

A Case Report: Angioedema Developing in Half of the Tongue with Captopril

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Abstract

Angioedema (AE) is a life-threatening condition that can be seen in hereditary or non-hereditary form, usually manifests in subcutaneous tissue and progressed with edema in the face, lips, tongue, larynx and gastrointestinal system. Captopril is the first generated angiotensin-converting enzyme (ACE) inhibitor. Since the inhibition of ACE and consequently the angiotensin II level in plasma and tissues is reduced and quinine degradation is also inhibited by ACE, the level of bradykinin increases in plasma and tissues. It is thought to that the bradykinin causes edema due to vasodilation and increased vascular permeability. In this case report, we reviewed a 63-year-old patient, who hospitalized in the general surgery ward with the preliminary diagnosis of acute cholecystitis, developed AE after treated with 25 mg captopril for high blood pressure.

Keywords: Angioedema, ACE inhibitor, Tongue

Introduction

AE is such a disorder, which manifests itself with the feeling of pressure and oedema covering the subcutaneous tissue and sometimes leads to life-threatening airway obstruction. Many etiological factors play a role in the aetiology of AE, such as drugs (especially; penicillins, non-steroid anti-inflammatory drugs-NSAIDs), foods, respiratory allergens, and physical factors. Angioedema due to ACE inhibitors has been reported to be observed between 0.1 and 0.42%¹⁻². ACE inhibitors are a widely used group of drugs. Although angioedema is known to be one of the side effects, angioedema that develops in single half of the tongue is a very rare condition. Therefore, we present a case of AE in one half of the tongue due to ACE inhibitor use.

Case Presentation

A 63-year-old female patient was hospitalized in the general surgery department of our hospital with the diagnosis of acute cholecystitis. The patient was consulted due to swelling of the tongue, difficulty in breathing and speech. Her medical and surgical histories are unremarkable and there is no history of allergy. There is no history of unusual food intake or drug use. It was learned that the patient was con-

sulted by her doctor because of high blood pressure (184/92 mmHg) and 25 mg Captopril was given by the nurse.

Physical examination revealed significant edema (approximately 5*5 cm) in the right half of the tongue (Figure 1). Her vital signs were; systolic blood pressure: 138/78 mmHg, pulse: 86/min, fever: 36.7°C, pulse oxygen saturation: 96%, and respiration rate: 18 / min. The patient was anxious.

The patient underwent 120mg of methylprednisolone, 45.5 mg ofpheniramine, 50mg of ranitidine. During her follow-ups, I observed the airway of the patient with minimal increase in oedema. After an hour of first treatment, I gave her 8mg of dexamethasone and 50 mg of ranitidine again and the patient, who underwent cold treatment application, expressed herself relieved in breathing and speaking. The patient who responded to the treatment was observed to have decreased oedema at approximately 50% after 8 hours and her oedema was completely normalized on 24th hour.

Patient underwent close monitoring for airway and vitals. Medical treatment was started for captopril induced AE, and 120 mg methylprednisolone, 45.5 mg pheniramine and 50 mg ranitidine were administered intravenously. There was minimal increase in edema during the follow-up period. One hour after the initial treatment, 8 mg Dexamethasone and 50 mg Ranitidine were repeated intravenously. The patient described relief in breathing and speech during follow up. After 8 hours, edema decreased by 50% and edema completely returned to normal at 24 hours.



Figure 1. Unilateral angioedema in the half of the tongue

Discussion

The presence of various forms of AE and its occurrence in different tissue locations indicate the presence of underlying genetic mutations, allergic reactions and non-allergic reactions³. AE is classified as allergic (mast cell or IgE-mediated) or non-allergic (bradykinin-mediated)⁴.

Due to the inhibition of ACE, the level of bradykinin in plasma and tissues is elevated, since the angiotensin II level in plasma and tissues is reduced and quinine degradation is also inhibited by ACE. It is thought that bradykinin leads to edema due to vasodilatation and increased vascular permeability. Therefore, it is thought that the pathophysiology of ACE-induced angioedema does not occur immunologically because there is no detectable antibody against the ACE inhibitor⁵.

ACEI-induced AE may begin at the first use without dose-dependent, and as well as may develop after months or years during ACEI use. ACE-related AE is often seen in the head and neck region such as the face, oral mucosa, tongue, lip, pharynx and larynx³⁻⁶.

As in our case, cases of AE due to ACE inhibitor in the half of the tongue have been rarely observed⁷.

When the literature review is performed, it is seen that; it is not usually considered in the first encounter among physicians and is not often taken into consideration even in fatal situations. In severe cases, life-threatening airway obstruction, respiratory failure and asphyxia death have been reported⁸⁻⁹.

ACE inhibitor-induced angioedema is a life-threatening condition and no specific treatment has been described¹⁰. In the management of these patients, it is important to protect airway first, and in severe cases endotracheal intubation and tracheostomy may be needed. In our case, we did not need

advanced airway intervention because of the response to medical treatment. Although treatment applications consist of epinephrine, steroids, H1 and H2 receptor blockers, Icatibant (bradykinin 2 receptor antagonist) has been used in AE treatment in recent years.

Treatment applications consist of epinephrine, steroid, H1 and H2 receptor blockers, but in recent years Icatibant (2 receptor antagonists of bradykinin) has been used in the treatment of Hereditary Angioedema (HAÖ).

Conclusion

ACE inhibitors should be remembered as one of the most common causes of AE, which can be life-threatening.

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