

## **Bradykinin mediated angioedema in patient using angiotensin-converting enzyme inhibitors (ACEI) presented with swelling of upper airways and body**

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We present a patient with ACEI induced angioedema after 5 years of therapy that caused not only swelling of mouth and upper airways but the whole body, which is an unusual clinical presentation. A 73-year old male patient was admitted to the emergency care unit with breathing difficulties due to the upper airway swelling that lasted for 2 hours. He had hypertension, diabetes mellitus, hypothyroidism, chronic renal disease and multiple myeloma. He had ACEI in his therapy for the last 5 years. The patient also had had swelling of the tongue 5 and 2 years ago. He was given corticosteroids and antihistamines which successfully resolved the edema, so allergic angioedema was diagnosed. This time he did not react to corticosteroids and antihistamines. Intubation was not possible due to tongue edema. Acute asphyxia occurred after 8.5 hours and emergency tracheotomy was performed. The swelling of upper airways and body continued to develop without the stabilization of clinical state. Because of suspected hereditary angioedema, a subcutaneous injection of icatibant was given as a lifesaving procedure. It caused the regression of the edema. ACEI were excluded from the therapy. After a week, blood analysis showed normal C1-inhibitor and C4 levels. Bradykinin mediated angioedema, including hereditary and ACEI induced forms, does not respond to conventional antihistamine and corticosteroid therapy. Also, they are not associated with urticaria. ACEI inhibit bradykinin degradation because angiotensin II is a key factor for the inactivation of bradykinin. Hereditary angioedema was suspected due to these severe symptoms and unusual clinical presentation. Type III hereditary angioedema with the normal level of C1-inhibitor is very rare but is considered as a differential diagnosis in our case.