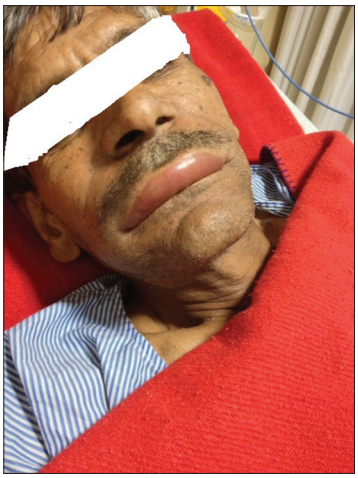

Figure 1: Upper lip angioedema

A rare case of angioedema after anaesthesia

Sir,

Angioedema is the acute onset swelling of skin or mucous membrane. Upper lip angioedema may herald life-threatening upper airway oedema.

A 57-year-old male weighing 50 kg was scheduled for direct laryngoscopy and biopsy of a laryngeal growth. He was a chronic smoker but non-alcoholic. He was a known hypertensive on tablet ramipril 5 mg OD since 1 year. In the operating room, he was premedicated with glycopyrrolate 0.1 mg, midazolam 1 mg and fentanyl 100 μg intravenously. Anaesthesia was induced with propofol 100 mg and vecuronium 5 mg was administered for neuromuscular paralysis. The trachea was intubated and anaesthesia was maintained with 2% sevoflurane in a 50:50 mixture of O_2 and N_2O. Diclofenac 75 mg was given intravenously for post-operative analgesia. At the end of the surgery, neuromuscular blockade was reversed with neostigmine 2.5 mg and glycopyrrolate 0.4 mg, the trachea extubated and the patient shifted to the post-anaesthesia care unit. After 2 h, the patient developed swelling of the upper lip [Figure 1]. The skin over it was stretched, shiny and warm. There was no difficulty in swallowing or respiration. The tongue, lower lip, eyelids and oropharynx were normal. There was no associated urticaria or rash. On auscultation, there were no added breath sounds. He had a pulse rate of 70/min, blood pressure 121/87 mm Hg, respiratory rate 14/min and SpO_2 100%. He had no similar episode in the past and there was no family history of such oedema. There was no evidence of insect bite and he had not been allowed oral intake. A diagnosis of drug induced angioedema was made. The patient was given hydrocortisone 100 mg and pheniramine 45 mg intravenously. Adrenaline was kept ready. Ice packs were used to soothe the upper lip. He was continuously monitored for an increase in oedema or stridor. The swelling began to decrease in 2 h and completely subsided by 48 h. The patient had a history of previous uneventful ingestion of diclofenac. Hence, angiotensin converting enzyme inhibitor (ACEI) induced angioedema was diagnosed and ramipril was discontinued. He was started on tablet amlodipine for hypertension. He was educated about angioedema and its alerts and advised to abstain from alcohol and smoking. Recurrence of angioedema would have prompted sequential discontinuation of non-steroidal anti-inflammatory agents (NSAIDs), opioids and amlodipine with further investigation for hereditary and acquired angioedema.

Angioedema may be idiopathic, extrinsic factor induced or due to C1 esterase inhibitor deficiency. It may be allergic (histamine mediated) or non-allergic (kinin mediated). Drugs implicated include NSAID's, ACEI and angiotensin receptor blockers.[1]

The pathogenesis of ACEI induced angioedema is decreased degradation of bradykinin, causing vasodilation and increased vascular permeability, especially in the lax tissues of the face.[2] Tissue bradykinin is increased in all patients but angioedema occurs only in about 0.1-0.7%.[1] ACEI have an

unpredictable temporal relationship with angioedema. It usually occurs in the 1st week of therapy but may occur after several years of regular intake or more than 24 h after the last ingestion.[2] Airway intervention is required in 11% of patients. The most common presenting signs are dyspnoea, lip and tongue swelling.[3] Oedema of the floor of the mouth and tongue is a harbinger of airway involvement requiring intensive care unit admission, monitoring and early fibre-optic guided intubation to prevent emergent surgical airway access.[4] The risk factors include black race, female gender, smoking, alcohol use and history of atopy. Several trigger factors like concomitant NSAID use, surgical gloves (latex allergy), local trauma (oropharyngeal airway, pressure from laryngoscope), upper airway instrumentation, local anaesthetic use, irritant fumes, stress and immunosuppression have been proposed.[1,2,5,6]

NSAID induced angioedema is due to increased leukotriene synthesis.[1] It may be caused by ibuprofen (57%), aspirin (19%), diclofenac (9.5%), or others and mostly involves the periorbital area.[7]

Propofol increases bradykinin levels, causing pain on injection but this is not greater than that observed after ACEI therapy. Secondly, the half-life of bradykinin is only 15 s and it is rapidly metabolised.[8] Therefore, it is difficult to establish a relationship between propofol and ACEI induced angioedema.

ACEI induced angioedema is a diagnosis of exclusion and there is no specific investigation. Patients with recurrent angioedema need workup with complete blood count, erythrocyte sedimentation rate and complement levels.[9]

The treatment is supportive. Corticosteroids and antihistamines have no proven therapeutic effect. Adrenaline is useful only in allergic angioedema, insect bite and anaphylaxis. The most important step after the initial management is recognition of ACEI as the cause and its immediate discontinuation as continued use can cause serious recurrent angioedema.[1,2]

A post-operative stridor after airway related procedures or head and neck surgery may be due to pre-existing infection, tumour haemorrhage, epiglottitis, anaphylaxis, or angioedema leading to a confusion in diagnosis. It is important for anaesthesiologists, as airway experts, to be aware of this condition as their expertise may be required.

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