Acta Scientific Otolaryngology (ISSN: 2582-5550)

Volume 4 Issue 9 September 2022

Case Report

Non-surgical Management of Laryngeal Oedema in Ludwig's Angina

Ukamaka Nwankwo[1]*, Raji Ayanwale Victor[2], Rony Paul Kirianthan[3] and Andriy Danyltsiv[4]

[1]ENT Surgeon, Evercare Hospital Lekki, Nigeria

[2]Director, Medical Critical Care, Evercare Hospital Lekki, Nigeria

[3]Director, Surgical Critical Care, Evercare Hospital Lekki, Nigeria

[4]Consultant Anaesthetist, Nigeria

***Corresponding Author:** Ukamaka Nwankwo, ENT Surgeon, Evercare Hospital Lekki, Nigeria.

Received: July 18, 2022

Published: August 05, 2022

Abstract

Ludwig's angina is a cellulitis involving the floor of the mouth and neck. Its spread is fast involving adjacent tissues and is most often caused by odontogenic infections. Associated oedema may involve vital structures like the larynx and hypopharynx causing upper airway obstruction and dysphagia. Management of laryngeal oedema is critical in patients with Ludwig's angina. Intravenous steroid administration and racemic epinephrine have been used successfully in selected cases for the management of laryngeal oedema.

This is a case report of the management of a 59-year-old male with one week history of submental mass and progressive dysphagia. Associated laryngeal oedema was managed non surgically with intravenous steroids and epinephrine nebulization. This report highlights the advantage of non-surgical management of laryngeal oedema in selected cases of Ludwig's angina.

Keywords: Ludwig's Angina; Laryngeal Oedema; Oxygen

Introduction

Ludwig's angina is an infection characterized by soft tissue cellulitis and inflammation involving the floor of the mouth and neck. It was first discussed in 1836 [1] and may involve any or all of the 3 compartments of the floor of the mouth: sublingual, submental and submandibular. This infection can progress fast leading to complications like upper airway obstruction, aspiration pneumonia, sheath abscess and carotid rupture [2]. Odontogenic infection involving mostly the second and third lower molars are responsible for Ludwig's angina in about 90% of cases [3]. This infection is polymicrobial involving Gram-positive, Gram-negative bacteria and anaerobes. Predispositions include diabetes, immunocompromising conditions, malignant lesions of the oral cavity and dental caries [3]. These predisposing factors can make management of this infection more difficult.

Early presentation of Ludwig's angina is managed conservatively with intravenous antibiotics and close monitoring provided there are no co-morbidities. The management of advanced or rapidly progressive cases depend on co-morbidities, associated complications, available resources, personnel and clinician's experience [4].

Laryngeal oedema causes a decrease in the size of the lumen increasing the work of breathing and presenting as stridor, tachypnea, use of accessory respiratory muscles and reduced oxygen saturation. Laryngeal oedema due to Ludwig's angina cannot be distinguished from that due to other pathologies so management is similar either conservatively or surgically. Marked laryngeal oedema may make endotracheal intubation difficult and surgical modalities like cricothyroidotomy, percutaneous

tracheostomy and surgical tracheostomy may have to be used to relieve upper airway obstruction. Therefore, airway protection is very critical in the management of Ludwig's angina.

Case Report

A 59-year-old male, known diabetic and hypertensive, presented with one week history of progressive submental swelling and 2 days history of progressive dysphagia. There is associated odynophagia. There was no recent history of dental extraction. On examination he was found to be afebrile (37 ℃), not pale, in painful distress with a respiratory rate of 22 and oxygen saturation of 100% on room air. Blood pressure was 153/86 mmHg and he had a pulse rate of 103. Mild trismus was noted on oral examination. There were numerous missing dentition and amalgam tooth filling in remaining molar teeth. Abutment teeth holding the bridge prosthesis was found to be mobile. Both tonsils were enlarged but not inflamed. A marked submental and submandibular swelling was noted with warm and mild erythema of overlying skin. It was firm in consistency and tender. His blood sugar level was 159mg/dl, HbA1C was 8.78%. White cell count was elevated with C reactive protein suggestive of marked acute inflammation. A diagnosis of Ludwig's angina was made with poor control of blood sugar and pressure. He was then admitted and commenced on intravenous ceftriaxone (Rocephin) and metronidazole, fluids and parenteral analgesics. He was reviewed by the endocrinologist and placed on oral hypoglycaemic medications.

The next day he had a contrast CT scan of the neck and chest done which revealed submental and submandibular soft tissue inflammation and lymphadenopathy. There was no finding of necrotizing fasciitis or deep neck abscess. Endoscopic laryngeal examination revealed marked oedema of the laryngeal and hypopharyngeal structures. The vocal cords could not be visualized due to the overhanging oedematous epiglottis (Figure 1).

Figure 1

Intravenous steroids and nebulized adrenaline were commenced to reduce laryngeal and hypopharyngeal oedema, thereby reducing the risk of upper airway obstruction. Blood glucose level from the time of admission was between 145 – 191mg/dl. He was commenced on insulin to control anticipated steroid induced hyperglycaemia. Fourteen hours later, he was able to swallow both solid food and liquids effectively but his blood glucose level increased significantly to 326mg/dl. Intravenous dexamethasone was then discontinued.

Orthopantomogram revealed marked radiolucency between the bridge prosthesis and the residual bone indicative of ongoing periodontal infection (Figure 2).

Figure 2

Surgical drainage could not be done then due to poor glycaemia control and was scheduled for the next day. Five hours later, there was marked increase in the size of the submental and submandibular mass with complaint of difficulty in breathing and swallowing. He was found to be tachypnoeic with oxygen saturation of 97% on room air. He was then transferred to the ICU for close monitoring and place on oxygen via nasal prongs at 4L/minute. Oxygen saturation became 100%, respiratory rate 16 and blood pressure recorded was 155/80 mmHg.

He was taken to the theatre for emergency surgical decompression. A consent for elective tracheostomy was taken in the event of failed endotracheal intubation. Video guided endotracheal intubation was successful though difficult due to marked laryngeal oedema. Surgical decompression was done and drains inserted. He was taken back to the ICU for close monitoring and mechanical ventilation. His intravenous antibiotic post op was changed to meropenem and metronidazole and insulin administration continued.

Endoscopic laryngeal examination done 2nd post operative day still revealed significant laryngeal oedema. He was then commenced on intravenous dexamethasone. The next day, the endotracheal tube was dislodged into the oral cavity by the patient in his attempt to pull out the tube. He was then closely monitored but had to be re-intubated due to stridor and use of accessory respiratory muscles. He was reintubated but with a bigger sized tube (7) as against size 6.5 used in the theatre signifying reducing laryngeal oedema. Endoscopic laryngeal examination done on the 4th post operative day revealed marked reduction of laryngeal oedema (Figure 3).

Figure 3

Cuff leak test done was negative. He was then extubated and monitored closely in the HDU for 48 hours. Surgical drains were also removed. He was transferred to the ward due to marked clinical improvement and discharged on the 8th postoperative day with good glycaemic and blood pressure control.

Discussion

Ludwig's angina is a cellulitis of the neck and floor of the mouth. Majority of the cases are due to odontogenic infections [1,3]. It is polymicrobial and antibiotic treatment should therefore cover anaerobes, Gram-positive and Gram-negative bacteria. Broad spectrum antibiotics should be commenced promptly. Penicillin, clindamycin and metronidazole are antibiotics used most often [5]. Pseudomonas should be covered in immunosuppressed patients [3].

There are no specifications for airway management in Ludwig's angina but the "Practice Guidelines for Management of the Difficult Airway" adopted by the American Society of Anaesthesiologists advocates intubation via fibre-optic bronchoscopy in conscious patients with difficult airway [6] but a surgical tracheostomy should be prepared for under local anaesthesia if intubation is not successful. The guidelines also noted that management of difficult airways can be modified depending on the situation at hand. Blind nasotracheal intubation should not be attempted due to complications like abscess rupture and bleeding [4,7]. Surgical tracheostomy may be difficult in some advanced cases due to distortion of the neck anatomy or difficult positioning of the patient [8].

There are different ways of evaluating the airway for laryngeal oedema. Video laryngoscopy can be used in evaluation both in intubated and non-intubated patients but cuff leak test (CLT) and laryngeal ultrasonography are used in intubated patients [9]. Cuff leak test and laryngeal ultrasonography are non-invasive tests which help to determine the available lumen, that is the space between the endotracheal tube and wall of the laryngeal lumen. Cuff leak test is very easy to do. A positive CLT, signifying no no air leak envisages the possibility of post extubation failure leading to a postponement of extubation process while air leak on cuff deflation is noted as negative, that is the possibility of post extubation failure is minimal [10,11]. But due to different sensitivity (15 - 85%) and specificity (70 - 99%) results noted with CLT prediction of stridor post extubation, it cannot be used alone to predict post extubation respiratory distress [9,12]. Meta-analysis of 14 studies noted that CLT reduced the incidence of stridor post extubation and frequency of re-intubation but increased the delay in extubating the patient by 9% [13]. Ultrasonography of the larynx determines the air column width (ACW). ACW is the acoustic shadow width at the level of the vocal cords pre and post cuff deflation [9]. There is increase in the ACW post cuff deflation when there is resolution of laryngeal oedema but adequate and gentle suctioning of the airway should be done prior to this measurement. With video laryngoscopy, the larynx can be visualized and it has the advantage of distinguishing laryngeal oedema from other laryngeal lesions that can compromise the airway. In this index patient, serial endoscopic laryngeal examination was done to assess airway patency and cuff leak test done when marked reduction in laryngeal oedema was noted. These 2 methods of assessments were used to guide extubation process.

Intravenous steroid use as a treatment modality for soft tissue oedema, thereby reducing the need for a surgical airway has been recommended [14-16] but no randomised controlled study has been done to confirm the benefits of its use in the management of laryngeal oedema. Its modality of action is the reduction of inflammatory response through decreased synthesis of pro-inflammatory cytokines, antibody Fc receptor expression and T-cell function [17]. Using intravenous steroids is problematic in patients with diabetes as it is known to cause drug induced hyperglycaemia [18]. This can also lead to prolonged hospital stay. Insulin is used in such conditions to deal with associated hyperglycaemia [19]. In this patient, though there was relief of dysphagia due to reduction of hypopharyngeal and laryngeal oedema with the commencement of intravenous dexamethasone, there was rapid and significant increase in blood glucose levels despite commencement of insulin necessitating the discontinuation of dexamethasone use and delay in surgical decompression. Hours later, there was marked increase in the size of mass with associated respiratory distress and dysphagia. Therefore, the benefits of intravenous steroid use in the management of laryngeal oedema in this patient is noted but the hyperglycaemia associated with it being used in a known diabetic with poor glycaemic control limited its beneficial use pre-op. Post operatively, its use significantly reduced laryngeal oedema and hastened extubation process.

Epinephrine nebulization is also advocated [16] because it reduces laryngeal oedema by vasoconstriction and reduced blood flow to involved tissues [20]. Its use has been found to be beneficial in the management of upper airway obstruction in children [21] and other conditions [20]. It is safe with little or no adverse effects. One milligram of adrenaline diluted with 5 ml of normal saline has been used effectively in the management of laryngeal oedema [22]. Nebulization with epinephrine is difficult to achieve in an intubated patient, in this case, intravenous steroid administration is more effective. In our patient, it was used pre-operatively and post extubation.

Conservative management of the airway has been advocated in selected cases. Surgical tracheostomy was required in 10% of patients with Ludwig's angina in a 2005 study [23], 10% of children and 52% of adults [24] and 3% in a 2007 study [25] where conservative airway management was done. Close monitoring is vital with all preparations made in the event that surgical intervention is required. Appropriate antibiotics treatment should be started promptly and surgical drainage performed if required [5]. Surgical decompression improves clinical outcome in those requiring endotracheal intubation [26]. Airway management should be based on respiratory rate, oxygen saturation and endoscopic laryngeal findings. Patient should always be closely monitored for disease progression and development of complications.

Conclusion

Management of laryngeal oedema in Ludwig's angina is critical to the patient's well-being. Intravenous steroid administration and epinephrine nebulization have shown good promise. Randomized controlled studies should be done to confirm the beneficial use of intravenous steroids in the management of laryngeal oedema.

Bibliography

1. Quinn Jr FB. "Ludwig angina". *Archives of Otolaryngology-Head & Neck Surgery* 125 (1999): 599.

2. Pak S., *et al*. "Ludwig's angina". *Cureus* 9:8 (2017): e1588.

3. An J., *et al*. "Ludwig angina". [Updated 2021 Oct 18]. In: Stat Pearls. Treasure Island (FLo: Stat Pearls Publishing: (2022).

4. Shockley WW. "Ludwig angina: a review of current airway management". *Archives of Otorhinolaryngology-Head and Neck Surgery* 125.5 (1999): 200.

5. Hassan W., *et al*. "Ludwig's angina – A controversial surgical emergency: How we do it". *International Journal of Otolaryngology* (2011).

6. Caplan RA., *et al*. "An updated report by the American society of anaesthesiologists task force on management of the difficult airway". *Anesthesiology* 98.5 (2003): 1269-1277.

7. Marple BF. "Ludwig's angina: A review of current airway management". *Archives of Otorhinolaryngology-Head and Neck Surgery* 125 (1999): 569-569.

8. Busch RF and Shah D. "Ludwig's angina: Improved treatment". *Otolaryngology–Head and Neck Surgery* 117 (1997): 5172-5175.

9. Wouter A., *et al.* "Post extubation laryngeal edema and stridor resulting in respiratory failure in critically ill adult patients: updated review". *Critical Care* 19: 1 (2015): 295.

10. Argalious MY. "The cuff leak test: does it "leak" any information?" *Respiratory Care* 57:12 (2012): 2136-2137.

11. Kuriyama A., *et al*. "Performance of the cuff leak test in adults in predicting post-extubation airway complications: a systematic review and meta-analysis". *Critical Care* 24 (2020): 640.

12. AK AK and Cascella M. "Post intubation laryngeal examination". [Updated 2022 Feb 5]. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing (2022).

13. Girard TD., *et al*. "An official American thoracic society/ American college of chest physicians clinical practice guideline: Liberation from mechanical ventilation in critically ill adults. Rehabilitation protocols, ventilation liberation protocols, and cuff leak tests". *American Journal of Respiratory and Critical Care Medicine* 195.1 (2017): 120-133.

14. Freund B and Timon C. "Ludwig's angina: a place for steroid therapy in its management?" *Oral Health* 82.5 (1992): 23-25.

15. Saifeldeen K and Evans R. "Ludwig's angina". *Emergency Medical Journal* 21.2 (2004): 242-243.

16. Greenberg SL., *et al.* "Surgical management of Ludwig's angina". *ANZ Journal of Surgery* 77.7 (2007): 540-543.

17. Ha Y., *et al*. "Glucocorticoid-induced diabetes mellitus in patients with systemic lupus erythematosus treated with high-dose glucocorticoid therapy". *Lupus* 20 (2011): 1027-1034.

18. van Raalte DH., *et al*. "Novel insights into glucocorticoid-mediated diabetogenic effects: towards expansion of therapeutic options?" *European Journal of Clinical Investigation* 39 (2009): 81-93.

19. Tamez-Perez HE., *et al.* "Steroid hyperglycemia: Prevalence, early detection and therapeutic recommendations: A narrative review". *World Journal of Diabetes* 6:8 (2015): 1073-1081.

20. Wittekamp BH., *et al*. "Clinical review: post-extubation laryngeal edema and extubation failure in critically ill adult patients". *Critical Care* 13:6 (2009): 233.

21. Bjornson C., *et al.* "Nebulized epinephrine for croup in children". *Cochrane Database System Review* (2013): 10.

22. MacDonnell SP., *et al*. "Adrenaline administered via a nebulizer in adult patients with upper airway obstruction". *Anaesthesia* 50 (1995): 35-36.

23. Larawin V., *et al*. "Head and neck space infections". *Otolaryngology-Head and Neck Surgery* 135.6 (2006): 889-893.

24. Kurien M., *et al*. "Ludwig's angina". *Clinical Otolaryngology and Allied Sciences* 22.3 (1997).

25. Greenberg SL., *et al*. "Surgical management of Ludwig's angina". *ANZ Journal of Surgery* 77.7 (2007): 540-543.

26. Bansal A., *et al*. "Otolaryngologic critical care". *Critical Care Clinic* 19:1 (2003): 55-72.