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CASE REPORT **OLGU SUNUMU**

AN UNUSUAL OCCURANCE MEDIAL MEDULLARY SYNDROME AND HEMIPLEGIA CRUCIATA; CASE REPORT

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ABSTRACT

Medial medullary syndrome (MMS) consists of ipsilateral lingual paresis, contralateral hemiparesis and lemniscal sensory loss. Hemiplegia cruciata (HC) consists of ipsilateral arm paresis and contralateral leg paresis. Both syndromes develop as a result of pathologies affecting V4 segment of the vertebral artery and anterior spinal artery. A 54-year-old man was admitted with left sided weakness. Neurological examination revealed left hemiparesis and right lingual paresis. Acute diffusion defect of anterior part of medulla oblongata was observed on Diffusion Weighted Imaging (DWI) and the patient was diagnosed with medial medullary syndrome. A month later, he developed another stroke causing a newly-emerging right arm paresis and progression of left leg paresis. DWI examination was repeated, which revealed an acute diffusion defect of craniocervical junction extending till third cervical vertebra. The patient was diagnosed with HC following MMS. We presented these two extremely rare syndromes which developed consecutively in a patient with stroke recurrence.
Keywords: Medial medullary syndrome, hemiplegia cruciata, vertebrobasilar stroke, recurren stroke.

SIRADIŞI BİR BİRLİKTELİK MEDİYAL MEDÜLLER SENDROM VE HEMİPLEJİ CRUCİATA; OLGU SUNUMU

ÖZET

Mediyal medüller sendrom (MMS) ipsilateral dil parezisi, kontralateral hemiparezi ve derin duyu kaybını içerir. Hemipleji cruciata (HC) ise ipsilateral kol parezisi ve kontralateral bacak parezisinden oluşur. Her iki sendrom vertebral arter ve anterior spinal arterin V4 segmentini etkileyen patolojilerin bir sonucu olarak gelişir. 54 yaşında erkek hasta sol yan güçsüzlüğü ile başvurdu. Nörolojik muayenesinde sol hemiparezi ve dil sağ yarımında parezi saptandı. Diffüzyon MRI incelemede medulla oblangatanın anterior bölümünde akut diffüzyon kısıtlılığı saptandı ve MMS olarak tanındı. Bir ay sonra hasta sağ kol parezisi ve sol bacak parezisinde artış sonucu yeni bir inme atağı ile başvurdu. Diffüzyon MRI incelemede kraniyo servikal bileşke ve üçüncü vertebra düzeyinde akut diffüzyon kıstlılığı saptandı. Olgu MMS sonrası inme rekürrensi ile HC olarak tanındı. Bu yazıda inme rekürrensi ile aynı hastada nadir iki sendrom sunulmuştur.
Anahtar Sözcükler: Mediyal medüller sendrom, hemipleji cruciata, vertebrobaziller inme, tekrarlayan inme.

INTRODUCTION

Vertebrobasilar strokes constitute about 20% of all strokes (1,2). They generally develop as a result of occlusion of the intracranial segment of the vertebral artery and cause various clinical pictures. Medial medullary syndrome (MMS) constitutes less than 1% of all ischemic strokes (2-4). MMS is characterized by ipsilateral hypoglossal paralysis, contralateral hemiplegia and loss of deep sensation. On the other hand, hemiplegia cruciata (HC), a much rarer form of vertebrobasilar stroke, causes ipsilateral arm and contralateral leg paresis. The most common cause of MMS is atherothrombosis, while HC mostly develops secondary to trauma (5-8).

We report a case initially presenting with MMS later develops HC.

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CASE

A 54-year-old, right-handed man was admitted with the complaints of acute dizziness, imbalance, nausea, vomiting and left-sided weakness. His past medical history was significant for diabetes mellitus and hypertension for five years, but he was not on any medication. Initial examination revealed a blood pressure of 150/80 mmHg. He was fully conscious. His speech was normal, but right lingual paresis was observed on tongue protrusion. He had mild hemiparesis, positive Babinski sign and hemihypoesthesia on the left side.

Vibration was impaired at the left lower extremity and his gait was paretic and mildly ataxic. Normal sinus rhythm was detected on electrocardiography. He was placed on acetylsalicylic acid 100 mg/day. Complete blood count, blood biochemistry and lipid profile results were within normal limits except for a high fasting glucose level (262 mg/dl) and a high HbA1c level (8.6%). Diffusion Weighted Imaging (DWI) revealed diffusion defects on the right paramedian side of the medulla oblongata and the right cerebellar parenchyma (Figure I).

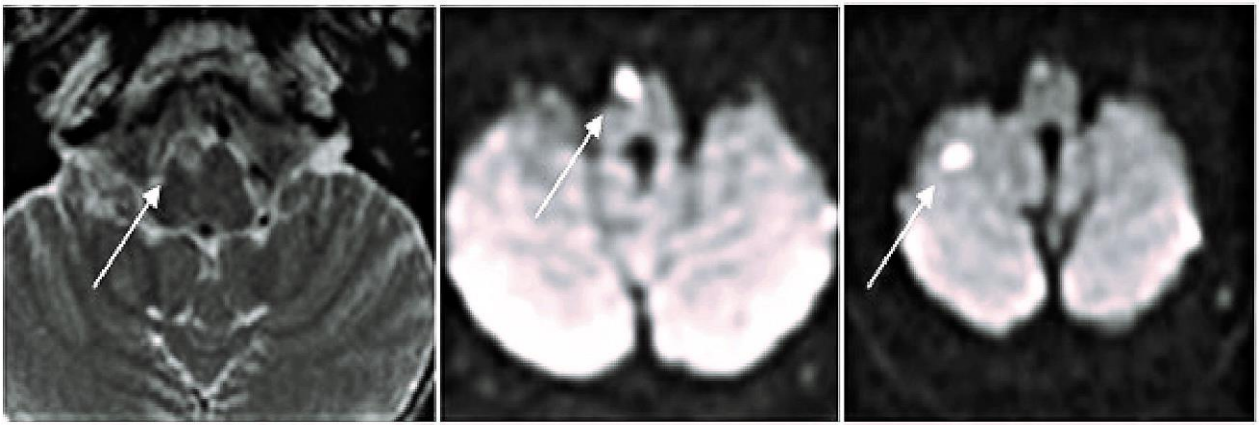

Figure I. Acute diffusion defect was detected at the anterior paramedian part of the right half of the medulla oblongata and right cerebellar parenchyma on Diffusion Weighted Imaging and T2 Flair examinations.

Echocardiographic examination revealed diastolic relaxation delay with an ejection fraction of 67%. Cerebral MR angiography demonstrated 85-90% stenosis at the proximal part of the basilar artery and 40-50% stenosis of the cavernous segment of the right internal carotid artery (ICA). Digital subtraction angiography (DSA) was performed, which revealed stenoses in the distal segments of the bilateral internal carotid arteries, V4 segments of the bilateral vertebral arteries and at the junction of the basilar artery, which were all more prominent on the right side. At the proximal basilar artery, more than 50% stenosis was also detected. (Figure II).

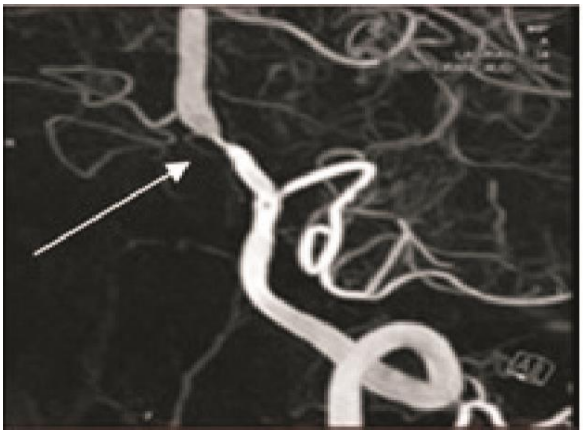

Figure II. Angiogram showing the right intracranial vertebral artery and basilar artery junction high degree stenosis.

Clopidogrel 75 mg/day was added to the treatment. Even though the patient had a right medial medullary infarct, endovascular stent was implanted into the V4 segment of the left vertebral artery due to the dissection occurrence at the DSA which was performed at the 7th day of the admission for diagnostic purpose. He was in stable condition with no progression and given Acetylsalicylic acid 100mg/day and Clopidogrel 75 mg/day at discharge. However, one month later, the patient presented with deteriorated left leg weakness and newly-emerging right arm weakness.

On neurological examination, he had right central facial paralysis, marked triparesis, left hemiparesis more prominent on the lower limb and right arm paresis, positive Babinski sign on the left. There was moderate dysmetria on the right and left-sided hemihypoesthesia. The patient was diagnosed with recurrent ischemic stroke. Present dual antiaggregant treatment was continued. He was placed on short-term anti-edema and glycoprotein IIb-IIIa antagonist (tirofiban) 0.01 mg/kg treatment.

Cerebral DWI examination revealed pathological areas at right inferior part of the medulla oblongata and at the craniocervical junction anterior to the medulla spinalis. Cervical MRI showed lesions at the craniocervical junction and the C2-C3 vertebra (Figure III).

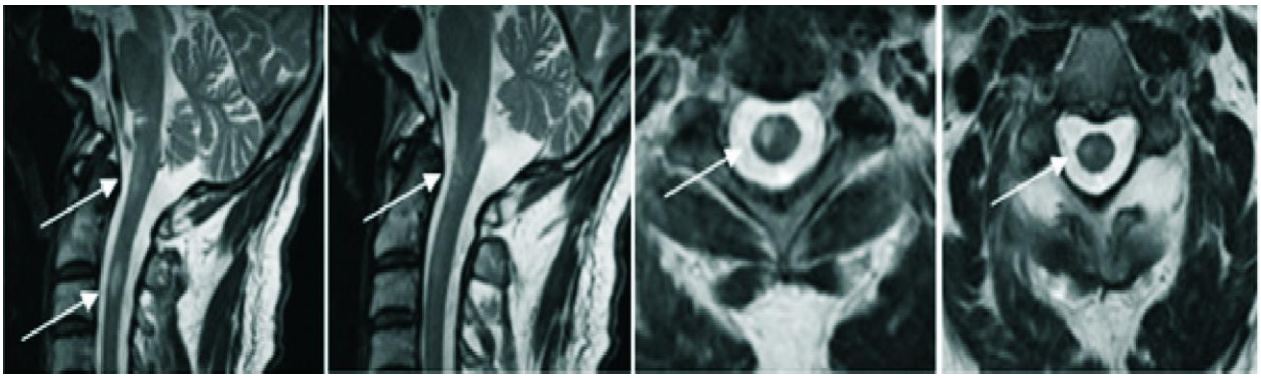

Figure III. Cerebral and cervical MR imaging studies showed ischemic changes at right inferior portion of the medulla oblongata, at the level of craniocervical junction and at C2-C3 vertebra.

DSA revealed that V4 segment of right vertebral artery was occluded, there were plaques in bilateral carotid bulbs in distal segments of ICA, the lumen of the cavernous segment was 40-50% stenotic on the right and 60-65% on the left side. Endovascular treatment and physical therapy-rehabilitation were recommended, and the patient was discharged in stable condition.

DISCUSSION

In this patient, the first cerebrovascular event was diagnosed as classical MMS. This patient had a stroke recurrence causing HC a month after his first stroke. The occurrence of these two infrequent syndromes in the same patient has not been reported before.

Medial medullary syndrome was first described at the beginning of twentieth century (9). It constitutes less than 1% of all ischemic strokes (2-4). Classical triad of MMS consists of ipsilateral lingual paresis, contralateral hemiparesis and deep sensation loss. In addition to these three classical findings, some other clinical signs and symptoms such as headache, vertigo, nausea, vomiting, facial paresis, nystagmus, dysarthria, dysphagia and diplopia may be detected (3,4,8). In the series of Kumral et al. (2002), seven of the eleven patients with MMS had classical triad, while Esen et al. (2008) detected classical triad in none of the twelve patients (3,4). In another study by Kim et al. (2009), 86 patients were included; among these, 91% had paresis, 73% had sensation disturbance and only 3% had tongue paresis (8). Our patient also had ataxia in addition to the classical symptoms of the disease. The most common cause of MMS is atherosclerosis, while hypertension, diabetes mellitus, atrial fibrillation, hyperlipidemia and smoking are risk factors in the development of anterior spinal artery and Vertebrobasilar

atherosclerosis (8,10,11). In our case, etiologies such as atherothrombotic, cardioembolic, hematologic and vascular anomalies were investigated and widespread atherosclerosis was detected in the anterior and posterior circulation. Since he had diabetes mellitus and hypertension as vascular risk factors, etiology of our case was most likely to be atherothrombosis. Radiological localizations of MMS lesions were described in detail in the series of Kim et al. (2009); 76% in upper medulla, 16% in middle part of the medulla, 20% in anterior medulla, 33% in mid-anterior medulla and 41% in anterior mid-posterior medulla (8). MRI examination of our case revealed an acute diffusion defect in the anterior part of the right side of the medulla. Twelve percent of the cases in the series of Kim et al. (2009) developed stroke recurrence during 71-month follow-up period, however HC was not reported in any of the patients with recurrence. Our patient had stroke recurrence leading to HC within a month after the first stroke.

Hemiplegia cruciata was described at the beginning of the twentieth century (10,11). Clinical findings include ipsilateral arm and contralateral leg paresis. HC is even rarer than MMC. Reported cases are generally series with few patients (5-7). Tractus corticospinalis decussates in decussatio pyramidum extending from the cervicomedullary junction to C2 vertebra. Fibers of upper extremity decussate in the rostral part of decussatio pyramidum and extend along the medial side within the tractus corticospinalis, while the fibers of lower extremity decussate caudally at the level of C1-C2 vertebra and extend along lateral side within the tractus corticospinalis. The anterior spinal artery emanates from near the vertebrobasilar junction,that is why infarcts occur in the cervical spinal cord. HC occurs as a result of the lesions of the craniocervical junction and the upper cervical region. In our case repeated cerebral and cervical MR imaging studies revealed lesions consistent with acute ischemia, which were located in the inferior part of the medulla oblongata and the anterior part of the medulla spinalis at the level of craniocervical junction. Cervical MRI revealed a spinal cord lesion consistent with acute ischemia at the level of C2-C3 vertebra and craniocervical junction. In contrast to MMS, etiologies of reported cases were mostly trauma, space occupying lesions and surgical complications. Cases related to multiple sclerosis were also reported (12).

In our case, endovascular stent was implanted into the V4 segment of the left vertebral artery due to the dissection occurrence at the DSA which was performed at the 7th day of the admission for diagnostic purpose. At the procedure and later, patient was hemodynamically stable at the clinical follow up. At the end on the first month, another stent was placed into the right vertebral artery, which was symptomatic. However, before placing the second stent, right vertebral artery occluded. In conclusion, we presented this case due to its rarity in that these two quite rare syndromes occurred consecutively in a patient with stroke recurrence.

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