5 radiculopathy or peroneal neuropathy caused by focal compression at the fibular head (2, 4, 6, 8). In a patient displaying foot drop, peripheral neuron lesions should always be considered primarily. However, parasagittal localized lesions like meningioma, astrocytoma, demyelinated plaque and hematoma can result in foot drop. A foot drop resulting from a central lesion is known as "spastic foot drop". Foot drop due to a brain lesion is very rarely presented in literature (7). We present a foot drop case of cerebral contusion origin.

CASE REPORT

A 74-year-old woman was referred to our hospital with multitrauma after a car accident. The patient who had been receiving anticoagulant therapy (for at least two years in the form of Coumadine 5 mg 1x1 tb/day had dorsiflexion

weakness in her left foot soon after the trauma and this was not accompanied by pain or sensory loss.

A detailed neurological examination revealed that all flexor, extensor, evertor and invertor muscle groups in the left foot were totally plegic (0/5) and no sensory loss was detected. Muscle tone was evaluated by the Modified Ashwort Scale from 0 to 5.

The patient's international normalized ratio (INR) level was 2.3 and activated partial thromboplastin time (APTT) was 36.2. Having been a multitrauma patient receiving anticoagulant therapy, she had gone through various diagnostic tests including spinal (cervical, toracal, lumbar) and cranial X-Ray, lumbar magnetic resonance imaging (MRI) and cranial computerized tomography (CT). Lumbar MRI revealed no pathology in her spinal cord or cauda equina. However, cranial CT showed a haemorrhagic contusion of 2x2 cm in the right parasagittal region (Figure 1). Nerve conduction studies revealed no abnormal response in the left lower extremity.

Anticoagulant therapy was stopped and clinical follow up revealed motor function improvement in her left foot

dorsiflexion from 0/5 to 3/5 on day 3 of admission. The patient's INR level regressed to 1.4. Antiaggregant treatment (Clexane 0.6 cc 2x1) was administered after consultation with a cardiologist. On the 6th day of admission, she was sent home as control cranial CT showed no progression and the patient's clinical condition continued to improve. The haematoma had regressed on the follow-up cranial CT that was performed 45 days after admission and the drop foot had totally improved to normal (Figure 2).

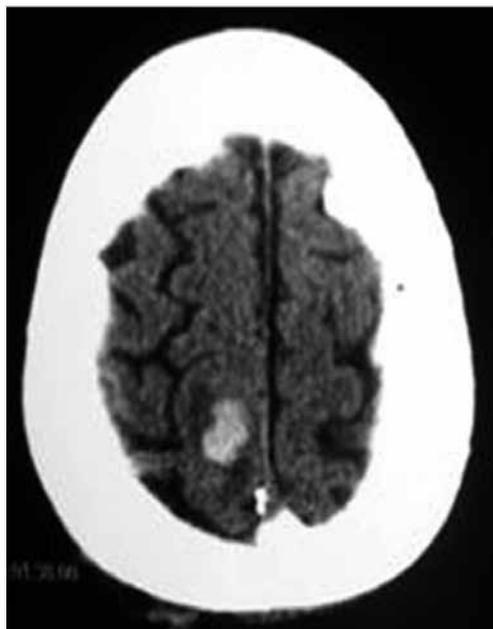


Figure 1: Cranial CT showed a 2x2 cm haemorrhagic contusion in right parasagittal precentral gyrus localization.

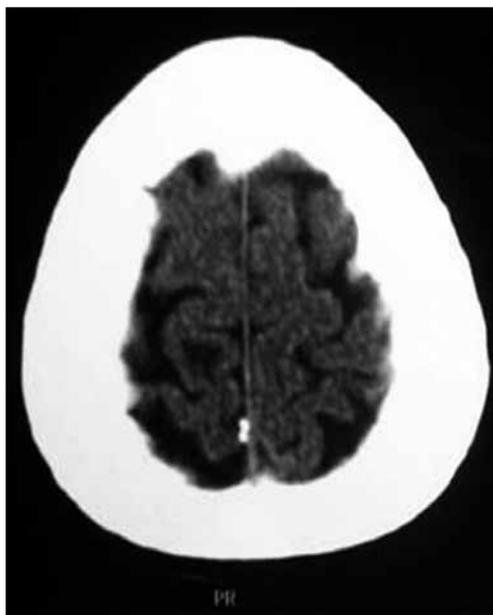


Figure 2: Follow-up cranial CT showed total resolution of the haemorrhagic contusion in the right parasagittal precentral gyrus localization.

DISCUSSION

The foot and ankle dorsiflexors include the tibialis anterior, extensor digitorum longus, and extensor hallucis longus; these muscles are innervated mostly by 4th and 5th lumbar, and partially by the 1st sacral nerve. They are controlled by the motor cortex. The primary motor cortex is in Brodmann area 4 and located in the precentral gyrus, lying as a thin layer from the dorsomedial surface of the hemisphere to the inferolateral surface of the precentral sulcus (9).

The most common cause of foot drop is L4-5 disc herniation and nerve root compression by a space-occupying mass or entrapment at the fibular head (2, 4, 6, 8). Foot drop with upper motor neuron (UMN) signs like Babinski's sign, hyperreflexia, and clonus may arise from a central origin. If the lumbar MRI reveals no pathology; the history, anamnesis, and physical examination of the patient will gain added significance. Electromyography (EMG) is the gold standard in the assessment of muscle innervations. In drop foot diagnosis, EMG and MRI findings were found to be correlated in 92% of the patients (37 out of 40 patients) (3). We performed no EMG study in our case as a cerebral lesion was found.

A good anamnesis and a detailed neurological examination are very helpful for the diagnosis of foot drop and to find out the etiology. We first performed spinal studies to reveal a more possible etiology for the foot drop like a spinal cord/cauda equina lesion as the patient had suffered a mild head trauma and had no UMN signs. However, no spinal pathology was found, and reviewing anticoagulant therapy anamnesis and head trauma together led to a cranial CT being performed and the drop foot etiology was cleared.

Several of the patients with cerebral pathologies had UMN signs like Babinski's sign and hyperreflexia as opposed to the peripheral originated pathologies (5, 7, 11). This centrally originated foot drop is referred to as "spastic foot drop" by Guthrie.

Cerebral causes should therefore be investigated if Babinski's sign, clonus or hyperreflexia are detected during the physical examination of a foot drop patient or if they are accompanied by headaches (7). Misdiagnosis of a foot drop after a head trauma is unlikely. However, in multitrauma patients, as in our case, the foot drop sign can mislead the examiner to peripheral causes.

Spinal tests including the lumbar MRI should be performed if UMN signs are not present while evaluating a patient with foot drop. If there exists no spinal pathology, the patient should be examined in terms of peroneal nerve compression at the fibular head and an EMG should be obtained. If these tests are negative, a cranial MRI should be carried out. A central pathology can be established in some patients without UMN signs, as in our case (1, 5, 10, 11). In one of 6 foot drop cases of central origin that Eskandary has published, foot drop was the result of cerebral contusion accompanied by a depressed fracture and UMN signs were present on physical examination (5).

In conclusion, negative spinal radiology and EMG, and lack of leg pain and sensory deficit in foot drop cases should direct the examiner to a cerebral etiology.

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