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Case Report Open Access

Lingual Angioedema due to ACE-Inhibitor

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A 67 year-old woman awoke in the morning with the feeling that her tongue was swollen but she was able to eat breakfast without difficulty. She slept for one hour after lunch and then noted worsened swelling so that she could not speak or swallow. She had been taking lisinopril for hypertension for the last three years and did not report any new medications or exposures to recognized allergens. In the emergency department, she could not swallow her secretions or speak clearly but she had not yet developed stridor or respiratory distress. She received intravenous diphenhydramine and methylprednisolone as well as nebulized racemic epinephrine without improvement (Figure 1). In order to prevent asphyxiation if her tongue swelling progressed she was nasally intubated while awake with a 6.5 endotracheal tube using a flexible bronchoscope, and was admitted to the ICU. After receiving a transfusion of four units of fresh frozen plasma her symptoms improved over the next 6 hours and she was extubated the following morning. She was started on an alternate antihypertensive medication and counseled not to take any additional ACE -inhibitor medications in the future.

Although the specific details of the pathway of angiotensin converting enzyme inhibitors inducing angioedema are still being studied, it is accepted that bradykinin accumulation causes increased capillary permeability allowing plasma extravasation into the sub mucosal and subcutaneous tissue [1-3]. The incidence of ACE-I induced angioedema is higher in women, patients over 65 years, and in African-American patients [1,4,5]. It may occur shortly after initiation of the medication or not until after several years of usage [5,6]. Since it is not an allergic disorder, such as the angioedema caused by beta-lactamexposure or hymenoptera envenomation, there is poor response to antihistamines, steroids, or epinephrine [2]. While clinical investigations are still ongoing, there are several treatments which target the underlying problem of elevated plasma bradykinin: C1-inhibitor concentrate, recombinant C1-inhibitor (ecallantide), bradykinin receptor antagonist (icatibant), and Fresh Frozen Plasma, which will replace angiotensin ll to catalyze the degradation of the excess bradykinin [7-9].

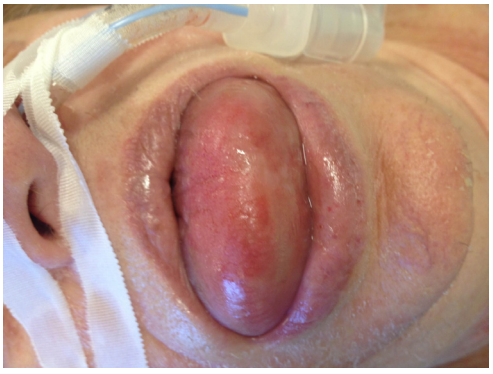

Figure 1: The incidence of ACE-I induced angioedema.

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