Ludwig's Angina in Post Partum Patient

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Abstract Ludwig's Angina is a rapid progressive cellulitis of the submandibular spaces, with potential for significant upper airway obstruction. Most reported cases follow an odontogenic infection. We present a case of 22-year-old female patient in her 2 weeks post partum period developing a life threatening infection. Immediate intubation, surgical decompression and antibiotic therapy successfully resolved the episode.

Keywords Ludwigs Angina · Post partum period · Odontogenic infection

Introduction

Ludwig's Angina is a rapidly spreading cellulitis involving the submandibular, sublingual and submental spaces. First described by Wilhelm Fredrick Von Ludwig in 1836, then by Camerer in 1837. Angina means choking in greek.

Here we report a case of Ludwig's Angina in a patient in her second week of post partum period.

Case Report

A 22-year-old female patient presented to the department of OMFS with a history of fever and malaise accompanied

by pain and swelling in her left submandibular area, which rapidly progressed to the sub mental region and to the right submandibular region.

The patient was in second week of her post partum period (lactating mother). There was previous history of pain and swelling in her left lower last molar tooth. For which she took medication and deferred extraction.

On examination, the patient had a temperature of 38.5°C pulse of 110 per minute and respiratory rate of 30 per minute. Clinical examination revealed indurated, tender erythematous swelling extensively involving the left submandibular space extending inferiorly to the level of the hyoid bone. The swelling crossed the midline extending to the other side of the neck. The patient had halitosis, trismus, dysphagia and respiratory distress (Figs. 1, 2).

Orthopantomograph (OPG) showed carious 38. Routine blood investigations (Hb% = 11 g per dl) TLC, DLC, CT. BT, HBsAg, HIV were done Neutrophil count increased to 16,000 cells. Anteroposterior view of neck was taken. The patient was admitted and started on a regimen of empirical antibiotics consisting of ampicillin 1 g, clindamycin 600 mg, gentamycin 80 mg, iv, voveran 3 ml, IM.

She underwent awake fiber optic intubation, then proceeded to surgical drainage with a deep incision in the submandibular triangle through the mylohyoid muscle into the sublingual space. Similarly on the right side submandibular space, a separate incision for submental space was done. Pterygomandibular space was also drained on the left side through the same E.O. incision. About 50 ml of thick pus was drained from the left submandibular space. Corrugated rubber drains were inserted and fixed. The carious lower molar 38 was extracted (Figs. 3, 4, 5).

The endotracheal tube was kept for 3 days postoperatively and subsequently extubated. There was no significant

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Fig. 1 Pre operative front view of the patient

Fig. 4 Post operative picture of the patient

Fig. 2 Intubation done before the drainage

Fig. 5 Post operative OPG

Fig. 3 Corrugated rubber drains inserted and fixed

perioperative complications. The patient made uneventful post operative recovery, during which time the infection completely resolved. The patient was discharged on 5th postoperative day, and continued on oral antibiotics for another 14 days.

Discussion

Ludwig's Angina most commonly originates from an odontogenic infection. Classically located in the second and third molar teeth. The apices of these teeth in particular are located just below the mylohyoid ridges, and are therefore in close anatomic proximity to the submandibular space [1]. At this time, the infection may develop and progress at such an alarming rate that special precautions regarding air-way maintenance must be taken. Because the

mandible, hyoid bone, and superficial layer of the deep cervical fascia, limit tissue expansion associated with the developing edema. The floor of the mouth and the tongue base will become displaced superiorly and posteriorly, resulting in severe airway compromise [2]. Further, extension of the infection may spread into the mediastinum and the carotid sheath resulting in severe thoracic infection [3, 4]. Rupture of abscesses may cause aspiration of pus into the lungs and/or even pericarditis. Untreated, the mortality is close to 100%, both from the acute sepsis and from airway obstruction. The patient with Ludwig's Angina, will have severe and obvious extraoral swellings, including bilateral submandibular, submental, and sublingual spaces. Common presentation is elevation and displacement of the tongue, trismus, drooling of saliva, airway obstruction, dysphagia and/or dyspnea, and a hoarse ("hot potato") voice. With extensive use of antibiotics [5], most facial infections improve before they have a chance to progress to Ludwig's Angina. The mortality rate from Ludwig's Angina, when recognized, has decreased from 50 to 5%. Therapy also includes early surgical removal of the source of infection (which is often grossly carious dentition) via extraction, aggressive, and vigorous incision and drainage procedures with appropriate placement of drains, along with intense and prolonged antibiotic therapy and maintenance of a patent airway [6, 7]. While penicillin administered intravenously and in high doses is the empirical antibiotic of choice, it is often recommended to use metronidazole as well. For patients who have had repeated episodes of dental infections, clindamycin is often the antibiotic of choice.

The majority of cases occur in previously healthy individuals. However, certain underlying conditions may predispose patients to severe periodontal infection, including diabetes-mellitus, neutropenia, aplastic anemia, and glomerulonephritis. Other authors have documented an increased susceptibility to Ludwig's angina, in the setting of immuno deficiency.

Pregnancy is accompanied by many physiological changes which place the mother at a higher risk of infection, or of doing worse, once infected [8]. First, the immune response is greatly diminished during pregnancy and immediate post partum period, thus resulting in potential faster progression of an infection. In addition, there is decreased neutrophil chemotaxis, cell mediated immunity, and natural killer cell activity [9, 10]. From an oral perspective, as pregnancy associated hormonal changes begin to affect a woman's body, the gingival tissues are affected as well. They become much more sensitive and thus susceptible to irritation from soft plaque. The plaque accumulates, becomes hard calculus deposits on the teeth, starting low-grade intraoral infection [11, 12]. Maternal infective processes sustained especially by gram negative

anaerobic bacteria, such as those leading to Ludwig's Angina, have been demonstrated to cause physiologic imbalance through inflammatory cytokine production. Even a low virulent infection may progress and spread rapidly to a fulminating and fatal condition. Ludwig's Angina is life threatening because of septicemia and asphyxia [13–15].

Conclusion

In order to prevent a similar life-threatening emergency, health care providers should not neglect even minimal complaints of dental pain. Often, if a problem is identified during the early stages of pregnancy, routine dental care can be planned to control active disease or eliminate potential problems that could increase in severity later in the pregnancy. Antibiotics that are acceptable include penicillin, amoxicillin, and clindamycin.

References

1. Topazian RG, Goldberg MH, Hupp JR (2002) Oral and maxillofacial infections, 4th edn. WB Saunders Co., Philadelphia
2. Shockley D, Williams W (1999) Ludwig's Angina: a review of current airway management. *Arch Otolaryngol Head Neck Surg* 125(5):600–604
3. Bounds GA (1985) Subphrenic and mediastinal abscess formation: a complication of Ludwig's Angina. *Br J Oral Maxillofac Surg* 5:313
4. Strauss HR, Tilghman DM, Hankins J, Angina Ludwigs (1980) Empyema, pulmonary infiltration and pericarditis secondary to extraction of a tooth. *J Oral Surg* 38:223–229
5. Gilbert D et al (1999) The Stanford guide to antimicrobial therapy, 29th edn. Lippincott Williams and Wilkins, Philadelphia
6. Schwartz HC, Baver RA, Davis NJ (1974) Ludwigs Angina: use of fibre optic laryngoscope to avoid tracheostomy. *J Oral Surg* 32:608
7. Bross-Soriano D, Arieta Gomez JR (2004) Management of Ludwig's Angina with small neck incision—18 years experience. *Otolaryngol Head Neck Surg* 130:712–717
8. Katz VC (1991) Physiologic changes during normal pregnancy. *Curr Opin Obstet Gynecol* 3:750
9. Feinberg BB, Gouik B (1991) General precepts of the immunology of pregnancy. *Clin obstet Gynaecol* 34:3–16
10. Mullin PM, Morrison JC (2006) Ludwig's angina in pregnancy. *J Matern-Fetal Neonatal Med* 19(2):119–120
11. Bhaskar Rao C, Gopalkrishna K (2005) Management of oral and maxillofacial infections during pregnancy. *J Maxillofac Oral Surg* 4:145
12. Lawrenz DR, Whitley BD, Helfrick JF (1996) Considerations on the management of maxillofacial infections in the pregnant patient. *J Oral Maxillofac Surg* 54:474
13. Juener M, Aziz SR (2002) Management of the pregnant oral and maxillofacial surgery patient. *J Oral Maxillofac Surg* 60:1479
14. Dashi JS, Gilstrap LC (1997) Antibiotics used in pregnancy. *Obstet Gynecol Clin North Am* 24:617
15. Abramowicz S, Abbrmowicz JS, Dolwick MF (2006) Severe life threatening maxillofacial infection in pregnancy presented as Ludwig's Angina. *Infectious Dis Obstet Gynecol* 2006:1–4