Not all ACE inhibitor related angioedema is always evident: A case which is misdiagnosed as panic attack and speech disorder

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ABSTRACT
Angiotensin-converting enzyme (ACE) inhibitors are the most common medications responsible for angioedema. Angioedema is a potentially life threatening condition especially in geriatric age patients that they have take a several medications include ACE inhibitors and non steroidal anti inflammatory drugs. We present a case an ACE inhibitor induced angioedema that confused many clinical events.

Key words: ACE inhibitors. Angioedema.

INTRODUCTION
ACE inhibitors are widely used drugs for several indications. Angioedema is an important side effect of this group, which is usually seen in oro-facial region. If the angioedema is overt, drug-symptom relation can be easily established. However, in atypical cases misdiagnosis or delayed diagnosis is always possible.

In this report, we described a patient with ACE inhibitor related angioedema whose symptoms wrongly interpreted and undergone several unrelated investigations and treatments.

REPORT
A 68-years old female patient, who is under anti-psychotic treatment with the diagnosis of panic attack, was referred to allergy clinic because of recent onset widespread urticaria. She has also speech disorder (?) and verbal communication with her was impossible. For this reason the medical history was taken from her husband:

Medical history
She has diagnosis of arterial hypertension for 5 years, and hyperthyroidism for 7 months. In addition repetitive admittance to emergency medicine service with the complaints of breathless and sense of strangle during the last 4 months was noted her medical records. These complaints were misdiagnosed as anxiety disorder and panic attack, and given drug treatment. However, since the same complaints were continuing, she was referred to ENT (ear nose throat) clinic. Nasal septal deviation and nasal
polyp were determined by ENT specialist, and she operated. On post-operative day 2, she experienced again with difficulty swallowing and breathless. Moderate degree tongue edema was revealed during physical examination. Because of ongoing anti-thyroid treatment, enlarged tongue was associated with thyroid disorder and the patient was referred to endocrinology specialist. However, both thyroid hormone levels were normal and the tongue size reduced, the possibility of myxedema was ruled out. Finally, after all mentioned stages, the patient was referred to our allergy clinic with an irrelevant complaint, acute urticaria.

Management

On the first “allergic” examination, in addition to urticarial plaques, the tongue was nearly immobile because of severe edema on its root. As a routine procedure of this kind of cases, drug history was interviewed in detail. Within the drugs she used, an ACE inhibitor, cilazapril, is the most suspicious one. This medication was discontinued before all else, and replaced by an alternative antihypertensive drug (a calcium channel blocker). As a symptomatic treatment, low dose corticosteroid (deflazacort, 0.5 mg/kg per day) and a second generation anti-histamine (cetirizine 10 mg per day) were prescribed.

Within five days, the patient became completely symptom-free; she has no urticaria, she was speaking clearly, further, she was happy. Antihistamine treatment continued for additional 10 days to complete wash-out period of the ACE inhibitor, than ceased. The patient has been following-up for 18 months. Angioedema of the tongue, consequently complaints of breathless and sense of strangle did not recur.

DISCUSSION

The aim of this report is emphasize how important is detailed medical history for the diagnosis and how wrong is to be focusing only apparent but misleading symptoms. Physicians in all medical sciences should be aware of some common sign and symptoms. The important one of them is the drug reactions. From this point of view, in angioedema cases affecting head and neck regions, ACE inhibitors (and AT-II antagonists) must be initially investigated etiology.

REFERENCES