Severe angioedema of laryngeal inlet in a woman receiving telmisartan therapy undergoing breast conservation surgery

Sir,

A 55-year-old female with carcinoma of the left breast was scheduled for breast conservation surgery with axillary node dissection. She was taking oral telmisartan 40 mg and thyroxine 25 μ g, once daily, for the treatment of hypertension and hypothyroidism, respectively. At

the time of pre-anaesthetic visit, her blood pressure was controlled, and she was clinically and biochemically euthyroid. Both oral medications were continued on the day of surgery; her telmisartan dose was however halved to 20 mg, as per the consultant's advice.

Following induction of anaesthesia, the lungs were ventilated through a size 3 laryngeal mask airway (LMA® ProSeal™). Anaesthesia was maintained with isoflurane in air-oxygen mixture and ventilation was adjusted to maintain an end-tidal $\rm CO_2$ of 35–40 mmHg. Before the commencement of surgery, it was noticed that the peak airway pressures were high (>40 cms $\rm H_2O$) and adequate tidal ventilation was not achieved (set tidal volume 450 ml; expired tidal

volume 100–130 ml). Bilateral air-entry was found to be decreased on auscultation with considerable adventitious sounds.

Suspecting malposition of the LMA, reposition was attempted but was unsuccessful. The LMA was removed, and direct laryngoscopy was performed which showed a grossly oedematous and immobile tip of epiglottis. The vocal cords could not be visualised. With intermittent mask ventilation to maintain oxygenation, flexible fibre-optic bronchoscopy was used to urgently secure the airway. On flexible fibre-optic bronchoscopy, the epiglottis and vocal cords were seen to be ballooned out and distorted [Figure 1a]. The patient was intubated with a 7 mm ID cuffed endotracheal tube (ETT) in the first attempt. However, the cuff was not deployed since there was no air leak as the ETT was completely encircled by the oedematous cords [Figure 1b and Video Loop 1].

Injection dexamethasone 8 mg stat IV was administered and repeated 12th hourly and the decision was taken to proceed with surgery since there was no haemodynamic compromise and the airway had also been secured. Her intraoperative course was uneventful, and she was shifted to the Intensive Care Unit (ICU) for elective ventilation. The trachea was extubated the next day after fibre-optic examination showed resolution of the epiglottic and vocal cord oedema [Figure 1c]. Before discharge from ICU, a repeat fibre-optic examination showed normal appearance of the epiglottis and the vocal cords [Figure 1d]. There was no incidence of stridor or any respiratory compromise following extubation.

This case adds to the existing clinical conundrum on continuation versus discontinuation of angiotensin converting enzyme inhibitor (ACEI)/angiotensin receptor blocker (ARB) treatment preoperatively. Angioedema is known with ACEI/ARB medication. [1] However, evidence on whether to continue or discontinue these drugs on the day of surgery is not yet convincing. [2-4]

The mechanism of angioedema due to ACEIs involves inhibition of degradation of bradykinin which is responsible for the classic non-pitting, non-pruritic, painless oedema commonly involving the tongue, oropharynx, periorbital and perioral areas.^[1] In severe cases (type 3), the epiglottis and vocal cords are involved, as happened in this case. In the perioperative period, the trigger for developing

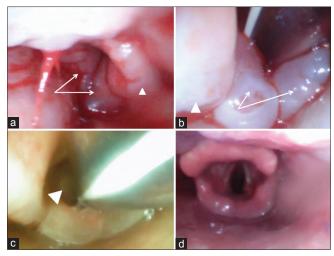


Figure 1: (a) Fibre-optic view of the vocal cord shows severe oedema of the epiglottis (arrow head) and the vocal cords (arrows). (b) Fibre-optic view after intubation shows the ballooned out epiglottis (arrow head) and vocal cords (arrows). There is no space between the endo-tracheal tube and the vocal cords. (c) Fibre-optic view of the vocal cords before extubation on post-operative day 1 shows resolution of the vocal cord oedema and there is now a clear space (arrow head) between the endo-tracheal tube and the vocal cords. (d) Fibre-optic view of the vocal cords before discharge to ward shows normal appearance of the vocal cords

angioedema may be airway manipulation in susceptible patients. [1] The incidence is less with ARBs, though not completely absent. [1]

The other possible causes of vocal cord oedema in this case could have been due to anaphylaxis to anaesthetics or antibiotics, latex allergy or airway injury. Anaphylaxis to anaesthetics or antibiotics will manifest with haemodynamic, cutaneous and/or airway symptoms such as bronchospasm. No features other than epiglottis and vocal cord oedema were present in this patient, and therefore, anaphylaxis can be ruled out; though a serum tryptase analysis would have been confirmatory. We used LMA® ProSeal™, which is latex free, hence latex allergy was ruled out. Airway injury was not present, as there was little visible trauma, even with repeated Instrumentation.

The lessons to be learnt are as follows (i) awareness of this complication among the anaesthesiologists, (ii) early recognition and treatment; treatment steroids and antihistaminics though commonly used is not very successful. Adrenaline (nebulised/subcutaneous) and fresh frozen plasma (for enzyme repletion) are reserved for more severe cases[1] and (iii) counselling of the patient to discontinue the therapy with ACEI/ARB once an episode of angioedema has occurred.[1]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Acknowledgement

Consent for writing this report and using the photographs and videos was obtained from the patient.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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	DOI: 10.4103/ija.IJA_122_18

How to cite this article: Behera BK, Misra S, Kar M. Severe angioedema of laryngeal inlet in a woman receiving telmisartan therapy undergoing breast conservation surgery. Indian J Anaesth 2018;62:393-5.

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