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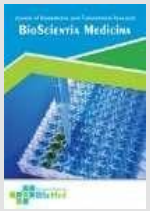

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Navigating a Life-Threatening Airway Emergency: Clinical Diagnosis and Surgical Management of Ludwig's Angina in a Resource-Limited Setting

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A B S T R A C T

Background: Odontogenic abscesses, common in clinical practice, possess the potential to escalate into life-threatening emergencies. The contiguous spread of polymicrobial infections into the deep fascial spaces of the neck can precipitate Ludwig's angina, a rapidly progressive cellulitis of the floor of the mouth characterized by acute airway compromise. This report details the diagnosis and management of such a case, emphasizing the critical role of clinical acumen when gold-standard diagnostic modalities are unavailable. **Case presentation:** A 43-year-old male presented with a one-week history of progressive, painful swelling of his right mandible, accompanied by fever and trismus. The working diagnosis was Ludwig's angina secondary to an odontogenic abscess originating from the mandibular right second molar (tooth #47). Despite an initial radiological report erroneously noting an issue with tooth #37, all clinical evidence pointed to a right-sided pathology. Diagnosis was established through physical examination and basic radiography, which confirmed significant soft tissue swelling. Management involved immediate surgical incision and drainage under general anesthesia, aggressive intravenous antibiotic therapy, and meticulous postoperative care. The causative tooth was subsequently extracted. The patient experienced a complete resolution of symptoms and a full functional recovery. **Conclusion:** Ludwig's angina remains a formidable surgical emergency. This case underscores that even with limited diagnostic resources, a decisive diagnosis based on strong clinical findings, followed by prompt and aggressive surgical decompression, is paramount to preventing mortality and ensuring a favorable patient outcome.

1. Introduction

An odontogenic abscess, defined as a localized accumulation of pus within the alveolar bone at a tooth's apex, represents a frequent endpoint of untreated dental disease.[1] While often perceived as a routine clinical entity, its potential for catastrophic complications cannot be overstated. The condition typically arises from dental caries, pulp necrosis, dental trauma, or the failure of endodontic therapy.[2] The oral cavity harbors a complex and diverse microbiota. Under pathological conditions, these microorganisms, particularly anaerobic and facultative anaerobic bacteria, colonize the root canal system, forming resilient biofilms. The egress of these pathogens and their toxins from the apical foramen into the periapical tissues initiates a potent acute inflammatory response, culminating in suppuration and abscess formation.[3] The true danger of an

odontogenic abscess lies in its capacity to spread beyond the confines of the alveolar bone. The mortality risk is directly linked to the potential for this purulent collection to dissect along the path of least resistance—the contiguous fascial planes of the head and neck—leading to airway obstruction.[4] While initial symptoms are typically localized, such as pain and swelling adjacent to the offending tooth, dissemination of the infection into surrounding soft tissues marks a critical turning point towards a life-threatening condition. The clinical presentation may then evolve to include systemic signs such as fever, malaise, and more ominous local signs like diffuse extra-oral swelling, severe trismus (lockjaw), dysphagia, and odynophagia.[5]

This aggressive, rapidly spreading cellulitis involving the bilateral submandibular, sublingual, and submental spaces is known as Ludwig's angina.[6] This condition constitutes a profound clinical emergency, where mortality is primarily due to asphyxiation from the expanding edema of the neck and superior displacement of the tongue. Management at this advanced stage is non-negotiable and twofold: aggressive surgical intervention to decompress the involved spaces and drain the abscess, coupled with high-dose, broad-spectrum intravenous antibiotic therapy to combat the typically polymicrobial nature of the infection.[7] This paper presents a detailed case report of a patient who developed Ludwig's angina secondary to an abscessed mandibular molar. The case is particularly instructive as it highlights the diagnostic and therapeutic pathway in a setting with limited access to advanced imaging, underscoring the indispensable value of thorough clinical evaluation and decisive surgical action.[8] We will provide a comprehensive analysis of the patient's presentation, the diagnostic reasoning, the specifics of the surgical and pharmacological management, and the final, successful outcome, framed within the context of contemporary evidence and best practices.

The novelty of this study lies in its detailed exposition of managing a life-threatening airway emergency based almost exclusively on clinical findings and basic radiographic support, a scenario commonly faced in resource-limited environments worldwide. While the principles of treating Ludwig's angina are well-established, this report provides a granular, real-world account of the diagnostic reasoning and therapeutic sequencing that can lead to a successful outcome when advanced technologies like computed tomography are not available.[9,10] The primary aim of this case report is therefore to reinforce the paramount importance of clinical acumen in the early diagnosis of Ludwig's angina and to illustrate the effectiveness of an aggressive, surgically-led management strategy in preventing mortality, thereby providing a valuable and practical guide for clinicians in similar settings.

2. Case Presentation

A 43-year-old male, otherwise healthy, presented to the emergency department of Bhayangkara Hospital as a man in profound distress. His journey to the hospital was the culmination of a harrowing week, during which a seemingly manageable toothache had exploded into a debilitating and frightening condition. His chief complaint was of a severe, unremitting, and throbbing pain coupled with a massive swelling of his right lower jaw. The patient recounted how the swelling had begun insidiously but had then rapidly expanded over the past several days, becoming progressively more tense and painful. This localized crisis was accompanied by systemic signs of infection; he reported feeling feverish and suffering from a deep sense of malaise that had left him weak and exhausted. The functional impairments were severe. The patient was experiencing profound difficulty in opening his mouth, a classic sign known as trismus, which he demonstrated with a pained effort, achieving an interincisal opening of less than a finger's width. This, combined with the intense pain, had led to severe dysphagia (difficulty swallowing) and odynophagia (painful swallowing), rendering him unable to consume any solid food or even drink liquids without significant suffering. His medical history was thoroughly reviewed and found to be non-

contributory; he had no known history of systemic conditions like hypertension or diabetes mellitus that might have predisposed him to such a severe infection or complicated its management. The patient's initial presentation details are summarized in Figure 1.

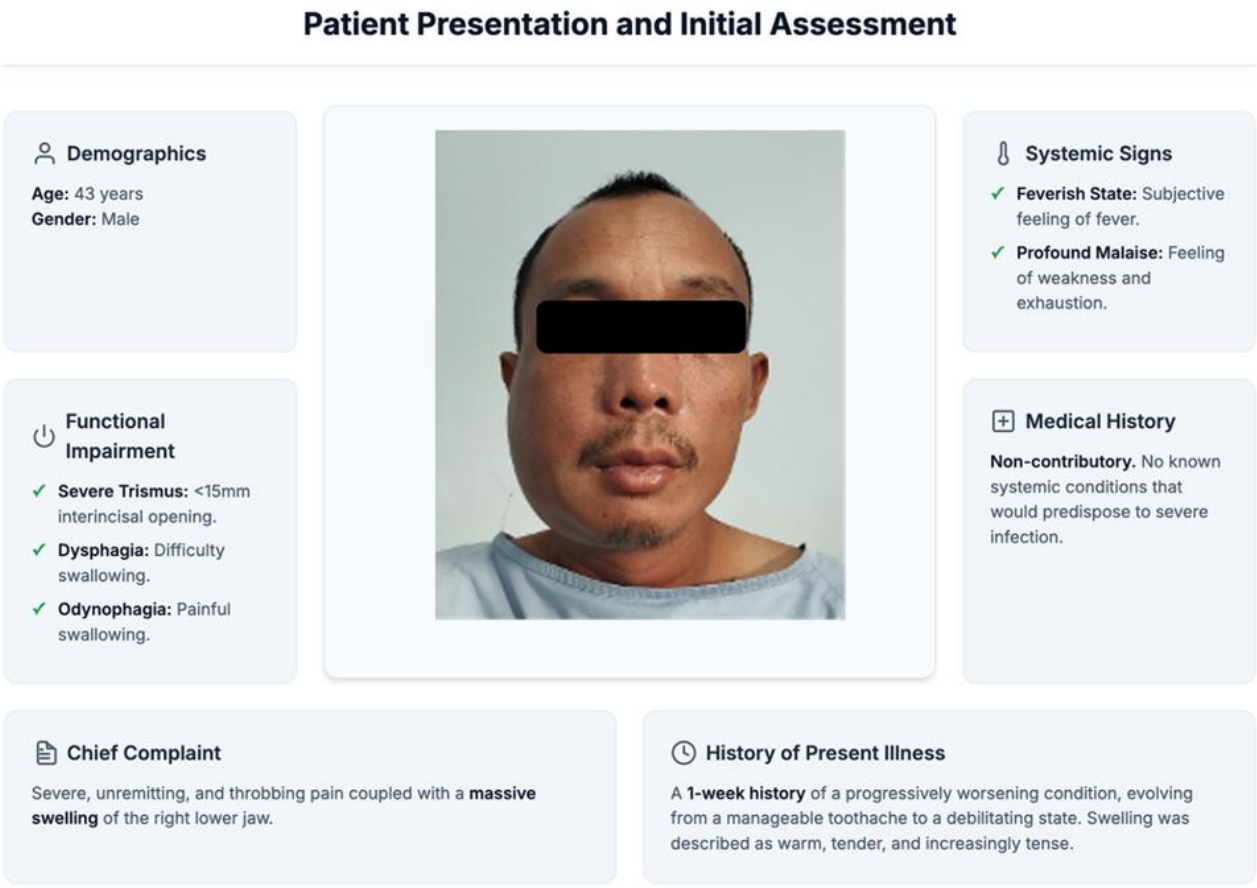

Figure 1. Patient presentation and initial assessment.

The gravity of the patient's condition was immediately apparent upon physical examination. He was visibly anxious and in significant pain, guarding the right side of his neck. His vital signs were stable, but he was febrile with a core body temperature of 38°C. The extra-oral examination revealed a dramatic and alarming pathology. A massive, diffuse, and profoundly indurated swelling engulfed the right submandibular and submental regions. The swelling was so extensive that it obliterated the normal contour of the jawline and neck, extending from the inferior border of the mandible down towards the hyoid bone and crossing the midline. The overlying skin was taut, erythematous, and distinctly warm to the touch. Palpation confirmed the clinical suspicion of Ludwig's angina; the tissues had a characteristic "woody" or brawny texture, being firm, non-pitting, and non-fluctuant, a sign of diffuse cellulitis rather than a discrete, liquid abscess. This area was exquisitely tender to even the lightest touch. Further examination revealed palpable, tender cervical lymphadenopathy on the right side. The intraoral examination was severely hampered by the trismus. With careful manipulation, it was possible to visualize parts of the oral cavity. The floor of the mouth on the right side was visibly elevated, pushing the tongue upwards and towards the left. The mucosa was erythematous and tense. There was no purulent discharge visible from

the gingival sulci or the orifice of Wharton's duct. An urgent Orthopantomogram (OPG) was procured. The initial radiological report noted a vertical radiolucent line on tooth #37 (mandibular left second molar). This finding was in stark contradiction to the entirely right-sided clinical presentation. Through careful clinical correlation, this was determined to be a reporting error, and the clinical team correctly focused on the right mandible as the nidus of infection. The OPG did reveal a grossly decayed mandibular right second molar (tooth #47) with associated periapical changes, confirming the odontogenic source. To assess for potential life-threatening complications, such as mediastinal extension or significant airway compromise, plain radiographs of the chest and neck were performed. The posteroanterior chest X-ray was clear, showing no signs of mediastinitis. The anteroposterior and lateral cervical radiographs were critically important. They confirmed the presence of marked soft tissue swelling in the right submandibular region, but, reassuringly, showed that the tracheal air column, while slightly narrowed, remained patent without gross deviation. This finding, while not as precise as a CT scan, provided a crucial piece of information that a complete airway collapse was not imminent, allowing for a controlled, albeit urgent, surgical plan. Laboratory investigations provided objective evidence of the severe systemic infection. The complete blood count was significant for a pronounced leukocytosis, with a white blood cell count of 22,000/µL (normal range: 4,500-11,000/µL). A mild normocytic anemia was also noted (hemoglobin: 12.9 g/dL), consistent with anemia of chronic inflammation from the week-long infection. Coagulation studies were within normal limits. These key findings are detailed in Figure 2.

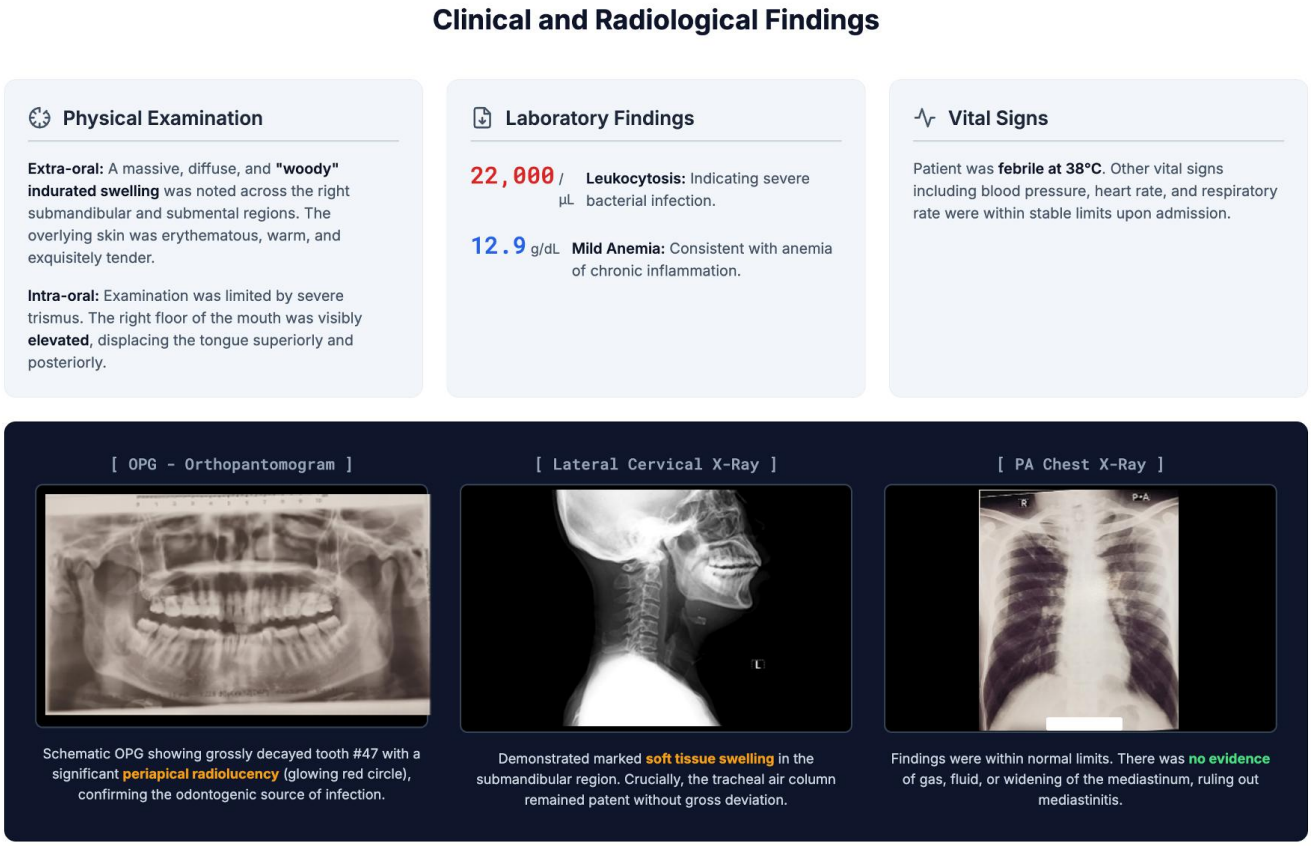

Figure 2. Clinical and radiological findings.

The constellation of findings—the history of a dental problem, the classic "woody" induration involving bilateral floor-of-the-mouth spaces, the superior displacement of the tongue, and the systemic signs of sepsis—led to a confident and definitive diagnosis of Ludwig's angina secondary to an odontogenic abscess originating from the mandibular right second molar (tooth #47). Recognizing this as a surgical emergency with high mortality risk, the patient was immediately admitted, and a multi-pronged treatment plan was enacted with urgency. The cornerstone of this plan was emergency surgical decompression of the involved fascial spaces under general anesthesia. The comprehensive treatment protocol is outlined in Figure 3. Intravenous access was established, and fluid resuscitation was initiated with 0.9% Sodium Chloride to correct dehydration and stabilize the patient. Pre-emptive intravenous analgesia was administered. The patient was taken to the operating theater on an emergency basis. Given the potential for a difficult airway, the anesthetic team was prepared for advanced airway management techniques. Following a safe induction of general anesthesia, the surgical procedure commenced. An extensive incision and drainage were performed. A wide transverse incision was made in a natural skin crease in the right submandibular region, providing broad access to the underlying fascial spaces. Upon entering the submandibular space, a copious amount of foul-smelling, serosanguinous, purulent fluid was evacuated. Using blunt finger and instrument dissection, the surgeon meticulously explored and decompressed all involved spaces—the submandibular, the sublingual (accessed by dissecting superior to the mylohyoid muscle), and the submental spaces—breaking up any loculations to ensure no pocket of pus was left behind. The spaces were thoroughly irrigated with sterile saline. Finally, a corrugated rubber drain was placed deep within the wound and sutured to the skin to facilitate continued drainage and prevent premature wound closure.

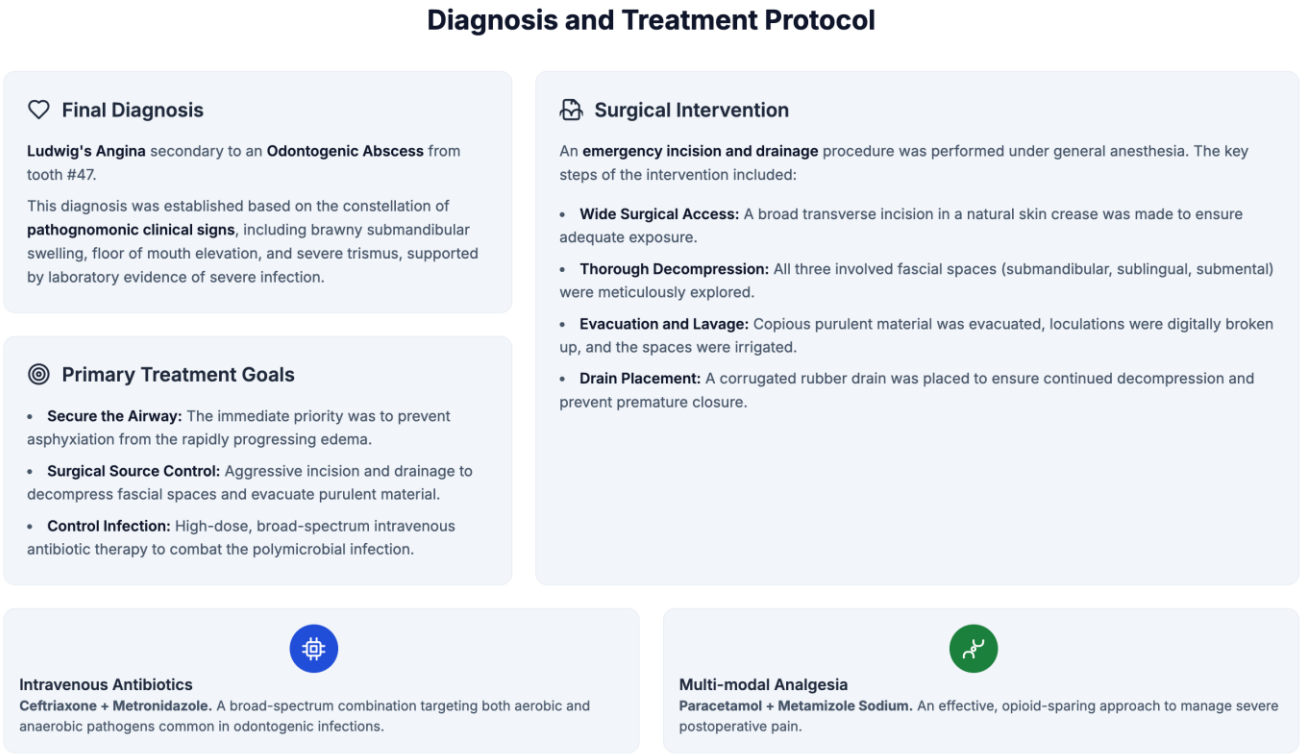

Figure 3. Diagnosis and treatment protocol.

The patient's response to the aggressive surgical and medical intervention was rapid and dramatic. He was hospitalized for a total of three days, during which he was closely monitored. Within the first 24 hours post-surgery, his fever resolved, and there was a palpable softening and reduction in the extensive neck swelling. His leukocytosis began a steady downward trend. Although his overall condition improved significantly, he continued to experience a persistent, localized pain centered on his lower right molar, confirming that the source of the infection, while now contained, still required definitive treatment. He was discharged on the third postoperative day with a course of oral antibiotics and analgesics and clear instructions for wound care. He was scheduled for a follow-up review one week later. At this first follow-up appointment, the surgical drain was removed, and the incision site was found to be clean and healing well. The trismus had significantly improved, allowing for a thorough intraoral examination. This confirmed that the crown of the mandibular right second molar (tooth #47) was grossly destructed by caries and fractured, rendering it non-restorable. It was unequivocally identified as the source of the initial infection and was deemed to have a hopeless prognosis. After a detailed discussion and obtaining renewed consent, the patient underwent the definitive treatment phase. The remnants of tooth #47 were surgically extracted under local anesthesia. The alveolar socket was meticulously debrided of all granulation tissue and irrigated, and primary closure was achieved with sutures. A short, prophylactic course of oral antibiotics was prescribed to prevent a secondary socket infection. The final phase of his recovery is detailed in figure 4. The patient returned one month later for suture removal. At this visit, he was a transformed man. He reported a complete and total resolution of all his symptoms. The nagging, localized pain he had experienced had vanished following the extraction. He had no residual swelling or discomfort. His trismus was completely resolved, with his interincisal opening returning to a normal range of over 40 mm. He had resumed a normal diet and had returned to his work and daily activities without any limitations. The surgical scar was healing beautifully, and the patient expressed immense gratitude and satisfaction with the final outcome.

Figure 4. Patient outcome and follow-up timeline.

3. Discussion

The case presented is a powerful narrative of a journey from a common dental affliction to the brink of a fatal airway catastrophe. The successful management of this patient with Ludwig's angina provides a platform to explore the intricate interplay of anatomy, microbiology, and clinical decision-making that defines this formidable condition. Ludwig's angina is, at its core, a disease of anatomical architecture.[11] It is not a random or chaotic infection but one that follows predictable pathways dictated by the fascial planes of the neck. The origin of the infection in this case—the mandibular right second molar (tooth #47)—is the archetypal starting point for this devastating cascade. The key to understanding this progression lies in the relationship between the tooth's roots and a single, critical muscle: the mylohyoid.[12] This flat, diaphragm-like muscle forms the floor of the oral cavity proper. Its line of attachment to the inner surface of the mandible, the mylohyoid line, serves as a crucial watershed. The roots of the anterior mandibular teeth (incisors, canines, and premolars) typically terminate superior to this line. An abscess from one of these teeth will therefore tend to drain intraorally into the sublingual space or buccally into the vestibule.[12]

The story changes dramatically with the posterior molars. The apices of the second and, almost invariably, the third molars lie inferior to the mylohyoid line. When an abscess from tooth #47 perforates the thin lingual cortical plate of the mandible, as it did in our patient, the pus is discharged directly into the submandibular space.[13] This space is a well-defined compartment containing the submandibular salivary gland and lymph nodes, but it is not a closed box.[14] It communicates freely with adjacent spaces. The infection, driven by hydrostatic pressure and facilitated by bacterial enzymes, begins its relentless march. It travels posteriorly around the free edge of the mylohyoid muscle, hooking upwards to invade the sublingual space. Simultaneously, it crosses the midline anteriorly, deep to the digastric muscles, to involve the submental space. It is this specific, multi-space involvement—bilateral submandibular, bilateral sublingual, and submental—that constitutes the formal definition of Ludwig's angina. The clinical consequences are profound. The infiltration of these spaces with inflammatory exudate and pus leads to a board-like, brawny induration of the suprahyoid neck tissues.[15] The swelling of the sublingual spaces, which lie directly beneath the tongue, causes a dramatic superior and posterior displacement of the tongue itself. This is the crux of the life-threatening emergency. The tongue is pushed up against the hard palate and back towards the posterior pharyngeal wall, effectively acting as a plug that occludes the oropharyngeal airway. In our patient, the elevated floor of the mouth and early tongue displacement were clear harbingers of this impending disaster, mandating immediate intervention. The infection's potential does not end there; from the submandibular space, it can track inferiorly along the carotid sheath or into the retropharyngeal space, the "danger space" of the neck, providing a direct conduit to the mediastinum, leading to the highly lethal complication of descending necrotizing mediastinitis. The clear chest radiograph in our patient was a crucial, albeit basic, check to rule out this devastating extension.[16]

The engine driving this rapid anatomical invasion is a synergistic conspiracy of microorganisms. Odontogenic infections are not caused by a single pathogen but are quintessentially polymicrobial. The oral cavity is home to hundreds of bacterial species, and in the anaerobic, nutrient-rich environment of a necrotic root canal, a specific consortium of pathogens flourishes.[17] This typically includes a mix of facultative anaerobes, predominantly *Viridans* group streptococci (*Streptococcus anginosus* group being particularly virulent), and a host of obligate anaerobes, such as *Prevotella*, *Porphyromonas*, *Fusobacterium nucleatum*, and anaerobic cocci like *Peptostreptococcus*.[16] This is not merely a collection of independent organisms; they work in concert. The initial invasion may be led by the streptococci, which, by consuming local oxygen, create an even more favorable environment for the

obligate anaerobes to thrive. These bacteria then unleash a formidable arsenal of virulence factors. They produce enzymes like hyaluronidase, collagenase, and streptokinase, which actively degrade the body's connective tissue matrix and dissolve fibrin clots. This enzymatic warfare is what allows the infection to spread so rapidly as a diffuse cellulitis, rather than being effectively walled off by the host's defenses into a contained abscess.[18] The thin, watery, and often foul-smelling pus characteristic of these infections is a direct result of this tissue-dissolving process. Furthermore, many of these bacteria, particularly the gram-negative anaerobes, produce gas as a byproduct of their metabolism. While not prominent in this case, the presence of subcutaneous emphysema or crepitus on palpation is an ominous sign of a gas-forming infection, often associated with an even more fulminant course. This understanding of the polymicrobial and synergistic nature of the infection is the foundation for rational antibiotic therapy. No single antibiotic can reliably cover the entire spectrum of potential pathogens. The empirical choice in our patient of a third-generation cephalosporin (Ceftriaxone) combined with Metronidazole was a sound and evidence-based strategy.[18] The cephalosporin provides excellent coverage for the aerobic and facultative streptococci, while Metronidazole is highly effective against the obligate anaerobes that constitute the bulk of the infection. This combination therapy attacks the microbial conspiracy from multiple angles, disrupting the synergy and helping to halt the infection's advance, but only as an adjunct to the definitive treatment: surgical drainage.

In an era increasingly reliant on advanced technology, this case serves as a powerful testament to the enduring art of clinical diagnosis. The gold standard for visualizing deep neck space infections is, without question, a contrast-enhanced CT scan. It can delineate the precise anatomical extent of the infection, differentiate between cellulitis and a drainable fluid collection, and provide a definitive assessment of airway caliber.[19] However, in many parts of the world, and even in well-equipped centers in off-hours, immediate access to CT scanning is not a reality. Moreover, in a patient with impending airway compromise, the time taken to obtain a scan and the risk of decompensation while lying flat in a scanner can be prohibitive. In this resource-limited context, the clinical team correctly relied on a synthesis of history and physical examination. The diagnosis of Ludwig's angina does not require a CT scan; it requires a clinician to recognize a specific and classic pattern. The key features, all present in our patient, are: An odontogenic source: A history of toothache or visible dental pathology; Rapidly progressive cellulitis: A swelling that is not soft and fluctuant but firm, brawny, and exquisitely tender; Bilateral involvement: The infection must involve the submandibular and sublingual spaces on both sides, even if it originates unilaterally; Elevation and posterior displacement of the tongue: This is the hallmark sign and the harbinger of airway loss; Absence of lymphatic involvement as the primary pathology: Ludwig's angina is a cellulitis of the fascial spaces, not an infection of the lymph nodes (though secondary lymphadenopathy is common). The team's ability to recognize this constellation of signs, despite a misleading initial radiology report, was the critical first step.[19] The use of plain radiographs was a pragmatic and intelligent choice. While not sensitive for detecting early abscesses, they served two vital purposes: ruling out other pathologies (like a fractured mandible or a radiopaque foreign object) and providing a gross assessment of the airway and mediastinum. The confirmation of significant soft tissue swelling on the lateral neck film provided objective support for the clinical diagnosis and justified the decision to proceed directly to the operating room. This case champions the principle that for Ludwig's angina, a confident clinical diagnosis trumps the need for confirmatory advanced imaging, especially when time is of the essence.

While antibiotics are a vital component of treatment, they are ultimately supportive.[19] For an established deep neck space infection with significant

tissue tension and impending airway compromise, there is no medical substitute for surgical intervention. Antibiotics have poor penetration into abscess cavities and areas of high pressure and low perfusion.[20] The core doctrine of management is, and has always been, early and aggressive surgical drainage. The surgical procedure performed on our patient exemplifies the key principles. The goal is not merely to "lance a boil" but to achieve wide and thorough decompression of all involved fascial compartments. A timid incision is doomed to failure. The wide transverse submandibular incision provided excellent access. The meticulous exploration of all three key spaces—submandibular, sublingual, and submental—is essential. It is common to find that while the swelling is diffuse, the actual amount of liquid pus is surprisingly small, trapped under high pressure within the edematous tissues. The act of opening these fascial planes relieves this pressure, which immediately improves tissue perfusion, allows antibiotics to reach their target, and, most critically, alleviates the pressure on the airway. The placement of a drain is not an afterthought but a crucial part of the procedure. It prevents the premature sealing of the skin incision, which would allow fluid to re-accumulate underneath, leading to a recurrence of the problem. The drain acts as a continuous vent, allowing any residual inflammatory exudate to escape until the underlying infection is controlled. The final step in source control was the extraction of the offending tooth, #47. While the initial incision and drainage surgery is a life-saving emergency procedure to manage the spread of infection, the original source must be eliminated to prevent recurrence. Delaying the extraction until the acute systemic phase of the infection had resolved was a sound judgment, as it allowed for a more controlled procedure under local anesthesia once the patient was stable and the trismus had improved.[20] The complete resolution of the patient's symptoms only after the extraction underscores the importance of this definitive step in the treatment chain. The entire therapeutic arc, from emergency decompression to definitive source removal, highlights a comprehensive and well-executed surgical strategy that directly led to the patient's full recovery.

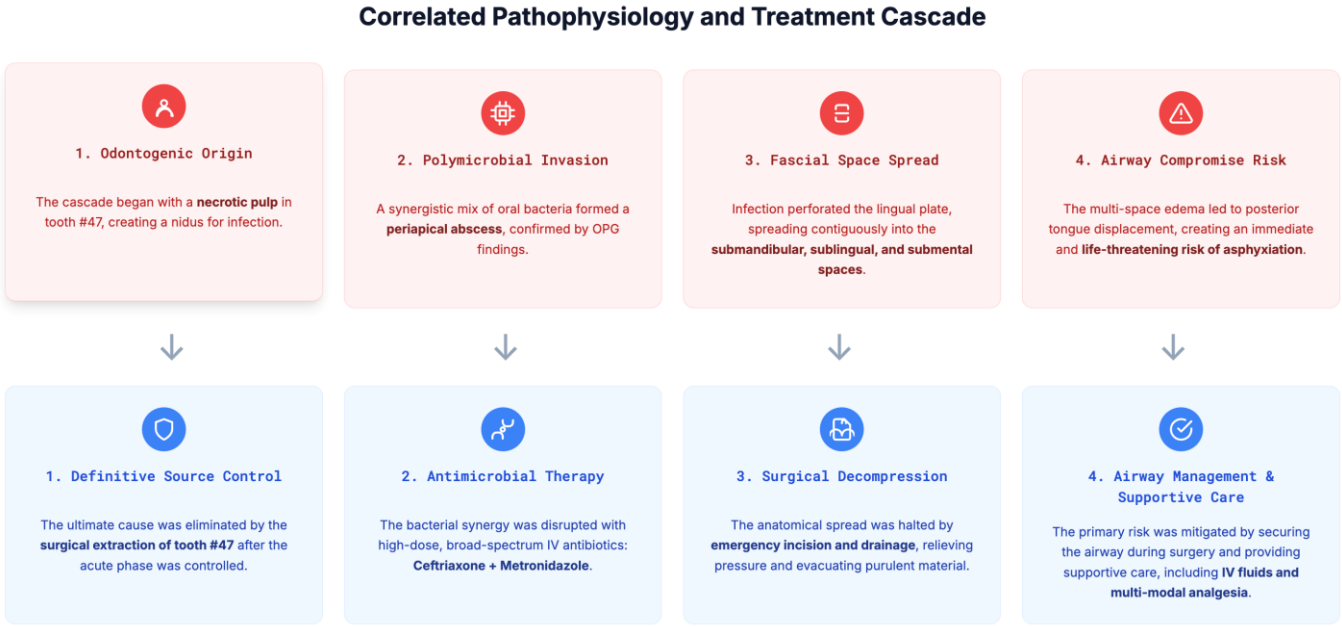

Figure 5. Correlated pathophysiology and treatment cascade.

Figure 5 showed a masterful and elegant depiction of a dual cascade, illustrating the grim, step-by-step progression of a life-threatening disease and the precisely correlated, life-saving interventions deployed to counteract it. It serves as a powerful visual narrative, mapping the journey from a single, localized dental failure to a systemic crisis, and simultaneously charting the course of a decisive and successful therapeutic response. The figure is bisected into two parallel flows: a top row, rendered in alarming shades of red, which meticulously details the pathophysiology of Ludwig's angina as it unfolded in this specific case; and a bottom row, in calming and authoritative shades of blue, which outlines the targeted treatment strategy that dismantled the pathological process at every stage. The top row of the figure tells a story of relentless anatomical invasion, a domino effect where each stage precipitates the next with devastating logic. Stage 1: The Odontogenic Origin The entire catastrophic sequence began from the most humble and common of origins: a single tooth. The first panel, titled "Odontogenic Origin," correctly identifies the nidus of infection as the necrotic pulp within tooth #47, the mandibular right second molar. This is the "patient zero" of the infection. Pulp necrosis is the death of the vital neurovascular tissue within a tooth, typically resulting from deep, untreated dental caries. Once this tissue dies, the hollow, protected space of the root canal system transforms from a living part of the body into a perfect incubator for bacteria. It becomes a sealed, anaerobic chamber, rich in necrotic tissue that serves as a nutrient broth for a burgeoning polymicrobial community. The icon, depicting a tooth with a central point of decay, perfectly encapsulates this initial, contained failure. The cascade, at this point, is silent and localized, a gathering storm hidden from view within the bone of the jaw. Stage 2: The Polymicrobial Invasion The second panel, "Polymicrobial Invasion," illustrates the consequence of this unchecked bacterial proliferation. The bacteria, having exhausted the resources within the tooth, begin to egress from the tiny openings at the root apices into the surrounding bone. Here, they establish a new frontier. This is not the work of a single bacterial species, but a synergistic conspiracy. Aerobic and facultative anaerobic bacteria, such as *Viridans* group streptococci, work in concert with a host of obligate anaerobes like *Fusobacterium*, *Prevotella*, and *Peptostreptococcus*. The aerobes consume local oxygen, creating an even more favorable environment for the anaerobes, which in turn produce potent toxins and enzymes. The body's immune system responds with a vigorous inflammatory reaction, sending neutrophils to the site to combat the invasion. The resulting battleground, filled with dead bacteria, dead white blood cells, and liquefied tissue, forms a periapical abscess. The figure correctly notes that this stage was confirmed by the OPG findings, which would have shown a distinct radiolucency—a dark area on the X-ray—at the apex of tooth #47, representing the bone being destroyed by the abscess. The icon, a representation of a bacterial cell, signifies this shift from a sterile problem to a septic one. Stage 3: The Fascial Space Spread This is the most critical juncture in the entire process, where a localized dental problem transforms into a regional, life-threatening crisis. The third panel, "Fascial Space Spread," explains the anatomical breakout. The immense pressure built up within the abscess seeks the path of least resistance. For a mandibular second molar, whose roots lie below the mylohyoid muscle, the thinnest wall of bone is typically on the lingual (tongue) side. The abscess perforates this lingual cortical plate, and the purulent material is discharged not into the mouth, but into the deep tissues of the neck. It gains immediate access to the submandibular space. This is the point of no return. The fascial planes of the neck, which normally compartmentalize structures, now act as superhighways for the infection. The bacteria, armed with their tissue-dissolving enzymes, spread with alarming speed. The infection travels around the posterior border of the mylohyoid muscle to invade the sublingual space above it, and it crosses the midline to involve the submental space. The icon, representing interconnected compartments, visually explains this contiguous spread through the deep fascial planes.

This multi-space involvement is the defining anatomical feature of Ludwig's angina. Stage 4: The Airway Compromise Risk The final panel of the pathological cascade, "Airway Compromise Risk," depicts the ultimate and most feared consequence of the anatomical spread. The infection of the sublingual and submandibular spaces leads to massive edema and a characteristic "woody" or brawny induration of the tissues of the floor of the mouth. This swelling has nowhere to go but up and back. It physically thrusts the tongue superiorly against the palate and posteriorly towards the back of the throat. This mechanical displacement of the tongue effectively turns it into a large, soft plug that can completely obstruct the oropharyngeal airway. This is not a slow, gradual process; it can happen with terrifying rapidity. The patient literally suffocates. This immediate and life-threatening risk of asphyxiation is what makes Ludwig's angina one of the most profound emergencies in all of medicine. The warning icon in the figure aptly conveys this imminent danger. The patient's presenting signs of trismus, fever, and a massively swollen neck were the clinical manifestations of this final, critical stage.

The bottom row of Figure 5, rendered in calming blue, illustrates the logical, multi-faceted, and perfectly correlated treatment strategy that was deployed to dismantle the disease process. Each therapeutic action directly targets and neutralizes a specific stage of the pathophysiology. Intervention 1: Definitive Source Control Directly opposing the "Odontogenic Origin," the first treatment pillar is "Definitive Source Control." While the emergency surgery addresses the spread, the infection will inevitably recur if the original bacterial factory is left in place. The ultimate cure, therefore, was the surgical extraction of the non-restorable tooth #47. This act removes the primary nidus of infection, eliminating the source of the bacteria and preventing any future episodes. As the figure notes, this was performed after the acute, life-threatening phase was controlled, allowing for a safer and more predictable procedure. The shield icon represents this act of protecting the body from the initial source of harm. Intervention 2: Antimicrobial Therapy Targeting the "Polymicrobial Invasion," the second intervention was aggressive "Antimicrobial Therapy." Recognizing that the infection was a synergistic conspiracy, the treatment could not rely on a single agent. The chosen combination of Ceftriaxone and Metronidazole provided a powerful, broad-spectrum attack. Ceftriaxone targeted the aerobic and facultative anaerobic streptococci, while Metronidazole is highly effective against the obligate anaerobes. This dual-pronged assault disrupted the bacterial synergy, halting their replication and enzymatic warfare. This high-dose, intravenous administration ensured that effective concentrations of the antibiotics reached the inflamed tissues, working in tandem with the surgical drainage to overwhelm the infection. The icon, representing a targeted attack on a microbe, perfectly illustrates this pharmacological strategy. Intervention 3: Surgical Decompression This is the most critical, life-saving intervention, directly countering the "Fascial Space Spread." The third panel, "Surgical Decompression," highlights the cornerstone of treatment for Ludwig's angina. Antibiotics alone cannot penetrate the high-pressure, low-perfusion environment of the swollen fascial spaces. The only effective treatment is to surgically open these compartments. The emergency incision and drainage procedure achieved several critical goals at once: it immediately relieved the intense pressure on the tissues, which in turn reduced the pressure on the airway; it evacuated the bulk of the purulent material and bacteria; and it improved blood flow to the area, allowing the body's own immune cells and the administered antibiotics to reach the site of infection more effectively. The placement of a drain ensured that these spaces remained open and decompressed during the initial, most critical phase of healing. The surgical tools icon signifies this active, procedural intervention. Intervention 4: Airway Management & Supportive Care Finally, directly addressing the "Airway Compromise Risk," the fourth panel details "Airway Management & Supportive Care." This is the

foundation upon which all other treatments are built. Before any surgical incision could be made, the patient's airway had to be secured by the anesthesiology team, a procedure fraught with difficulty given the anatomical distortion. This act of securing the airway is the single most important step in preventing mortality. Concurrently, supportive care in the form of intravenous fluids corrected the dehydration caused by fever and inability to drink, while multi-modal analgesia managed the severe pain. This comprehensive support stabilized the patient, allowing his body to better fight the systemic effects of the infection, such as the fever and the pronounced leukocytosis. The checkmark icon symbolizes the successful mitigation of the primary risk and the restoration of stability, bringing the patient back from the brink and setting the stage for a full recovery.

4. Conclusion

This case of Ludwig's angina, born from the silent decay of a single molar, serves as a dramatic and compelling narrative on the profound potential of odontogenic disease. It stands as a powerful testament to the enduring principles of clinical medicine, where the astute interpretation of signs and symptoms can, and must, guide life-saving action, even in the absence of modern technological adjuncts. The journey of this patient from the precipice of an airway catastrophe to a full and complete recovery was navigated not by reliance on complex machinery but by the fundamental pillars of surgical care: a rapid and accurate clinical diagnosis, a deep understanding of the anatomical pathways of infection, and the courage to undertake immediate, aggressive surgical decompression. The successful outcome reinforces a timeless clinical lesson: in the face of a rapidly progressive deep neck space infection, there is no substitute for decisive action. The "woody" floor of the mouth is not merely a sign but a clarion call; the displaced tongue is not a symptom but a countdown. This case champions the doctrine that early surgical intervention is not just a treatment option but the definitive antidote to the lethal progression of Ludwig's angina. It reminds us that behind every dental abscess lies the potential for a systemic crisis, and it is our solemn duty as clinicians to recognize the pattern, understand the threat, and act with the conviction and skill necessary to turn the tide and restore our patients to health.

5. References

1. Bala M, Abubakar MK, Bawa AT, Adigun OI, Sulaiman S. A rare case report of Ludwig's angina-like presentation following a viper bite. Malaysian Journal of Oral and Maxillofacial Surgery (MJOMS). 2025.
2. Chen S-J, Ji N, Chen Y-X, Xiao J-R, Wei X-Z, Liu Y-K. Effectiveness of negative pressure wound therapy in Ludwig's angina: a retrospective study of 18 cases. BMC Surg. 2025; 25(1): 223.
3. Ünal R, Mollaoğlu N. A case of Ludwig's Angina: a rare and potentially lethal neck infection. ADO Klinik Bilimler Dergisi. 2025; 14(2): 136–9.
4. Karanth T, Lal N, Anand V, Raina S. Ludwig's angina in an otherwise healthy adult. BMJ Case Rep. 2025; 18(6): e263024.
5. Okonkwo N, Montoya K, Shokoohi H. Floor of the mouth ultrasound assessment in Ludwig's angina. J Emerg Med. 2025; 74: 86–91.
6. Crawford J. A new culprit on the block: Slackia exigua causing severe Ludwig's angina with mediastinal involvement. Int J Oral Maxillofac Surg. 2025; 54: 205.
7. Acharya S. Navigating complexity: Precision assessment paves the way for minimal intervention in Ludwig's angina. Int J Oral Maxillofac Surg. 2025; 54: 177.
8. Distler K, Hammad A, Ryder E, Pokharel S. Ludwig's angina and a complicated course of *Streptococcus constellatus* management. J Intensive Care Med. 2025; 8850666251357488.

9. Dhusia DAH, Shah DY, Rekulwad DN. Transmigrated impacted mandibular canine: Cause of Ludwig's angina. Int J Appl Dent Sci. 2022; 8(3): 82–5.
10. Yelibora, Obiri-Yeboah, Larmie, Olesu, Acheampong O, Donkor. A retrospective study of Ludwig's angina at the maxillofacial unit of the Komfo Anokye Teaching Hospital-Kumasi, Ghana. GDJ. 2023; 20(1): 26–9.
11. Sabrina B. Unusual pathogen isolated in patient with Ludwig's angina. Int J Oral Dent Health. 2023; 9(2).
12. Balaji VCR, Vani K. Ludwig's angina during pregnancy-A case report. Indian J Dent Res. 2024; 35(1): 104–6
13. Tejura N, Kapila R, Dever L. Ludwig's angina complicated by descending necrotizing mediastinitis and superimposed Candida mediastinitis: Case report and literature review. J Dent Maxillofac Surg. 2018; 1(1): 33–6.
14. Thirunavakarasu R, Ganapathy D, Rohinikumar S. Awareness of Ludwig's angina among dental practitioners. Int J Res Pharm Sci. 2020; 11(Suppl 3): 996–9.
15. Cardoso LL, Gasperini G, Cardoso LC, Scartezini GR, Campos AIMS, Marão HF. Successful management of Ludwig's angina due to dental implant displacement: a rare case report. Case Rep Dent. 2020; 2020: 6934286.
16. Owobu T, Ekuase E, Azah OO, Adamu SS, Bamgbose BO. Ludwig's angina - an emergency complication arising from delayed dental treatment: The experience in a Nigerian tertiary institution. J Dentomaxillofacial Sci. 2020; 5(2): 129.
17. Gonzalez JM, McGhee S, Nadeau C, Ortega J. Facial swelling after dental work done: a case of Ludwig's angina. Adv Emerg Nurs J. 2023; 45(1): 23–8.
18. Angrish P, Kamboj A, Chopra SS, Sharma A, Kaul R. Management of life-threatening facial cellulitis (Potential Ludwig's Angina) due to dental infection in a high altitude setup- A case report. Int J Oral Health Dent. 2023; 9(3): 225–9.
19. Abu-Shanab A, Vangala A, Albdour Z, Nasr H, Rathod M, Du D. From routine to ruin: An astonishing computed tomography scan reveals catastrophic Ludwig's angina and necrotizing mediastinitis after a simple dental procedure. Eur J Case Rep Intern Med. 2025; 12(5): 005285.
20. Abu Shanab A, Vangala A, Nasr H, Rathod M, Anwar MS, Du D. From routine to ruin: An astonishing CT scan exposes catastrophic Ludwig's angina and necrotizing mediastinitis post simple dental procedure. Am J Respir Crit Care Med. 2025; 211: A3914.