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CASE REPORT

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Hemichorea due to Ischemic Stroke: Case Report

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ABSTRACT

Hemichorea is a recurrent, non-rhythmic, abnormal, and involuntary hyperkinetic movement disorder of one side of the body. The most common post-stroke hyperkinetic movement disorder is hemichorea. Similar cases have been presented in the literature before. Choreiform movements occur when the balance between direct and indirect pathways in the contralateral basal ganglia is disturbed. In hemichorea-hemiballismus; middle cerebral artery, posterior cerebral artery and least anterior cerebral artery are affected. Hemichorea is frequently reported after ischemic events affecting the lentiform nucleus or thalamus. We present a patient who presented with sudden onset of choreiform movements in the right upper and lower extremities and an acute infarct was found at the level of the left lentiform nucleus.

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1. INTRODUCTION

Hemichorea is a repetitive, non-rhythmic, abnormal and involuntary hyperkinetic movement disorder of one side of the body. The most common post-stroke hyperkinetic movement disorder is hemichorea [1,2]. Choreiform movements occur when the balance between direct and indirect pathways in the contralateral basal ganglia is disturbed. In hemichorea-hemiballismus; middle cerebral artery, posterior cerebral artery and least anterior cerebral artery are affected. Hemichorea is frequently reported after ischemic events affecting the lentiform nucleus or thalamus [1,3,4]. Vascular etiological factors seen in the formation of involuntary movements are ischemia, hemorrhage and vascular malformations such as arteriovenous malformation, venous angioma and cavernous angiomas. Hyperglycemia and hypoglycemia can also cause hemichorea, hemiballismus, generalized chorea and paroxysmal chorea by vascular mechanisms [3,5,6].

We present a patient who presented with sudden onset of choreiform movements in the right upper and lower extremities and an acute infarct was found at the level of the left lentiform nucleus.

2. OBSERVATION

A 58-year-old female patient was admitted to the neurology outpatient clinic because of involuntary movements in her right arm and leg that had started the day before. She had no known disease or medication. In her physical examination, her blood pressure was 120/70 mmHg, heart rate was 80/min, temperature was 36.5, and oxygen saturation was 97% in room air. Electrocardiography (ECG) was in normal sinus rhythm. In the neurological examination of the patient, non-rhythmic, repetitive involuntary movements in the right arm and leg suggested hemichorea, and muscle strength was normal. (Video 1.) Apart from that, other systemic examinations was normal. No infarct was detected in computerized brain tomography (CT). On

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magnetic resonance imaging (MRI) of the patient, a linear signal change was recorded at the level of the lentiform nucleus on the left, relative hyperintense in the diffusion sequence and relative hypointense in the ADC map, which may belong to acute/subacute infarction (Figure 1).

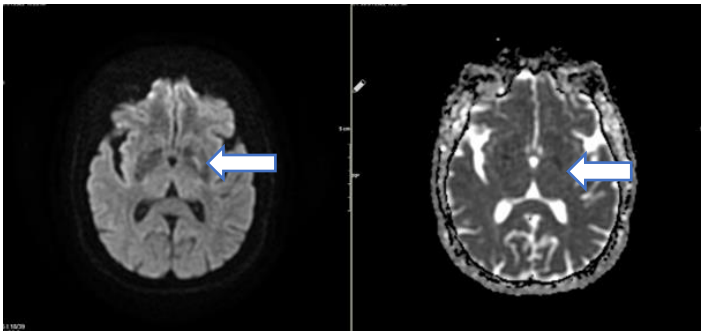

Figure 1. Acute-subacute diffusion restriction with linear ADC equivalent of left lentiform nucleus.

The patient was started on acetylsalicylic acid 300 mg one a day, clopidogrel 75 mg one a day, omeprazole once a day and 2 mg clonazepam daily and was hospitalized in the neurology service for further examination and treatment.

No significant pathology or cardioembolic stroke focus was detected in the cardiac examination. No vessel occlusion was found in the brain-neck CT angiography.

In laboratory tests, complete blood count, platelet function tests, coagulation tests, routine biochemistry tests, complete urinalysis, protein electrophoresis, antinuclear antibody, vitamin b12, thyroid function tests, HbA1c, erythrocyte sedimentation rate, autoantibody screening (anti-SSA, anti- SSB), antithyroid antibodies, syphilis serology (fluorescent treponemal antibody), Schirmer test, homocysteine, lupus anticoagulant, Protein C and S, anticardiolipin, Anti-streptolysin O (ASO), rheumatoid factor (RF), immunoglobin A,G,M was normal. Elisa tests (Hepatitis A, HIV, hepatitis B, hepatitis C) were negative. When viral meningitis agents (Herpes simplex virus 1-2 (HSV), varicella zoster virus (VZV), enterovirus, parechovirus, ebstein barr virus (EBV), cytomegalovirus (CMV), adenovirus) were investigated, the agent were not found. Brucella tests were negative. The pathergy test for Behçet was negative. No significant pathology was detected in the abdominal ultrasonography (USG) and chest X-ray.

It was observed that the symptoms of the patient were greatly reduced in the follow-up one week later.

3. DISCUSSION

Hemichorea and hemiballism are rarely seen in hyperkinetic movement disorders. It is well known that movement disorders may develop secondary to acute ischemic stroke. Hemichorea is a nonrhythmic, especially distal, abnormal hyperkinetic movement disorder. It is usually temporary but can last for weeks. [1,2,3] Acute hemichorea is seen in subthalamic nucleus and lentiform nucleus infarcts. Our patient had acute diffusion restriction at the level of the left lentiform nucleus on MRI.

In its pathogenesis, with the interruption of gamma amino butyric acid (GABA) transmission from the contralateral striatum to the globus pallidus externa, the inhibitory effect decreases, neuronal activity increases in the globus pallidus externa and inhibits the subthalamic nucleus. With inhibition of the subthalamic nucleus, control over the globus pallidus interna decreases, and as a result, the thalamus is not inhibited, and cerebral cortex stimulation increases and hemichorea occurs on the opposite side of the body. [2,3] In the case we presented, non-rhythmic movement disorder affecting especially the distal right upper and lower extremities suggested hemichorea.

While making the differential diagnosis of chorea, genetic diseases, Huntington's chorea, autoimmune diseases, neurodegenerative diseases, malignancy, infections, olivopontocerebellar atrophies, systemic diseases (systemic lupus erythematosus, chronic kidney failure, hepatocerebral degeneration), metabolic disorders (hypernatremia, hyponatremia, hyperthyroidism, hypothyroidism, hyperglycemia, hypothyroidism), hypoglycemia, hypomagnesemia, hypocalcemia, thiamine, niacin deficiency), drug exposure, stroke, intoxications should be considered. [2-4]

In order to control symptoms in stroke-related hemichorea, chorea-directed and stroke-oriented treatments should be given. The first choice in the treatment of chorea is neuroleptic drugs. We started our patient with 2 mg of clonazepam and her symptoms largely regressed. Other drugs that can be used in the treatment are haloperidol, chlorpromazine, tetrabenazine and perphenazine, sodium valporate and topiramate. [2,5,6]

As a result; In patients presenting with hyperkinetic movement disorders and having risk factors for stroke; it should be considered that cerebrovascular diseases may play a role in the etiology of hemichorea, causes that may cause hyperkinetic movement disorders should be excluded in the differential diagnosis, and both stroke and chorea treatment should be given.

Conflicts of interest: The authors declare that they have no known competing financial or personal relationships that could have appeared to influence to work reported in this paper.

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4. REFERENCES

1. Alarcón F, Zijlmans JC, Dueñas G, Cevallos N. Post-stroke movement disorders: report of 56 patients. J Neurol Neurosurg Psychiatry, 2004;75:1568-1574
2. Özdemir H. H, Demir C. F, Taşcı İ, Balduz M. Hemikore ile ortaya çıkan geçici iskemik atak olgusu. Türk Beyin Damar Hastalıkları Dergisi 2013; 19 (3): 108-111. doi: 10.5505/tbdhd.2013.68077
3. Sağlam S, Ekmekçi B, Tak AZA, Altun Y, Çağ İ, Gedik E. İskemik inme sonrası gelişen hemikore. Cukurova Med J 2016;41(Suppl 1):25-28. DOI: 10.17826/cutf.254590
4. Handley A, Medcalf P, Hellier K, Dutta D. Movement disorders after stroke. Age Ageing. 2009;38:260-6.
5. Chung SJ, Im JH, Lee MC, Kim JS. Hemichorea after stroke: clinical-radiological correlation. J Neurol. 2004;251:725-29.
6. Emir CB, Ağaoğlu J, Özyedek S, Seğmen H, Demiralp E, Yavlal FÖ, Tanık O. İnme sonrası gelişen hemikore. Parkinson Hast. Hareket Boz. Der., 2004;7(2): 95-100

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