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Unmasking Bradykinin: A Case of ACE Inhibitor-Induced Angioedema Responsive to Tranexamic Acid

Musyirah Amaran[1], Johnny Ngu Tai Hock[1], Mexmollen Marcus[2]
[1] Hospital Queen Elizabeth II, 88300 Kota Kinabalu, Sabah, Malaysia.
[2] Department of Emergency Medicine, Faculty of Medicine and Health Sciences, Universiti Malaysia Sabah, 88400 Kota Kinabalu, Sabah, Malaysia.

INTRODUCTION: Angioedema is a potentially life-threatening upper airway obstruction caused by plasma leakage into skin and submucosal tissues. While histamine-mediated forms often respond to standard anaphylaxis treatment, bradykinin-mediated angioedema—such as that caused by ACE inhibitors—resists conventional therapy and poses a greater management challenge.

KEYWORDS : Angioedema, Bradykinin, ACE-inhibitors, Tranxenamic acid

CASE DESCRIPTION:

We report the case of a 77-year-old woman with a known seafood allergy who presented with progressive facial swelling, throat discomfort, and dyspnoea over a 3-day period. She had recently been discharged for acute coronary syndrome and had been prescribed oral perindopril 8 mg among other medications. She denied exposure to any known allergens. Clinically, there were no signs of airway compromise, and her vital signs remained stable. Despite administration of standard anaphylaxis treatment, no clinical improvement was observed. Flexible nasoendoscopy performed by the ORL team revealed edema of the retropharyngeal space, bilateral arytenoids, epiglottis and vocal cords.

Given the clinical presentation and recent initiation of perindopril, ACEi-induced angioedema was suspected. A trial of intravenous tranexamic acid (1 gram) was administered, resulting in marked improvement in the angioedema.

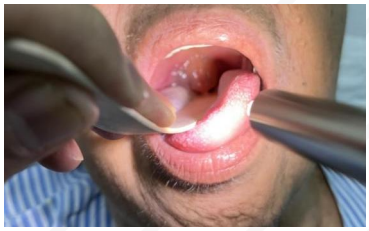

Image 1 : Edematous soft palate and uvula

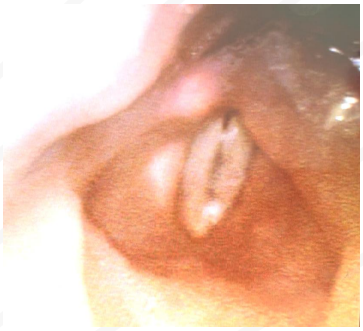

Image 2 : Edematous arytenoids and vocal cords

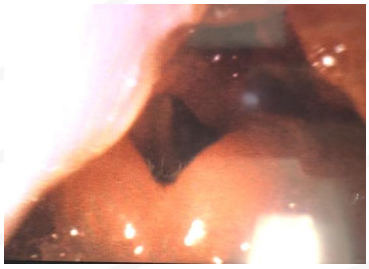

Image 3 : Edematous epiglottis

DISCUSSION:

Early recognition of ACEi-induced angioedema hinges on thorough medication history and a high index of suspicion. Bradykinin-mediated angioedema does not respond to traditional anaphylaxis therapies. Tranexamic acid, through inhibition of plasminogen activation, reduces bradykinin production which is a potent vasodilator and may be an effective early intervention[1]. Other therapeutic options reported in the literature include fresh frozen plasma, C1-esterase inhibitor concentrates, icatibant, and ecallantide, with variable efficacy[2].

CONCLUSION:

ACE inhibitor-induced angioedema may present insidiously but possess a threat to airway compromise thus securing the airway remains a priority. While standard anaphylaxis treatments are appropriate initial measures, persistent symptoms should prompt consideration of bradykinin-mediated angioedema. In such cases, agents like tranexamic acid, fresh frozen plasma, or C1-esterase inhibitors may be life-saving.

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