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Negative Pressure Wound Therapy for Ludwig's Angina

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INTRODUCTION

Although Wilhelm Friedrich von Ludwig who was a court physician to the king of Württemberg described the now well-known condition of Ludwig's angina in 1836, it was not until the early twentieth century that the correlation between dental (odontogenic) infections and life-threatening manifestations of the head and neck region were established [1-6]. The incidence, severity, morbidity, and mortality of odontogenic-related head and neck infections have declined significantly over the past 60 years, largely impart to the principles of treatment substantiated by Dr. Ashbel Williams and Dr. Walter Guralnick, most notably consisting of early establishment of a secure airway in addition to early and aggressive surgical intervention [4-10]. Appropriate management of infections of the head and neck, particularly Ludwig's angina, require fundamental knowledge of complex head and neck anatomy, ultimately allowing for an understanding of the potential deep spaces created by the fascial planes of the head and neck through which infections can progress [7].

Achieving source control is the most important step in the management of odontogenic infections of the head and neck and is comprised of incision and drainage of the involved anatomic spaces with blunt dissection, typically with placement of drains and extraction of any necessary teeth serving as the etiologic culprit of the infection. Obtaining culture and sensitivity testing at the time of incision and drainage can help guide antibiotic therapy, particularly in those with more severe infections, other co-morbidities, and who have already been treated with multiple courses of antibiotics. Once the source of the infection has been eliminated and the bacterial load is reduced, it is important to medically optimize the patient through supportive measures (hydration, nutrition, pain control, blood glucose control, and antibiotic therapy) to enhance the patient's ability to clear any residual infection [8]. The choice of antibiotic is governed by the eight principles of modern antibiotic therapy, which is important to be aware of but beyond the scope of this discussion [4]. The final step with respect to the principles in management of odontogenic infections is frequent re-evaluation of the patient. For odontogenic infections involving deep fascial spaces requiring hospitalization, clinical signs of improvement should be evident 2-3 days post-operatively, including decreased swelling and drainage,

defervescence, down-trending white blood cell count, and decreased swelling of the airway allowing for thought to be given towards possible extubation [4].

However, there are times when the severity of the disease exceeds or necessitates multiple interventions, including medical and surgical. Negative pressure wound therapy is one such technique that has been more recently employed in the management of complex wounds through the creation of a negative pressure environment. It is an underutilized resource in management of complex, odontogenic-related head and neck infections and can reduce the number of operative interventions and create an opportunity for bedside management of complex neck wounds. Furthermore, it accelerates healing and promotes healthy tissue growth obviating the need for more complex procedures.

The foundation of negative-pressure wound therapy (NPWT) which is also known as vacuum-assisted closure was first described by Dr. Louis Argenta and Dr. Michael Morykwas in 1997, as they described the findings in their animal studies and clinical experience [2,3]. Negative-pressure wound therapy involves the application of sub-atmospheric pressure (125 mmHg) to a wound by adapting a porous sponge within a wound that is covered by an airtight occlusive dressing extending approximately 5cm beyond the wound to which a vacuum is applied [5,9]. This technique should be most practically thought of as an adjunct to eventual surgical closure by preparing the wound bed for closure by tertiary intention [9].

The basis of success of NPWT relies on the removal of excess interstitial fluid, improved vascularity, a decrease in bacterial colonization, and tissue response to mechanical forces [3]. Through these changes, an environment can be created that promotes wound healing and the formation of granulation tissue at an expedited rate. The collection of interstitial fluid (third-space fluid) causes mechanical compromise of the microvasculature and lymphatic system, resulting in increased capillary and venous afterload, which ultimately leads to decreased oxygen and nutrient delivery [3]. Interstitial fluid is also known to contain higher levels of inhibitory factors and matrix metalloproteinases (collagenases and elastases) that are detrimental to healing [3]. Through the removal of interstitial fluid with NPWT, small blood vessels are decompressed and capillary afterload is therefore decreased, resulting in improved blood flow and subsequent oxygen and nutrient delivery to tissues [2,3]. Increased oxygen delivery is inversely-related to bacterial colonization, as higher levels of oxygen hinder the growth of anaerobic organisms and adequately supply neutrophils with oxygen to be used for eliminating bacteria through oxidative burst [2].

It important to remember that wound healing is a complex interdependent process between cells, the surrounding microenvironment, biochemical mediators, and extracellular matrix molecules [2]. By modulating the aforementioned factors involved in wound healing, NPWT can promote and expedite wound healing. In this report, we detail the many benefits and clinical evidence of negative pressure wound therapy as an intervention for complex head and neck infections.

CASE REPORT

A 20-year-old male smoker sustained blunt trauma to the chin on when he was struck by a piece of lumber at work. Over the following days he developed progressive right greater than left submandibular and cervical swelling with associated pain.

He initially presented outside of our institution two days later, where CT imaging of the neck demonstrated diffuse soft tissue swelling concerning for cellulitis or phlegmon. He was treated with intravenous dexamethasone and ampicillin-sulbactam with metronidazole, but he left against medical advice the next morning.

On July 11, he presented to a neighboring hospital in Pennsylvania with continued worsening swelling. A contrast-enhanced CT neck revealed extensive bilateral cervical soft tissue fluid versus infection and periapical lucencies of the bilateral mandibular molars concerning for odontogenic abscesses.

He was started on IV dexamethasone and cefazolin, then transferred to Penn Presbyterian Medical Center (PPMC) for a higher level of care that evening due to concern for evolving Ludwig's angina. Upon arrival to PPMC, ENT was immediately consulted and performed a flexible nasopharyngolaryngoscopy, which demonstrated marked adenoid hypertrophy without complete nasopharyngeal obstruction, as well as moderate right greater than left edema of the epiglottis, aryepiglottic folds, and arytenoids. No elective intubation or immediate airway intervention was recommended, and medical management with IV steroids and Unasyn was continued. He was admitted to the MICU for airway monitoring and with a plan to re-scope in 6 hours.

Four days after the inciting event, OMFS was consulted by the MICU for potential odontogenic source control. Upon evaluate by the OMFS team, the patient was afebrile and hemodynamically stable with a WBC of 21.2. His exam was notable for voice changes consistent with "hot potato voice" and moderate, warm, firm and fluctuant swelling of the right mid- and lower face extending to the submental region and mild swelling of the left submandibular region and trismic to 1.5 finger breadths. He had brawny erythema and edema of the neck with severe neck pain and immobility. (Figure 1).

Figure 1: Patient presentation on arrival.

Interval CT neck with contrast was obtained which showed:

"Extensive edema and subcutaneous stranding along the right side of the face extending into the neck, from the right buccal and parotid spaces, tracking inferiorly into the submandibular, submental, and carotid spaces. It also extends into the right parapharyngeal and retropharyngeal space, and there is substantial edema of the epiglottis and extensive edema along the right aryepiglottic fold with effacement of the right piriform recess. Large extent of mass effect and substantial narrowing of the laryngeal airway. The edema also extends across the midline into the left retropharyngeal space."

There was also substantial edema of the epiglottis and right aryepiglottic fold with mass effect and narrowing of the laryngeal airway. (Figure 2).

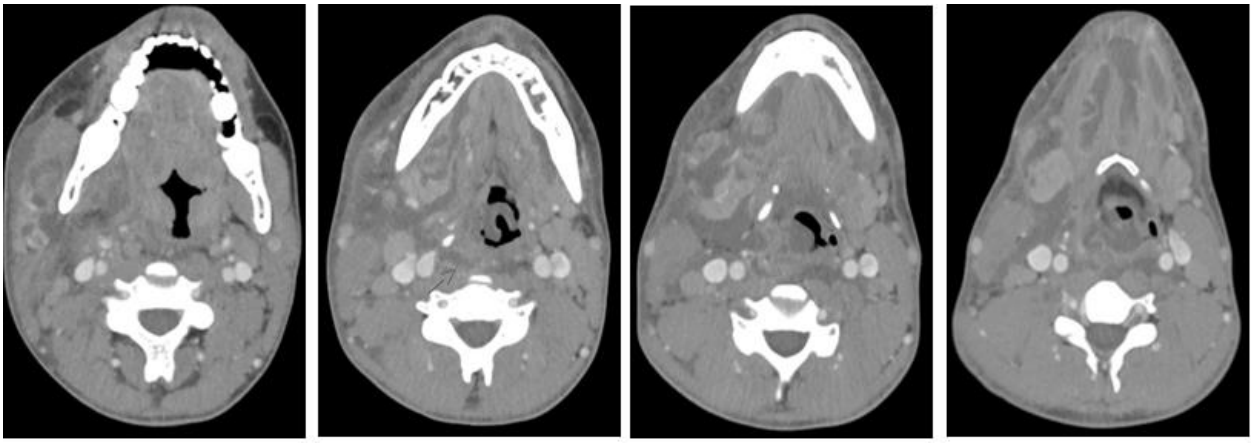

Figure 2: CT scan of the neck with intravenous contrast.

The patient was taken to the operating room for urgent incision and drainage. Upon opening the submental and bilateral submandibular spaces, purulent exudate exuded from the spaces. It then became clear that it was necessary to perform incision and drainage of the neck. Additionally, the masticator and pharyngeal spaces were opened and bluntly instrumented where purulence once again exuded.

The oral cavity was examined and teeth #31 and 32 were found to be grossly carious and determined to be the likely source of infection. Teeth #31 and 32 were extracted and adjacent soft tissue and bone involved were removed.

Given the severity and extent of the infection, the decision was made to leave the neck open as there was concern that closing it would allow for progression of the infection and therefore negative pressure wound therapy was instituted

Penrose drains were placed from the neck incision through various involved anatomic spaces including the right lateral pharyngeal space, right submandibular space, and submental space through the left submandibular space to provide dependent drainage (Figure 3).

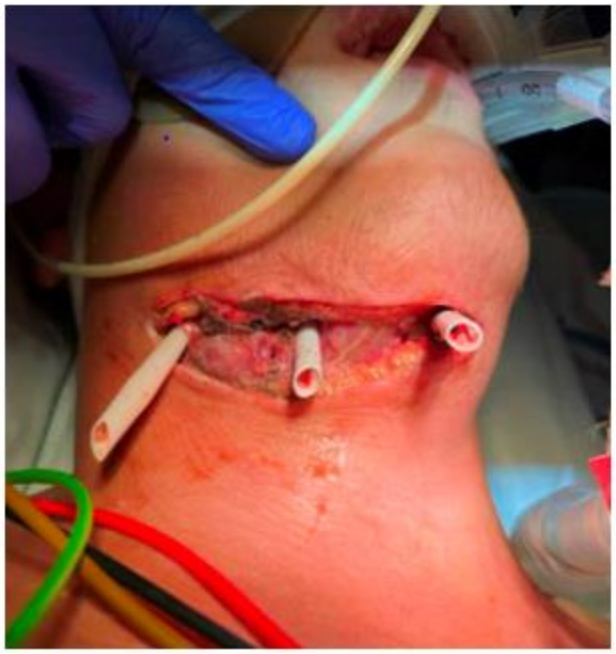

Figure 3: Placement of Penrose drains.

These drains were left in place to stent open the involved anatomic spaces and to prevent the spread of the infection. The drains were sutured to the deep aspect of a VAC sponge, which was then fit to the neck. The sponge was then sealed in standard fashion and 125 mmHg of negative pressure was generated as the tubing was hooked up to the impact device without a leak (Figure 4).

Figure 4: Application of NPWT.

He was transferred postoperatively to the ICU, where he remained sedated and intubated for airway protection with a dobhoff tube for enteral nutrition. The patient was maintained on negative pressure wound therapy for 72 hours. Postoperative CT neck at 24 hours post-op revealed interval decrease and improvement in all Spaces. CT chest showed no mediastinitis. Therefore the wound VAC was left in place and removed in the operating room at 72 hours post initial surgery. The sites were irrigated with copious amounts of normal saline solution. At the time of wound VAC change, there was significant improvement in the overall quality and appearance of the underlying tissue which now appeared as more red-exuberant healthy tissue, as opposed to prior necrotic, sloughing tissue (Figure 5).

Figure 5: Tissue appearance on VAC change.

Negative pressure therapy was then reinstituted as a new VAC was placed with a slightly smaller sponge and similar Penrose drains were placed into the involved anatomic spaces and sutured to the deep aspect of a fitted VAC sponge. After an additional 72 hours (day 6 from initial surgery) the VAC was changed at bedside.

Unfortunately on hospital day 7-8, the patient reported malaise and feeling unwell. Repeat imaging at this time showed a collection in the central compartment of the neck adjacent to the right thyroid gland, extending to the sternal notch. He was then taken to the operating room for a final time, 8 days after his initial surgeyr and a lower neck incision made with return of purulent, sanguinous discharge from the lower anterior neck and sternal notch region. At this time, penrose drains were left and VAC removed given overall progress. The wounds were closed Around the penroses which were removed within 72 hours and the patietn discharged on hospital day 14 with prolene sutures in

place and wound closed. (Figure 6). He returned for follow up and had dobhoff tube removed and was improving functionally.

Figure 6: Closure of wound at VAC site.

To conclude, Ludwig's angina is a surgical emergency, and these infections often require multiple procedures for aggressive source control. Negative pressure wound therapy is an adjunct to surgical treatment of this disease, optimizing and accelerating wound healing by creating an environment supportive of new tissue growth. It also offers opportunity for bedside wash out and exploration at the time of wound VAC change. By decreasing healing time and the number of trips to the operating room, it remains a critical adjunctive therapy in the management of complex infections, such as Ludwig's angina.

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