Abstract

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Angioedema Related To Angiotensin-Converting Enzyme Inhibitors: A Case Series

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Introduction: Angioedema (AA) is a clinical condition characterized by blood vessel dilation and increased vascular permeability that can occur in every area of the body. Frequently it involves the face, upper airways or gastrointestinal tract, especially lips, tongues and larynx, so can be life-threatening. ACE-I-induced angioedema (ACE-I-AA) is more frequent in female sex, advanced age (> 65 years), African American ethnicity, allergic patients, during concomitant intake of other drugs (oral anti diabetic drugs, immunosuppressant's, anti-inflammatories, other antihypertensive like calcium channel blockers). The pathophysiology of ACE-I-AA involves inhibition of bradykinin and substance P degradation by ACE (kininase II) leading to vasodilation and plasma extravasation. There are no specific diagnostic biomarkers, so diagnosis is based on exclusion of other causes of angioedema in patients (pts) taking ACE-I. In this study we present the clinical features of a cohort of pts with ACE-I-AAE followed up at the Immunology and Allergy outpatient clinic of A.O.R.N. Cardarelli, Naples.

Diagnostic methods: All pts who presented at our centre over a 9-months period with history of recurrent angioedema without wheals after the initiation of ACE-I therapy, were enrolled in the study. The diagnosis of ACE-I-AA was based on their clinical, personal and familiar history, drug treatment and lab results.

We have excluded pts with hereditary and acquired C1 INH deficiency based on detection of blood levels of complement proteins; in fact, C1-INH and C4 are lower than normal in C1 inhibitor deficiency angioedema, while they are normal in ACE-I-AA. All the AA pts have been also studied for specific genetic mutations to exclude hereditary angioedema with normal C1 inhibitor.

Results: A total of 20 pts with a history of recurrent AA without urticaria were observed over a 9-months period. In 7 patients we have made a final diagnosis of ACE-I-AA. The median age of these pts was 77 years old (age range 50-86); 4 out of 7 cases were female. The onset of symptoms was usually reported 1 or 2 years before our evaluation, revealing a quite long diagnostic delay. The attack duration was about 12-24h. Nobody reported itching, flushing, or hives. The most used ACE-I was ramipril (28%) and enalapril (28%), followed by perindopril, lisinopril and zofenopril (14% each). Regarding comorbidities, 28% of pts were diabetics, 58% had hypercholesterolemia and 14% were smokers. No pts were using gliptins, but 3 of them were taking gliflozins. The most common involved anatomic sites were lips (58 %), tongue and face (21% each) and 1 patient had at least one episode of laryngeal oedema. After suspension of ACE-I 14% of patients have reported persistence of AA attacks. 5 out of 7 pts required hospitalization for AA almost once.

Conclusions: ACE-I-AAE is a rare side effect but it can be a medical emergency. Our data confirm that the majority of ACE-I-AAE is severe enough to require hospitalization. Despite the increasing awareness of this potential life-threating side effect within the medical doctors, the diagnostic delay is higher than 1 year. Discontinuation of ACE-I can stop the edema attacks but sometimes is not enough to break up symptoms.