Anaesthetic management of case of Ludwig’s angina: Forewarned is forearmed

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Abstract
Ludwig’s angina and deep neck infections are potentially lethal entities because of their tendency to cause oedema, distortion, and obstruction of the airway and may arise as a consequence of airway management mishaps. In the early stages of the disease, patients may be managed with observation and intravenous antibiotics. Advanced infections, however, require the airway to be secured in addition to immediate surgical drainage. This is complicated by pain, trismus, airway oedema, and tongue displacement creating a compromised airway. Awake fibreoptic intubation under topical anaesthesia may be the ideal method to secure the airway in advanced cases of Ludwig's angina. Here we discuss the successful anaesthetic management with fibreoptic nasal intubation of a 20-year-old female with Ludwig's angina scheduled for emergency drainage with a brief review of airway management options.

Keywords: Ludwig angina, fibre optic bronchoscopy, difficult airway

1. Introduction
While described as far back as the writings of Hippocrates and Galen, the necrotizing fasciitis Ludwig's angina was first detailed by the German surgeon Wilhelm Friedrich von Ludwig in 1836 as a rapidly and frequently fatal progressive gangrenous cellulitis and oedema of the soft tissue of neck and floor of mouth. It originates in the region of the submandibular gland with elevation and displacement of the tongue and rapidly progresses to involve the sub-lingual, sub-mental, and sub-mandibular spaces. Airway compromise is always synonymous with the term Ludwig’s angina and it is the leading cause of death. Therefore, airway management is the primary therapeutic concern. The treatment plan for each patient should be individualised and based on a number of factors. The stage of disease and comorbid conditions at the time of presentation, physician experience, available resources, and personnel are all crucial factors in the decision making.

2. Case report
A 20-year-old female, weighing 48 kgs and 153 cms in height, presented to our hospital with complaints of facial and neck swelling. She gave history of difficulty in breathing, swallowing, and inability to open the mouth since 2 days. She described a five-day history of lower left quadrant tooth pain, and a three-day history of fever and chills. On presentation, her vital signs were: temperature 39.7°C, blood pressure 100/54mm of Hg, pulse 128/min, respiratory rate 24/min, oxygen saturation on room air 90%, and haemoglobin of 4 g%, white cell count of 18,000/μL. Her clinical examination revealed severe pallor, severe facial oedema, large soft tissue swelling under her mandible, extending bilaterally to the angles of the mandible and inferiorly up to suprasternal area. On airway examination mouth-opening was restricted, with an interincisor gap of 1 cm. She had respiratory distress on sleeping and was uncomfortable because of pain and intra-oral drainage of pus. Neck extension was very painful and extremely limited. Both the nares were patent and the trachea was not palpable. She was nil by mouth for more than 8 hours. (Figure 1).

Soft tissue neck x-rays showed an increase in the submandibular and pretracheal space. Ultrasonography (USG) reported fluid collection in the submandibular region and neck, anterior to the thyroid and between the strap muscles of neck, bounded laterally by the carotids, tracking deep along the lateral border of the thyroid.
A diagnosis of Ludwig's angina was made and she was scheduled for emergency drainage of the abscess. Awake fibreoptic intubation was planned, with tracheostomy as a backup. The procedure and need for awake nasal intubation was explained to the patient and written informed consent was obtained for awake intubation and tracheostomy.

2.1 Anaesthetic management

Pre-operative preparation

Patient received nebulisation with 2ml 4% Lignocaine half hour before surgery. Difficult airway cart and emergency tracheostomy kit were kept ready. Surgeons were on standby for tracheostomy.

In the operating room, an 18 gauge intravenous line was secured and standard monitoring was started. Patient was premedicated with inj. Glycopyrrolate 0.2 mg, inj. Ranitidine 50 mg, inj. Ondensatron 4 mg and inj. Dexamethasone 8mg intravenously as per protocol. Nasal decongestion was accomplished using xylometazoline nasal drops, and lignocaine 4% topical, was used to anesthetize the nasal mucosa. The base of the tongue and pharyngeal walls were anesthetized with lignocaine 2% viscous gargle 5 ml and 10% lignocaine two puffs, which was sprayed onto the posterior pharyngeal wall.

The fiberoptic bronchoscope was checked and loaded with a 6.5 mm ID cuffed endotracheal tube. The fiberscope was inserted into the right nostril. Lot of difficulty was encountered in visualisation, as pus was draining intra-orally and periglottic oedema was present. An orally placed suction catheter, however, made the fiberoptic easier. Two ml of 2% lidocaine was sprayed on the vocal cords down to the trachea and after waiting for a minute, tracheal intubation was achieved successfully. After confirmation of tracheal intubation anaesthesia was induced with fentanyl 80 mcg and propofol 80 mg. Vecuronium 4 mg was also given. Anaesthesia was continued with nitrous oxide in oxygen, propofol infusion and intermittent positive pressure ventilation. Intra-operatively the vitals were stable. Incision and drainage were performed by the sub-mandibular route and a drain was left behind.

At the end of the procedure residual neuromuscular blockade was antagonized using neostigmine 3 mg and glycopyrrolate 0.6 mg. In view of the periglottic oedema and severity of the patient’s condition, the trachea was not extubated, and the patient was moved to a SICU with the endotracheal tube in situ and placed on a T-piece with oxygen at a flow rate of 5 litres per minute. (Figure 2)

After 2 days of monitoring in SICU, oedema had considerably subsided, a thorough oral suction was performed and the trachea was extubated. Post extubation recovery was uneventful. The patient was discharged 4 days later.

3. Discussion

Ludwig's angina was formerly invariably fatal but now, with adequate surgical and antibiotic treatment, has a much reduced rate of mortality. It remains, however, a potentially life-threatening condition because of the risk of impending airway obstruction. Thus, because of its invasive nature, early recognition and treatment of Ludwig's angina is extremely important. The most serious complication of Ludwig's angina is asphyxia caused by expanding oedema of soft tissues of the neck. Another common cause of death is the acute loss of airway during
interventions to control the condition. Stridor, difficulty managing secretions, anxiety, cyanosis, and sitting posture are late signs of impending airway obstruction and indicate the need for an immediate artificial airway. Streptococcus viridans (40.9%), Staphylococcus aureus (27.3%), and Staphylococcus epidermis (22.7%) were isolated from deep neck infections. Intravenous penicillin G, clindamycin or metronidazole are the antibiotics recommended for use prior to obtaining culture and antibiogram results. Some authors also recommend the association of gentamycin. Recent case reports advocated the use of intravenous steroids which potentially avoided the need for airway management.

Airway management of patients with Ludwig’s angina presenting for surgical drainage is a challenging task for the anaesthesiologist. The suggested methods include tracheostomy, conventional laryngoscopy and intubation (after administration of muscle relaxant), awake blind nasal intubation and awake fibreoptic intubation. Decompression of Ludwig’s angina under cervical block has also been reported.

We prepared the patient preoperatively with intravenous dexamethasone and nebulsed adrenaline as they have been shown to reduce upper airway oedema in such cases to defer or avoid artificial airway instrumentation altogether. As our patient had distorted airway anatomy, neck and tissue immobility, and limited access to the mouth orotracheal intubation by direct laryngoscopy was not considered. Blind nasal intubation was avoided as, besides having a high failure rate; it could cause catastrophic bleeding, laryngospasm, airway oedema, rupture of pus into the oral cavity, and aspiration. In advanced cases, induction of general anaesthesia is dangerous because this may precipitate complete airway closure and make mask ventilation and intubation impossible.

Considering the above factors we planned to secure the airway in the awake state as it was the safest option for our patient. Elective awake fibreoptic intubation has been suggested for all patients with deep neck infections in order to avoid the dangers of emergency tracheostomy in a severely compromised airway. We considered tracheostomy as a backup plan if FOB failed. Although distorted anatomy, oedema, and secretions may contribute to difficulty with fiberoptic intubation, in skilled and experienced hands, flexible fiberoptic nasal intubation is the preferred method of airway management and has a high rate of success. Application of topical anaesthesia enables the patient to tolerate the procedure with greater comfort.

4. Conclusion

Awake fibreoptic intubation under topical anaesthesia is a sophisticated and less invasive method of securing airway in patients with deep neck infection and should now replace the gold standard tracheostomy in managing the same. The choice of airway manoeuvres and management must be individualized, depending on the judgment and experience of the anaesthetist and surgeon in charge.

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