ACE Inhibitor Induced Angioedema

REFRESHER

RECOGNIZE IT

- High index of suspicion for any angioedema patient taking an ACE inhibitor
- Caused by excessive accumulation of bradykinin
- Usually starts soon after starting an ACE inhibitor, but may occur years later
- Higher incidence among women; five-fold higher incidence among African Americans
- Versus histamine angioedema: no allergic trigger (may follow minor trauma)
- Starts with focal, often asymmetric swelling of tongue or lips; evolves over hours
- No urticaria or itching; fails to respond to antihistamines, steroids, epinephrine

ASSESS

- Time-sensitive: assess airway, oxygenation, and ventilation
- Avoid tunnel vision
- Other diagnoses to consider: burns, epiglottitis, croup, trauma

ACT — FIRST MOVES

1. Do not rely on steroids, antihistamines, or epinephrine — ineffective for ACEI-RA
2. Epinephrine is given mainly for alpha-1 vasoconstriction to reduce swelling; results unimpressive in case studies
3. H1 and H2 blockers are reasonable when the angioedema's cause is histaminic or unknown
4. Protect the airway — swelling is focal, progressive over hours, and bradykinin-driven

PEARLS

- Patients recovered nearly three times faster with a bradykinin blocker than standard glucocorticoid/antihistamine treatment (NEJM study)
- Slow evolution over hours separates it from faster histamine-mediated angioedema

Take Home Message:

Angioedema on an ACE inhibitor is bradykinin-driven until proven otherwise — standard allergy drugs fail, so protect the airway early.

[Guideline/Source: Small study published in the New England Journal of…]