Clinical picture

ACE inhibitor related angioedema

A 51-year-old woman presented with sudden onset of lip and facial swelling. She denied any trauma, insect bites, dyspnea or urticaria. She had been on lisinopril since last 3 years. Clinical findings on presentation were suggestive of angioedema (Figure 1). She did not have stridor or respiratory compromise. There was no personal or family history of angioedema. The patient was treated with steroids and antihistamines with marked improvement in next 24h. Lisinopril was discontinued and she was found to be doing well on follow up.

Angiotensin converting enzyme inhibitors (ACEI) are one of the most commonly prescribed classes of medications. Angioedema is characterized by self-limited, localized, nonpitting edema involving the deeper layers of skin including subcutaneous tissue. ACEI-related angioedema commonly involves lips, tongue and face. ACEI-related angioedema is primarily mediated through elevation in bradykinin levels, which is a potent mediator of vasodilation and increased vascular permeability. This is a class effect and can happen with all ACEI. Pruritis and urticaria, symptoms of mast cell-mediated angioedema are usually not seen in ACEI-mediated reaction, but intestinal wall edema can occur. Although the risk is highest within the first week of therapy, it can occur even after several years of treatment with an ACE inhibitor. Angiotensin receptor blockers (ARB) can cause angioedema in minority of patients who had reaction to ACEI. The cornerstone of management is to stop the offending agent.

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Conflict of interest: None declared.

References