CASE REPORT / OLGU SUNUMU

Transient Ischemic Attack After Scorpion Sting: A Case Report

Akrep Sokması Sonrası Gelişen Geçici İskemik Atak

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ABSTRACT

Aim: Stroke is a rare complication that occurs in addition to life-threatening respiratory and cardiovascular symptoms as a result of a scorpion sting. In this case, we presented a patient who had a transient ischemic attack that developed due to a scorpion sting, which had not been presented in the literature before.

Case: Our case was a 69-year-old male patient who felt pain in his left leg when he woke up in the morning. Weakness and speech disorder developed in the left arm and leg, developed approximately 1.5 hours after this pain. In the first examination of the patient, dysarthria, facial asymmetry, and loss of strength on the left side were detected. No significant pathology was observed in magnetic resonance imaging, brain computed tomography, and brain computed tomography angiography. In the physical examination, a dead scorpion has been found among the clothes, and mild redness and swelling at the level of the left ankle were observed. All neurological deficits in the patient resolved completely after 3 hours. Since no risk factor was detected in the examinations, it was thought that there was a transient ischemic attack due to scorpion venom.

Conclusion: While investigating the etiology of transient ischemic attack, careful physical examination besides neurological examination and anamnesis is very important for differential diagnosis of rare causes.

Keywords: Transient ischemic attack; scorpion sting; venom

ÖZ

Amaç: Akrep sokmasına bağlı gelişen akrep zehirlenmesi sonucu, yaşamı tehdit eden solunum ve kardiovasküler bulgular yanında inme nadir görülen bir komplikasyondur. Bu olgu sunumunda literatürde nadir sunulan akrep sokmasına sekonder, geçici iskemik atak geçiren bir hastayı sunduk.

Olgu: Olgumuz 69 yaşında erkek hasta sabah uyandığında sol bacağında bir ağrı hissetmiş. Bu ağrıdan yaklaşık 1.5 saat sonra gelişen sol kol ve bacakta güçsüzlük, konuşma bozukluğu gelişmiş. Hastanın yapılan ilk muayenesinde dizarti, fasial asimetri ve sol tarafında güç kaybı tespit edildi. Diffüzyon manyetik rezonans görüntüleme, beyin tomografisi ve beyin tomografik anjiosunda patoloji izlenmedi. Yapılan fizik muayenede hastanın kıyafetleri arasında ölü akrep ve sol ayak bileği düzeyinde hafif derecede kızarıklık ve şişlik izlendi. Hastadaki tüm nörolojik defisitler 3 saat sonra tamamen düzeldi. Yapılan tetkiklerde risk faktörü saptanmaması nedeni ile akrep zehrine bağlı geçici iskemik atak olduğu düşünüldü.

Sonuç: Geçici iskemik atak etyolojisi araştırılırken nörolojik muayene ve anamnezin yanında dikkatli fizik muayene yapılması nadir nedenlerin ayırıcı tanısı açısından çok önemlidir.

Anahtar Kelimeler: Geçici iskemik atak; akrep sokması; zehir

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Introduction

Ischemic cerebral infarction due to scorpion sting is very rare. The clinical presentation of scorpion sting poisoning can be elaborated with respiratory distress associated with arterial hypertension, cardiac arrhythmias, myocarditis, or pulmonary edema (1,2). These symptoms occur as a result of the release of catecholamines in the circulation or due to the direct cardiac toxicity of the venom. Intracranial hemorrhages occur in cases of acute increases in arterial blood pressure due to sympathetic overstimulation. Cerebral infarctions are associated with cerebral hypoperfusion, consumptive coagulopathy, vasculitis, or cardiogenic cerebral embolism (3).

In this study, we reported a patient who had a transient ischemic attack approximately 1.5 hours after the scorpion sting.

Case Presentation

A 69-year-old male patient with no known pre-morbid disease had severe pain in his left leg when he woke up in the morning. He refers to the emergency service with the complaints of speech disorder, loss of strength in his left arm and leg, and inability to walk, which developed approximately 1.5 hours after this pain. The patient's blood pressure and pulse rate were within normal limits. Coagulation parameters, D-Dimer, cardiac enzymes, and electrocardiography (ECG) were within normal limits. In the neurological examination, he was conscious and cooperative, his speech was dysarthric, and there was dimness in the left nasolabial groove. Motor loss of 1/5 and left hypoesthesia were detected in the left upper and lower extremities. During the systemic examination, a dead scorpion was found among his clothes and a slightly erythema and swollen area on the back of his left leg. The patient's brain computed tomography (CT), brain computed tomography angiography (CTA), and magnetic resonance imaging (MRI) was within normal limits. After approximately 3 hours, all neurological deficits improved completely. Etiologically lipid profile, carotid and vertebral arteries Doppler ultrasonography were within normal limits. Echocardiography and rhythm holter was performed and no cardiac pathology was detected. No change was observed in the patient's blood biochemistry follow-up. The patient's findings were thought to be transient ischemic attacks (TIA) due to scorpion venom. Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Discussion

Scorpion venom consists of enzymes that act on free amino acids, serotonin, hyaluronidase, and trypsinogen (4). These enzymes are toxins to the sodium and potassium channels, which lead to prolonged depolarization and thus to the stimulation of the autonomic nerves and the release of neurotransmitters. The venom alters the physiological functioning of voltage-dependent ion channels. Presynaptic opening of the sodium channel by inhibition of calcium-dependent potassium channels triggers the *"autonomous storm"*. Patients may present with bradycardia and excessive salivation secondary to parasympathetic stimulation. This may be followed by longer-term sympathetic stimulation, which may cause tachycardia, hypertension, and pulmonary edema (5,6,7). In addition, anoxia due to cardiovascular complications may cause central nervous system symptoms such as coma, seizures and hypoxic-ischemic encephalopathy in patients (8).

Cerebrovascular events in scorpion poisoning have been attributed to multiple mechanisms. Hemorrhagic stroke due to rupture of perforating arteries as a result of hypertension due to autonomic storm (9), cardioembolic stroke secondary to myocarditis (10), watershed infarct secondary to vasospasm due to catecholamines (11,12), and stroke secondary to increased platelet aggregation due to disseminated intravascular coagulation by poisoning (11,12) can be counted as these mechanisms.

The cerebrovascular damage effect of the venom usually occurs after 48 – 72 hours. In strokes occurring before 36 hours, it is primarily due to vasospasm secondary to catecholamines (12). In our case, other mechanisms were ruled out, and we thought that it was a transient ischemic attack secondary to vasospasm.

The cerebrovascular damage effect of the venom usually appears after 48-72 hours. In strokes occurring before 36 hours, it is primarily due to vasospasm secondary to catecholamines (12). In our case, the exact mechanism of the transient ischemic attack was uncertain, but due to the early onset of neurological findings and exclusion of other mechanisms, it was thought to be a transient ischemic attack secondary to vasospasm.

Conclusion

A case of transient ischemic attack due to scorpion sting has been reported very rarely in the literature before. While investigating the etiology of transient ischemic attack, careful physical examination as well as neurological examination and anamnesis are important in terms of differential diagnosis of rare causes.

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Authors' contributions : MA contributed to the drafting, clinical follow-up of the patient and literature review; MT evaluated the patient in terms of stroke, and differential diagnosis and contributed to the design of the draft; SK made the initial evaluation of the patient and contributed to the critical revision of the text.

Informed Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review in this journal.

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