

Severe Presentation of Acute Upper Airway Obstruction – A Case Report

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Abstract

Tongue swelling (glossitis) can be caused by many conditions. We present a case of severe tongue swelling leading to severe, acute upper airway obstruction and its anaesthetic management in the emergency setting.

Keywords: Airway Obstruction, Angio-oedema, Anaphylaxis, Tracheostomy,

Abbreviations: A&E - Accident and Emergency, IV - intravenous, IM - intramuscular, O₂ - oxygen, GCS - Glasgow Coma Scale, ICU - Intensive Care Unit, ENT - Ears Nose and Throat, ALS - Advanced Life Support, CPR - Cardio-pulmonary Resuscitation, ETT - endotracheal tube

Case

A 74 year old female presented to the A&E department after waking with a swollen tongue. She called for an ambulance and the paramedic crew initially treated her for an allergic reaction with 200mg hydrocortisone IV, 20mg chlorphenamine IV and four doses of 0.5mg adrenaline 1 in 1,000 IM. She did not improve and was transferred to the local A&E.

In A&E, she was initially stable with no stridor or difficulty in breathing but noticeably swollen tongue. Saturation were 98% on 5L nasal O₂. Blood pressure was stable at 135/75 mmHg. GCS was 15/15, but the patient was agitated from not being able to speak or retract the tongue. No further history was taken other than an allergy to shellfish, with no recent exposure. An obese habitus was noted.

A&E doctors called the anaesthetic on-call team, and on their request called the on-call ICU consultant and the on-call ENT consultant. Further adrenaline 100mcg IV, hydrocortisone 200mg IV and chlorphenamine 10mg IV were given. An attempt to look inside the mouth with a tongue depressor and torch was made by the anaesthetics/ICU team and it was quickly realised that the swelling continued into the mouth and larynx and was rapidly progressing. Given the lack of suitable equipment and the severity of the case, a decision was made to transfer to emergency theatres.

Once in emergency theatres, the on-call ENT consultant was scrubbed and ready. 100% oxygen via facemask and routine monitoring was instituted. At the request of the ENT consultant, a micro-tracheostomy was attempted with local anaesthesia but failed to pass into the trachea given the patient's habitus. The ENT surgeons attempted an awake tracheostomy, but this was difficult due to her being agitated and unable to lie still, and a calcified trachea.

The rapidly progressing swelling compromised oxygen delivery to the lungs and the saturations began to drop quickly. The patient became bradycardic and lost consciousness. At this point, it became easier to attempt the tracheostomy. The ALS protocol was followed and CPR started. An attempt was made for direct laryngoscopy – a grade 3b view was obtained and a size four microlaryngeal tube was passed successfully. 100% O₂, two doses of 1mg adrenaline IV, and 3mg atropine IV were given and the heart rate improved. Pulses were present and the defibrillator showed sinus rhythm; CPR was stopped and tracheostomy was continued. Due to abundant peri-tracheal fat, a number of tracheostomy tubes were tried before a secure tracheostomy was placed. However the microlaryngeal tube maintained airway patency.

An arterial line and larger cannula was secured. Propofol infusion and fentanyl IV were given to maintain anaesthesia and the patient was transferred to ICU.

On ICU, there was further difficulty in ventilation, with high airway pressures and saturations falling to the low 90s despite 100% O₂. It was thought the tracheostomy tube was abutting the carina or posterior tracheal walls. The ENT surgeons were called urgently and in the interim the patient was re-intubated with a size 7 ETT to maintain the airway. The ENT team changed the size 8 cuffed non-fenestrated tracheostomy tube for a size 7 Shiley with a proximal extension. Despite the change, ventilation remained difficult. An urgent chest X-ray was performed which showed a right-sided pneumothorax. A chest drain was inserted and a 'hiss' was noted on insertion, indicating a possible tension pneumothorax. Ventilation then improved.

Further history was taken from previous notes and discharge letters. It was noted that the patient had allergies to shellfish, penicillin, erythromycin, and diclofenac, but no history of

exposure. She had a background of hypertension, angina, chronic kidney disease and previous breast cancer. She was taking Lisinopril, Diltiazem, Nicorandil, Tamoxifen, Sertraline and Omeprazole. Her tongue swelling improved markedly over the next two days with antibiotic treatment and regular dexamethasone. She was awakened once appropriate and haemodynamically stable. A nasendoscopy was performed by the ENT team on day six and nothing remarkable was noted, with swelling having regressed totally. She was decannulated on day eight and transferred to the ward. Lisinopril was stopped.

Discussion

This case demonstrated a severe, acute presentation of tongue swelling (glossitis) leading to upper airway obstruction. Although a number of conditions may cause glossitis: infection, trauma, anaemia, liver disease, malnutrition. Acute glossitis is a hallmark of angio-oedema. This is a rare, but life threatening condition that requires prompt recognition and treatment.

Angio-oedema may result from anaphylactic, hereditary, acquired or idiopathic processes. Some 12-24% of anaphylaxis cases present with angio-oedema.^{1, 2} Hereditary and acquired cases usually result from a deficiency of C1 esterase inhibitor deficiency, which causes an accumulation of bradykinin, leading to soft tissue oedema. Such an increase in bradykinin may be caused by angiotensin converting enzyme inhibitors, leading to angio-oedema.³ If no cause can be found, it is termed idiopathic.

Management of angio-oedema requires rapid airway assessment and management; resuscitation; and treatment of the underlying cause. Anaphylaxis should respond to standard management as outlined by the AAGBI.⁴ Angio-oedema from other causes requires cessation of the suspected causative agent³, and in an emergency, nebulized adrenaline to reduce airway swelling. Infusion of plasma derived or recombinant C1 Esterase Inhibitor may also rapidly improve symptoms.¹

Although airway support is the cornerstone of anaesthetic management, an acute, rapidly progressing case such as this requires a multi-disciplinary approach.⁵ Anaesthetists will require help from operating department practitioners and nurses to manage the initial airway compromise. A compromised airway presents a significant hazard to any form of anaesthesia, especially if it results in cardio-respiratory depression, which will expedite hypoxemia and impair tissue oxygenation. An awake, spontaneously ventilating approach to secure the airway needs to be undertaken. ENT staff should be scrubbed and ready to perform an emergency surgical tracheostomy if complete airway obstruction occurs or airway access cannot be secured.

An awake fibre-optic intubation can be attempted but this requires an experienced anaesthetist, timely access to equipment, preparation and a co-operative patient. These are unlikely to be provided in the resuscitation room. Fibre-optic

manipulation causing bleeding or further swelling can lead to complete airway obstruction.

An inhalational induction to maintain spontaneous ventilation and then followed by direct laryngoscopy or fibre-optic intubation is another option and reduces the required co-operation of the patient. But this may cause haemodynamic instability in an already compromised patient and can lead to complete airway collapse.

An elective awake tracheostomy under local anaesthetic is the most likely route to ensure airway access without haemodynamic compromise.⁶ This will require a co-operative patient, senior help from trained operating department staff, and the ENT surgeons scrubbed and ready to perform a surgical tracheostomy if a percutaneous approach fails.

The Intensive Care Unit should be aware of the patient and ICU teams will be required to help with airway access as well as manage haemodynamic instability, secure arterial and central venous access. The patient will need further airway support and treatment in the Intensive Care Unit.

An acute upper airway obstruction therefore requires an emergent, yet controlled, approach to secure airway access and maintain oxygenation. Staff should have clear roles, ideally decided beforehand and practiced, with experience in the use of the equipment available. Senior ENT help should be available readily and again be versed in securing an emergency awake tracheostomy should other means fail or are not suitable.

As with any critical incident a debriefing should be undertaken to highlight points in the management of such patients that were handled well and those that were not, so that existing management plans can be improved and skills honed to improve management of future incidents.

Competing Interests

None declared

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