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ACE-inhibitors and angioedema

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To the editor:

Karim and Masood¹ recently reported a life-threatening angiotensin-converting enzyme (ACE) inhibitor–associated episode of angioedema, successfully treated with fresh-frozen plasma. They report that the estimated prevalence of ACE-inhibitor angioedema is 0.1% to 0.2%. Our prevalence rate is different and in line with that of Agostoni et al.²

We evaluated (a) the prevalence of angioedema in a cohort of 159 post–myocardial infarction patients (134 male, 25 female) treated with ACE-inhibitors for at least 6 months and (b) the prevalence of ACE-related angioedema in 276 patients with angioedema (85 male, 191 female) attending our outpatient allergy clinic (with or without urticaria).

In the first group of 159 patients, all of whom were under evaluation for metal contact allergy related to stent stenosis, angioedema was reported in 3 white male patients on Ramipril (2.2%). Conversely, in the 276 cases of angioedema, ACE-inhibitors were implicated in 7.9% cases (11.7% of the males, 6.3% of the females), Enalapril being the most frequently involved. The indication for treatment in the latter group was uncomplicated hypertension in all. Never in any of these cases was the angioedema life-threatening, and, in accord with

what was noted by Karim and Masood,¹ it affected head and neck in most cases. All of the patients were white.

As far as treatment is concerned, most patients had received parenteral antihistamines and steroids during the acute attack in the emergency department and were put on oral antihistamines for several days afterward. In each case, neither the emergency department physician nor the family doctor had discontinued the treatment or informed the patient about the possibility of a causal relationship. Apparently, information about this important side effect of ACE-inhibitor treatment has not yet reached all categories of physicians. This might be explained, at least in part, by the bizarre pattern of presentation of ACE-inhibitor–related angioedema: It can appear at various times after the onset of treatment as well as disappear spontaneously despite the continuation of treatment.³ The lack of information about a causal relationship between ACE-inhibitors and angioedema, in turn, might account for the variability in the prevalence rates reported thus far.

When we observed the patients, our therapeutic option was not to withdraw the drug in the post–myocardial infarction patients in consideration of the beneficial effect of ACE-inhibitors on their heart condition and the benignity of the associated angioedema; in each case, however, we informed both the family doctor and the patient about the possibility of more severe reactions.

In the other group, we suggested to every patient that he or she switch to an antihypertensive drug of different class. So far, no recurrence of angioedema has been reported.

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Reply

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