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Case Report

Acute Ischemic Stroke Associated with Allergic Reaction

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Abstract

Stroke is a common condition particularly for the population over the age of 65 and it constitutes of two types including haemorrhagic and ischemic. It is known that allergic reaction may trigger coronary artery vasoconstriction and myocardial infarction. On the other hand, cerebrovascular vasoconstriction has been reported rarely owing to bee sting allergy. The aim of this rare case report to present a 55-year-old man admitted to emergency department with ischemic stroke resulted from allergic reaction.

Keywords: Stroke, Allergy; Ischemia; Haemorrhagic

Introduction

Stroke is a common condition particularly for the population over the age of 65 and it constitutes of two types including haemorrhagic and ischemic [1]. Accordingly, ischemic stroke occurs majority of the patients. The symptoms of the patients show varieties according to the location of the stroke [1]. Different symptoms might be seen such as hemiplegia, aphasia or facial paralysis [1,2]. Whereas atrial fibrillation and thromboembolic events are common causes of stroke in particularly elderly patients, impaired cerebral circulation may also lead to cerebrovascular diseases [1-3]. Moreover, thromboembolic events occurring after the instability of plaques formed in vessels such as carotid or vertebral arteries providing cerebral blood flow may also cause ischemic stroke. Additionally, aortic dissection may induce mortal ischemic stroke [1-3].

The collapse of cerebral circulation associated with allergic and anaphylactic reaction may cause vasodilatation, hypotension, bronchospasm, angioedema, confusion or death [4]. It is known that allergic reaction may trigger coronary artery vasoconstriction and myocardial infarction. On the other hand, cerebrovascular vasoconstriction has been reported rarely owing to bee sting [4,5]. The aim of this rare case report to present a 55-year-old man admitted to Emergency Department (ED) with ischemic stroke resulted from allergic reaction.

Case Presentation

A 55-year-old male patient presented to the ED with a complaint of speech disorder that developed after a bee sting at the workplace. There were no chronically diseases, medication or allergies in the patient's medical history. The current smoker patient did not have alcohol or substance abuse. The patient was conscious, cooperative and oriented, his vitals were; pulse: 90/min, blood pressure: 130/70 mmHg, oxygen saturation: 97%, fever: 36.9°C, respiratory rate: 14/min, blood sugar: 111 mg/dl. According to the anamnesis taken from his colleague, it was learned that the patient's speech disorder started approximately 30 minutes after the bee sting. There is no history of trauma or syncope. On physical examination of the patient, respiratory sounds were normal, no additional sounds, S1-S2 normal and rhythmic, no additional sounds or murmur, pulses were clear and bilaterally equal. In his neurological examination, there were no loss of muscle strength, neck stiffness, lateralization findings, nystagmus, dysmetria and dysarthria, pupillary light reflexes were bilaterally positive (PLR ++ / ++), Babinski reflexes were bilaterally negative, and deep tendon reflexes were normal. He had right homonymous hemianopsia and motor aphasia. His ECG was in normal sinus rhythm and any ST-T differences were detected. After blood tests were taken from the patient whose general condition was stable, imaging tests were requested. There were no pathological findings in his brain Computed Tomography (CT) scan (Figure 1).

Diffusion and brain Magnetic Resonance Imaging (MRI) was requested from the patient who was thought to have a stroke. On the gyral surfaces in the left frontal and parietal regions, and in the subcortical area, there were areas consistent with acute ischemia, which were hyper intense in diffusion A images and hypo intense in ADC mapping (Figures 2 and 3).

National Institutes of Health Stroke Scale (NIHSS) was calculated as 7. Alteplase was started at 0.9 mg/kg and patient's clinical finding did not change after thrombolytic. On neck CT angiography, both vertebral arteries and both common carotid arteries were observed normally along the trace, while non-calcified plaque formation was observed in the proximal part of the left internal carotid artery with approximately 50% stenosis.

In brain CT angiography, both internal carotid arteries, anterior and posterior communicating arteries, anterior, posterior and middle cerebral artery lumens were clear. Lumen diameters, contours and intraluminal densities were normal (Figure 4).

Discussion

Allergic reactions occur as a result of our defence mechanism against the pathogen that enters the body. It can cause many clinical presentations ranging from simple findings such as local oedema and hyperaemia to death. It may cause regional ischemia by making systemic vasoconstriction in the vascular system and severe dyspnea by causing bronchospasm and uvula oedema in the respiratory system. These situations are evaluated in favour of anaphylactic reaction [5-7].

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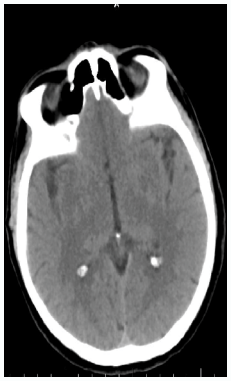

Figure 1: Brain CT of the patient.

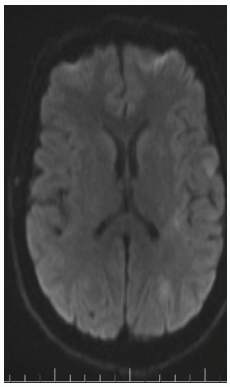

Figure 2: Diffusion MRI of the patient showing acute ischemia.

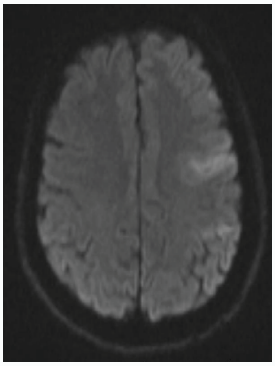

Figure 3: Diffusion MRI of the patient showing acute ischemia.

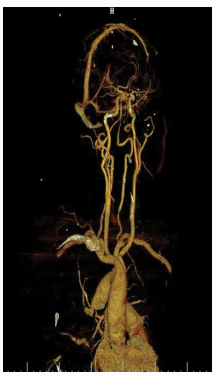

Figure 4: Brain CT angiography of the patient.

In our case, vitals were stable, and uvula oedema or bronchospasm were not detected. It was learnt that the clinical condition of the patient with motor aphasia started 30 minutes after contact with the pathogen. Brain CT and diffusion MRI were obtained. There was no intracranial bleeding, but diffusion restriction was found that would cause cerebral ischemia. Ischemic stroke due to an allergic reaction was considered in the patient whose clinical presentation did not improve after thrombolytic. The absence of pathological findings in brain CT angiography supports our diagnosis.

Ischemia due to an allergic reaction is a rare presentation. Myocardial infarction after contact with the pathogen in a patient without critical coronary artery stenosis is called Kounis Syndrome. However, a special name has not been given to the development of cerebral ischemia as a result of an allergic/anaphylactic reaction, and it is extremely rare [8].

Presentation of our case with sudden onset stroke clinic directed us to an intracranial pathology. Ischemic areas seen in diffusion MRI also confirmed our initial diagnosis. Therefore, thrombolytic therapy was given, but there was no clinical improvement in the patient. Normal visualization of vascular lumens in brain CT angiography led us to the diagnosis of ischemic stroke due to allergic reaction.

This case has shown that we may encounter many pathological conditions as a result of an allergic reaction. If a patient presents with any symptoms, it should be questioned when was the last time he/she asymptomatic and whether the patient has contact with a pathogen that may cause an allergic reaction before the symptoms.

Conclusion

Decent anamnesis should be taken from the patients who come with stroke symptoms after contact with an allergic pathogen considering that thrombolytic therapy will not improve the clinic and increase the risk of intracranial bleeding. Treatment for both allergic reaction and vasoconstriction should also be considered.

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