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Case Report

Massive Tongue Swelling Following General Anaesthesia for an Interventional Cardiology Procedure

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ABSTRACT

The case report describes the perioperative events during general anaesthesia for an interventional cardiology procedure that resulted in massive tongue swelling with significant morbidity to an elderly patient. The article revisits this life threatening complication that is rarely mentioned in recent case reports. The article discusses the various likely causes which anaesthetists should be aware about.

Key words: General anaesthesia, Interventional cardiology procedure, Massive tongue swelling.

INTRODUCTION

Upper airway obstruction during recovery from general anaesthesia may have several causes. [1] This report presents a case of massive tongue swelling which resulted in extended postoperative ventilation and prolonged hospital stay after an otherwise uneventful interventional cardiac procedure. Discussion regarding this potentially life-threatening scenario is very scant in recent anaesthesia literature.

CASE REPORT

A 76-year-old gentleman (84 kg, 173 cm, body mass index 28 kg/m^2) presented to the operating theatre to undergo an elective percutaneous insertion of a left atrial appendage (LAA) occluder device for his chronic atrial fibrillation (AF). The patient had extensive medical history including AF, permanent pacemaker (for heart block), ischemic heart disease, hypertension, obstructive pulmonary disease, and obstructive sleep apnoea on CPAP mask, hypothyroidism, gout and gastroesophageal reflux. Long term medications included warfarin, metoprolol, ramipril, frusemide, pregabalin, thyroxine, amiodarone, atorvastatin, pantoprazole, allopurinol, alendronate and paracetamol. He had no known allergies. No complications with previous operations were noted by the patient or in the medical record. He was independent with his daily activities and was living at home with his family.

Clinical examinations including airway assessment were observed as unremarkable except for being edentulous. The warfarin had been ceased 4 days prior to the procedure and admission point of care testing of capillary Prothrombin Time/INR was 13/1.1(normal range 8-14/ 0.9-1.3). As per protocol, loading dose of oral dual anti platelet therapy (Aspirin 300 mg, Clopidogral 300 mg) was given at 09.30 am; half an hour prior to transfer to theatre. After securing peripheral venous access and standard monitoring including an invasive arterial pressure line, the patient was induced with 100 µg fentanyl, 100 mg propofol and 50 mg rocuronium. Initial bag and mask ventilation was manageable

without any use of airway device. Laryngoscopy done by the primary anaesthetist who had more than 10 years experience showed a Cormack and Lehane (C&L) grade 2b view using Macintosh size 4 blade. Ramping patients are often difficult on operating tables in hybrid theatres. Hence second attempt was tried after optimising the view by keeping an additional pillow to flex the neck and extend the head. Since there was no change in laryngoscopy view it was decided to use a bougie. Successful intubation was achieved in the third attempt of laryngoscopy by rail-roading a 8.5 mm internal diameter endotracheal tube (ETT) over the bougie. A transesophageal (TEE) probe was subsequently inserted under manual tactile guidance by the same anaesthetist. Head was maintained in neutral position throughout the procedure.

Anesthesia was maintained using desflurane and no further sedatives/opioids were administered. Antibiotic prophylaxis was with Teicoplanin. In addition to granisetron (1mg) a higher dose dexamethasone (8mg) was given for anti-emetic prophylaxis, considering the risk of laryngeal edema from multiple attempts at intubation. In total around 750 ml of crystalloid solution was administered in theatre during the case. The interventional procedure went uneventful and the LAA occluder device was inserted successfully. Intravenous heparin 8500 i.u was given intra operatively (one hour after intubation) and subsequent Activated clotting time (ACT) was 401 seconds. The anaesthetic duration was 2 hours and muscle relaxant was reversed with standard dose of neostigmine/ glycopyrrolate (2.5/0.4 mg). The oropharynx was suctioned using a Yankauer suction tip, and the trachea was extubated with the patient awake and without coughing. After ensuring patients ability to breathe and maintain airway he was shifted to recovery by the anaesthetic team.

By the time the patient arrived in the post anesthesia care unit (PACU) at 12.30 pm, he was observed to be un-arousable. A size 4 Guedel airway was inserted to open the airway. Suspecting continuing residual motor blockade 200 mg Sugammadex was given which resulted in immediate effect in the form of response to commands and good tidal volume spontaneous breaths. The 5 minute observations were recorded as haemodynamically stable, maintaining own airway and saturating 99% on a 6 L per minute Hudson mask. Sedation score was observed as mild (occasionally drowsy, easy to arouse). The ACT at 13.30 was 339 sec. By 13.45 the patient had met all the PACU discharge criteria and was ready for discharge to the coronary care unit (CCU); but the transfer was delayed due to administrative reasons (CCU bed not available).

The condition of the patient was stable until 14.30 h when he was observed to develop a noisy breathing. PACU nurses noticed "a significant swelling of the tongue with some bleeding". The patient also verbalised that he was having trouble breathing. Thereafter, his saturation dropped rapidly to less than 80% with associated bradycardia. A MET (medical Emergency Team) call was activated. Multiple anaesthetists arrived immediately and successful bag and mask ventilation was initiated with the help of a #4 Guedel airway. ACT repeated at that time was 230 sec and Protamine 50 mg was given intravenously to reverse the residual heparin effect. At 14.40 a needle cricothyroidotomy was done using a 20 G venous cannula as a backup for either jet ventilation or tube insertion through seldinger technique. Considering the airway swelling and potential for clinical deterioration it was decided to re-intubate the patient. After oral and transtracheal topicalisation with lignocaine, laryngoscopy was done using a video laryngoscope(C-MAC #3 Mac Blade). There was a massive swelling of the tongue which made the insertion of the laryngoscope difficult. After multiple attempts the blade was pushed into the oropharynx which showed a C&L grade 2 views on the video laryngoscope screen. A

size 7.5 mm ID ETT was inserted with the help of a bougie and the patient was transferred to intensive care unit (ICU) for further management.

The initial assessment in ICU noted "a large subungual hematoma obstructing the entire mouth and unable to pass finger beyond tongue". Consultation was done with the otolaryngology (ENT) team who suggested continuing observation and computed tomography (CT) imaging. Heavy bruising on both the groins where the vascular access sheaths had been inserted was also observed. The presumptive diagnosis at this time was bleeding into tongue caused by trauma from laryngoscopy/ TEE probe in an anti-coagulated patient. However, CT imaging done the next day at 16.00 hrs was reported as "Loss of the parapharygenal fat plane suggestive of soft tissue oedema surrounding the pharynx. No definite collection or haematoma is identified within the floor of mouth or neck" (See Figure. 1). The ENT team was happy with the progress and suggested no further intervention was required. The patient was kept intubated and ventilated until airway swelling was deemed on visual inspection to be not significant enough to prevent a trial of extubation. Rest of his ICU stay was uneventful. Unfortunately the swelling was slow to resolve and he was successfully extubated only on day 5 post procedure. In addition, even after transfer to ward, he appeared de-conditioned and required further regular follow-up by speech therapist and physiotherapy team until he was fit enough to be discharged from hospital on day 13.

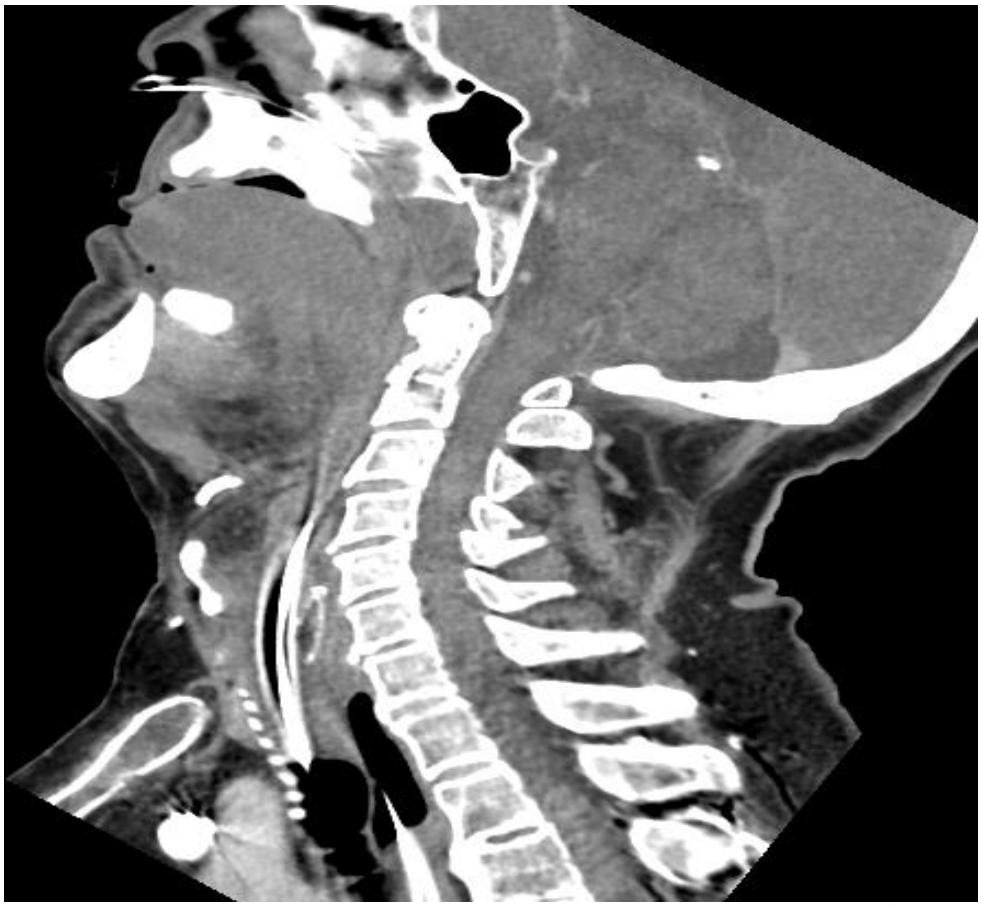

Figure 1: Sagittal plane computed tomography neck (IV contrast dual phase CT neck with multiplanar reformats) showing soft tissue oedema surrounding the pharynx. No definite collection or haematoma identified within the floor of mouth or neck.

DISCUSSION

The events described display a rapid decline in the clinical condition of a patient resulting in a potentially life threatening emergency, saved only by the vigilance of the PACU staff and the appropriate

intervention by the anaesthetists who responded. It is possible that airway swelling was progressing slowly and reached a critical point which resulted in the dramatic presentation. Multiple mechanisms for swelling of the oropharynx in the perioperative period that could have resulted in the tongue swelling in the patient can be postulated.

Direct tissue trauma [2] or an obstruction in venous, lymphatic [3] or salivary gland [4] flow can result in a swollen tongue. Perioperative tongue swelling or even necrosis in the setting of TEE has also been described in the past, but the majority of tongue pathology in the perioperative period is attributed to endotracheal tube position, the duration of intubation, or the surgical procedure itself (i.e., head and neck surgery, presence of throat packs, prone positioning, and risk for venous congestion). [5] Most patients who come for the cardiac interventional procedures are given a loading dose of dual anti platelet agents with the peak effect around 2-5 hours. It is our practice to have a very low threshold to use video laryngoscopes (VL) to decrease trauma associated with intubation in such cases. VL was not our primary choice in the current case since preoperative airway assessment was assessed to be of no concern. However on obtaining inadequate C&L view, the primary anaesthetist had taken appropriate rectifying measures in the form of re-positioning and use of bougie to avoid excessive force during the attempts of intubation.

Other potential reasons include hypersensitivity and local tissue reaction. Hypersensitivity response to any stimulant including medications could potentially result in airway swelling. However in our patient, there were no other associated symptoms or other systems involved which could compel to suspect an allergic reaction. Local tissue reaction to chemicals used to clean the laryngoscope [6] or TEE could potentially cause this type of swelling. Even though the laryngoscope blades used were disposable single use, the TEE had been sterilised as per current standard of sterilisation. There was no other incidence of airway reaction reported during that period in our department which could have made us suspect any concern with our practice of TEE sterilisation.

Angioedema is another interesting cause of airway swelling that has been reported in the literature. [7] It is a condition in which small blood vessels leak fluid into the tissues, causing swelling which may last between 1 and 3 days. Even though angioedema owing to other causes may occur anywhere on the body, angioedema associated with ACE inhibitors, in contrast, demonstrates an unusual predilection for the head and neck.

In retrospect, it was providential that an administrative delay ensured that the event occurred in PACU where the presence of one to one nursing care, immediate accessibility of multiple anaesthetists with inherent airway management skills and availability of emergency airway equipments resulted in successful resuscitation of the patient from a peri-arrest situation due to respiratory failure. A significant number of post operative patients experience considerable pain in the PACU that necessitates administration of opioids with inherent sedative effects. This could easily result in a patient with early stages of airway swelling that is not noticed or brought to attention by a sedated patient. It is also essential that receiving PACU staffs are made aware of all intra operative airway incidents through a structured hand over and appropriate documentation. Unless specifically examined for, minor degrees of airway swelling could be unobserved until they reach a tipping point as demonstrated by our case report. It is advisable to exert low threshold to delay discharge from PACU if any concerns exist regarding airway in the peri operative period.

CONCLUSION

Our case report presents an unfortunate rare complication from an

unconfirmed aetiology resulting in considerable morbidity for an elderly patient. Care must be taken during insertion of ETT or TEE probe in elderly patients especially if on anticoagulants. Extreme caution should be exercised before transferring patients with history of any potential peri-operative airway trauma from high care nursing area to peripheral wards.

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