Anaesthetic management of Ludwig's angina with comorbidities

INTRODUCTION

Ludwig's angina is a rapidly progressing necrotising cellulitis affecting the posterior oropharynx, submaxillary and sublingual spaces. It can cause elevation and displacement of the tongue resulting in airway obstruction. Death in such cases occurs due to airway compromise.[1] The most common cause is tooth extraction or dental infection. It can be complicated by airway compromise or extension into the mediastinum or extension into deeper soft tissues like cervical fascia.[2] The treatment involves appropriate antibiotics, airway control and surgical intervention (incision and drainage).

CASE REPORT

A 35-year-old male patient weighing 80 kg presented with a swelling in the left side of the face and neck since 5 days following dental infection. The patient was admitted to the hospital 2 days before and started on antibiotics by the surgeon (ciprofloxacin, metronidazole), in spite of which swelling was progressive, associated with pain, with inability to swallow saliva. There was no fever, chills or rigors. The patient was diagnosed as hypertensive and diabetic 15 days ago and started on tablet telmisartan 20 mg. He was not treated for diabetes. The patient was a known asthmatic since childhood on salbutamol, ipratropium and budesonide nebulisation and was a smoker since 5 years, smoking 4–5 cigarettes/day. He was not able to lie down supine due to difficulty in breathing and was feeling better in left lateral position. On examination, heart rate was 126 beats/min and blood pressure 150/110 mm of Hg. Airway examination showed mouth opening of one finger breath with trismus and limited neck extension and sternomental and thyromental distance could not be assessed. The swelling was diffuse extending from the left side of the face to the entire anterior aspect of the neck down until the upper end of the sternum and front of the manubrium sternum [Figures 1 and 2]. There were brawny oedema and erythema over this region. Nasal flaring and bilateral rhonchi were present. Investigations revealed increased fasting blood sugar (210 mg/dL) and post-prandial blood sugar (345 mg/dL). Arterial blood gas showed pH of 7.50, PCO_2 of 34.5 mm Hg and PO_2 of 64 mm Hg and HCO_3 of 26.5 mmol/L.

The patient was explained about the risk associated and plan for awake fibreoptic intubation. Informed consent

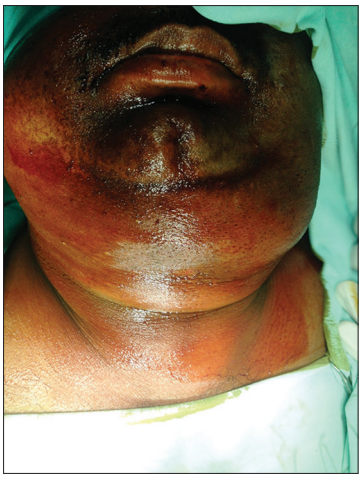

Figure 1: Diffuse neck swelling

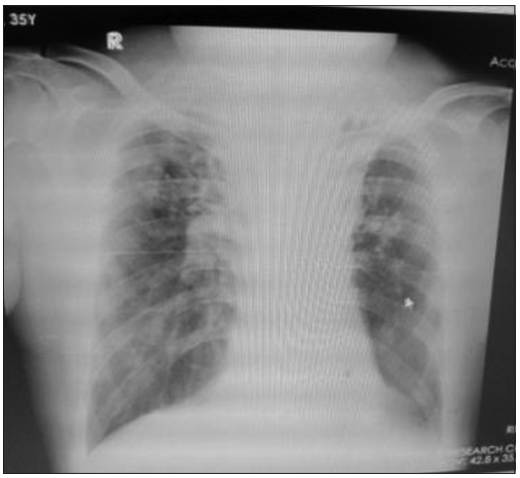

Figure 2: Anteroposterior X-ray chest and neck

and consent for fibreoptic intubation were taken. Bronchodilator nebulisation was given pre-operatively. In the OT, non-invasive blood pressure, pulse oximeter and 5 lead electrocardiography were connected and vitals noted. He was maintaining a saturation of 93% on room air. Intravenous (IV) glycopyrolate 0.2 mg and fentanyl 40 μg was administered. Airway was prepared with nasal and oropharyngeal sprays with 10% lignocaine. A size 7 endotracheal tube was rail-roaded on the fibre optic bronchoscope. Nasal fibreoptic bronchoscopy was performed with the patient in a lateral position with spray as you go technique to anaesthetise the larynx and trachea and airway secured. We encountered a slight difficulty while threading the endotracheal tube. The patient was induced with injection proprofol 100 mg and injection fentanyl 60 μg with injection vecuronium 6 mg as the muscle relaxant and maintained on oxygen, nitrous oxide and isoflurane. Injection dexamethasone 8 mg was administered. Vitals were maintained within normal limits. At the end of the surgery, muscle relaxation was reversed with injection neostigmine 2.5 mg and injection glycopyrollate 0.4 mg and patient was shifted to intensive care unit (ICU) with endotracheal tube *in situ* for elective extubation next morning. In ICU, oxygen was supplemented via T-piece. Fentanyl and IV paracetamol, dexamethasone, antibiotics, and nebulisation with bronchodilators were continued and patient was started on insulin. The next day patient was maintaining saturation without oxygen.Vitals were within normal limits and trachea was extubated keeping bougie as a standby.

DISCUSSION

Ludwig's angina described by Ludwig and Grodinsky is cellulitic infection of submandibular space, usually involving more than one neck space, producing firm induration of floor of mouth and posterior displacement of tongue, and spreads by continuity along the fascial planes, then by lymphatics and rarely involving the glandular structures.[3]

Mortality of patients with Ludwig's angina in the pre-antibiotic era was around 54% and 0–8% after availability of antibiotics.[4] The most common organisms involved are *Streptococcus*, *Staphylococcus* and anaerobes. In most cases, it is the submandibular space which is the primary source of infection. Following dental abscess or dental extraction the infection spreads from the root of second or third molars to the submaxillary space to the sublingual space. The infection further spreads contiguously to involve the pharyngomaxillary and retropharyngeal space.[1] The primary management of the patient involves appropriate antibiotic therapy covering gram-positive, gram-negative and anaerobic infection. Patients not responding to antibiotic therapy or those associated with airway compromise require surgical manipulation. Computed tomography or magnetic resonance imaging is a very useful aid in diagnosing the extent of spread and airway involvement.[5]

In our patient, the co-morbidities included airway compromise which was managed successfully, and hypertension (on tab. telmisartan), which was managed intraoperatively with optimal analgesia, using fentanyl and lignocaine.His fasting sugar level was 210 mg% and random sugars came down to 175 mg%, and was not treated.

Airway management involves either conventional laryngoscopy, awake fibreoptic intubation, surgical tracheostomy under local anaesthesia or

cricothyrotomy in an extreme situation. Blind nasal intubation can be an alternative in the absence of an intraoral extension of the infection.

In our patient, tracheotomy (could be difficult) was the last resort if fibre-optic intubation was difficult. The anaesthetic challenge in our patient was that he had already progressed to a stage where he had symptoms of airway obstruction and was becoming exhausted even with slight movements. Tracheostomy was not possible for this patient because the infection had spread around the neck anteriorly and laterally and lower down, till the anterior aspect of upper sternum obliterating the anatomical landmarks. Moreover, the patient was not able to lie in the supine position. Airway oedema can be reduced using steroids[6]

Fibreoptic intubation following induction and muscle paralysis can cause the collapse of the airway and inability to mask ventilate due to anatomical distortion. Hence, the best option was to go ahead with awake fibreoptic nasotracheal intubation. It also helps in visualisation of the airway, minimising trauma if done carefully. The risk associated with this method include chances of intraoral rupture of the abscess with risk of aspiration and in case of bleeding, airway compromise. We went ahead with nasal fibreoptic intubation in left lateral position supplementing oxygen by facemask kept in front of the face as continuous suction was necessary. Our patient had desaturated to 75% by which time airway was secured.

Airway management expertise is essential in every medical speciality and maintaining a patent airway is essential for adequate oxygenation and ventilation and failure to do so, even for a brief period, can be life-threatening.[7]

CONCLUSION

Airway management of a patient with Ludwig's angina depends on the condition of the patient and the facilities available in the institution. The presence of an experienced anaesthesiologist and the decision making regarding the airway is very important as there are no set guidelines for airway management here. Fibreoptic laryngoscope is a very useful aid in such situations. These patients usually have a good recovery following surgery.

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Conflicts of interest

There are no conflicts of interest.

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