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# Palatal Abscess of Odontogenic Origin Causing Dyspnea in an Elderly Patient: A Case Report and Critical Review of Management Strategies

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**A B S T R A C T**

Odontogenic infections are prevalent clinical entities, typically presenting as localized pathologies within the alveolar process. However, a palatal abscess—a specific manifestation arising from the spread of infection through the palatal cortical plate of maxillary teeth—rarely escalates to cause life-threatening airway compromise. This report details an exceptional case of a palatal abscess causing significant dyspnea in an elderly patient, challenging the conventional understanding of the hard palate as a robust anatomical barrier preventing deep space extension. A 68-year-old female presented to the emergency department on October 1st, 2022, with a five-day history of progressively enlarging palatal swelling, dysphagia, and dyspnea. Clinical examination revealed a massive, fluctuant mass extending from the hard to the soft palate, obstructing the oropharyngeal inlet. Despite a leukocyte count at the upper limit of normal and an afebrile status—indicative of geriatric immunosenescence—the patient exhibited objective tachypnea (24 breaths/min). Diagnosis was confirmed via needle aspiration. Management involved immediate airway stabilization, broad-spectrum antibiotics (Ceftriaxone and Metronidazole), and corticosteroids. On October 3rd, 2022, the patient underwent incision and drainage under general anesthesia, yielding 15cc of purulent material. The source was identified as carious residual roots of the maxillary right second molar (FDI #17). Post-operative recovery was rapid, with discharge on October 6th, 2022. In conclusion, this case underscores that palatal abscesses can evolve into critical airway emergencies, particularly in geriatric patients with diminished physiological reserves and blunted immune responses. Prompt recognition, aggressive multidisciplinary management, and definitive dental treatment are paramount to preventing mortality.

## 1. Introduction

Odontogenic infections, originating from the dental, periodontal, or pericoronal structures, constitute a significant proportion of the caseload in oral and maxillofacial surgery and otorhinolaryngology. These infections are predominantly polymicrobial, driven by a synergistic alliance of indigenous oral aerobes and anaerobes.[1] While the majority of these infections remain localized to the alveolus or spread into the vestibular space, the anatomical trajectory of the infection is dictated strictly by the relationship of the root apex to the muscle attachments and cortical bone plates.[2]

In the maxilla, when the root apices of the lateral incisors or palatal roots of premolars and molars lie closer to the palatal cortical plate, the infection may

perforate medially, resulting in a palatal abscess.[3] Clinically, this presents as a well-demarcated, tense, and exquisitely painful swelling, confined by the dense adherence of the mucoperiosteum to the hard palate. Because of this tight anatomical compartmentalization, palatal abscesses are traditionally viewed as localized events with a low propensity for descending into deep neck spaces or compromising the airway.[4]

However, the clinical trajectory described in this report challenges this paradigm. Severe complications such as maxillary sinusitis, orbital cellulitis, or cavernous sinus thrombosis are documented but rare.[5] Even more exceptionally, the posterior extension of a palatal abscess into the soft palate can precipitate acute upper airway obstruction. A review of the current literature highlights that while deep neck space infections (Ludwig's angina) are well-known causes of airway compromise, dyspnea resulting specifically from a palatal abscess is an exceedingly uncommon and sparsely reported phenomenon.[6,7]

This clinical scenario is further complicated in the geriatric population. The physiological phenomenon of immunosenescence results in an attenuated systemic inflammatory response, often masking the severity of infection.[8] Elderly patients may present with normal body temperature and leukocyte counts despite harboring severe infections, leading to dangerous diagnostic delays. Furthermore, the reduced physiological reserve in this demographic means they tolerate respiratory compromise poorly, with a rapid trajectory toward respiratory failure.[9,10]

The novelty of this study lies in the detailed documentation of a rare pathophysiological sequence: the progression of a localized palatal abscess into a life-threatening airway obstruction in a geriatric patient. Unlike standard reports, this study critically analyzes the silent progression typical of the elderly, where subjective dyspnea outweighs objective systemic markers. The aim of this study is to report this unusual presentation, to meticulously discuss the anatomical mechanisms by which palatal infections compromise the airway, to highlight the diagnostic pitfalls associated with geriatric immunosenescence, and to propose an aggressive, multidisciplinary management algorithm that prioritizes airway security and rapid surgical intervention to avert catastrophic outcomes.

## 2. Case Presentation

A 68-year-old female patient (initials NKS) was transferred from a regional general hospital to the Emergency Department of Prof. Dr. I.G.N.G. Ngoerah General Hospital on October 1st, 2022. The referral was precipitated by a constellation of worsening symptoms, including severe oral pain, difficulty swallowing (dysphagia), and, most critically, difficulty breathing (dyspnea). The patient's demographic profile and admission details are summarized in Table 1.

Table 2 provides a chronological reconstruction of the patient's clinical deterioration, mapping the trajectory from a latent, neglected dental pathology to a fulminant, life-threatening airway emergency over a critical 120-hour window. This temporal framework is essential for understanding the insidious nature of deep space odontogenic infections, particularly in how they can initially masquerade as benign localized events before rapidly escalating. The chronology begins not on Day 1 of acute symptoms, but weeks prior, establishing a crucial baseline of chronic, untreated dental disease. The patient's report of a persistent dull ache in the maxillary right quadrant signifies a long-standing necrotic pulp and chronic periapical periodontitis at tooth #17. This latent phase represents a period of equilibrium where host defenses temporarily contain the polymicrobial flora within the immediate dentoalveolar structure. The acute phase, marked as Day 1, initiated with the breach of the palatal cortical plate. The presentation of a small, tender lump on the hard palate is pathognomonic for the initial formation of a subperiosteal abscess.

Table 1. Patient Demographics and Admission Profile

| PARAMETER | PATIENT DETAILS |
|---|---|
| Patient Initials | NKS |
| Age / Sex | 68 Years / Female |
| Referral Source | Regional General Hospital (Negara) |
| Admission Date | October 1, 2022 |
| Admission Time | 21:00 WITA |
| Chief Complaint | **Dyspnea, Dysphagia, Palatal Swelling** |
| Occupation | Housewife |
| Comorbidities | None Reported (No DM, HTN, CVD) |

At this stage, the tight adherence of the mucoperiosteum to the hard palate bone served as an anatomical barrier, limiting the spread of purulence and resulting in symptoms that were discomforting but not alarming. However, the progression through Days 2 and 3 illustrates the relentless hydraulic pressure exerted by continued pus production. The transition of pain from intermittent to continuous and throbbing clinically correlates with increasing tension within the abscess cavity and the resultant ischemia of the overlying mucosa, alongside the recruitment of acute inflammatory mediators. The critical inflection point in this patient's course is documented on Day 4. The onset of dysphagia for both liquids and solids signals a distinct anatomical shift: the infection had burst beyond the confines of the hard palate and dissected posteriorly into the soft palate (velum). Unlike the fixed hard palate, the soft palate is a dynamic muscular curtain rich in loose connective tissue. Invasion of this space allows for rapid, massive distension. This posterior extension is the harbinger of airway compromise, as the swelling begins to encroach upon the oropharyngeal inlet, interfering with the complex muscular coordination required for deglutition. By Day 5 (AM), the clinical picture had evolved into a bona fide emergency. The development of dyspnea and the characteristic hot potato voice (rhinolalia clausa) provided definitive clinical evidence that the massive, edematous soft palate was now acting as a physical obturator, significantly increasing upper airway resistance. The patient's subjective sensation of airway blockage was a direct correlate of this mechanical narrowing. The final entry on Day 5 (PM), marking admission to the Emergency Department, underscores the rapidity of decompensation. The presence of objective tachypnea (24 breaths/min) at this stage indicates that the patient's physiological compensatory mechanisms were under significant strain to maintain adequate gas exchange against the fixed obstruction. This detailed timeline serves as a stark pedagogical reminder that in odontogenic infections, the interval between localized discomfort and critical airway threat can be deceptively short, necessitating early and aggressive intervention before the soft palate is compromised.

Table 2. Chronology of Symptom Progression

Visual mapping of clinical deterioration over 120 hours

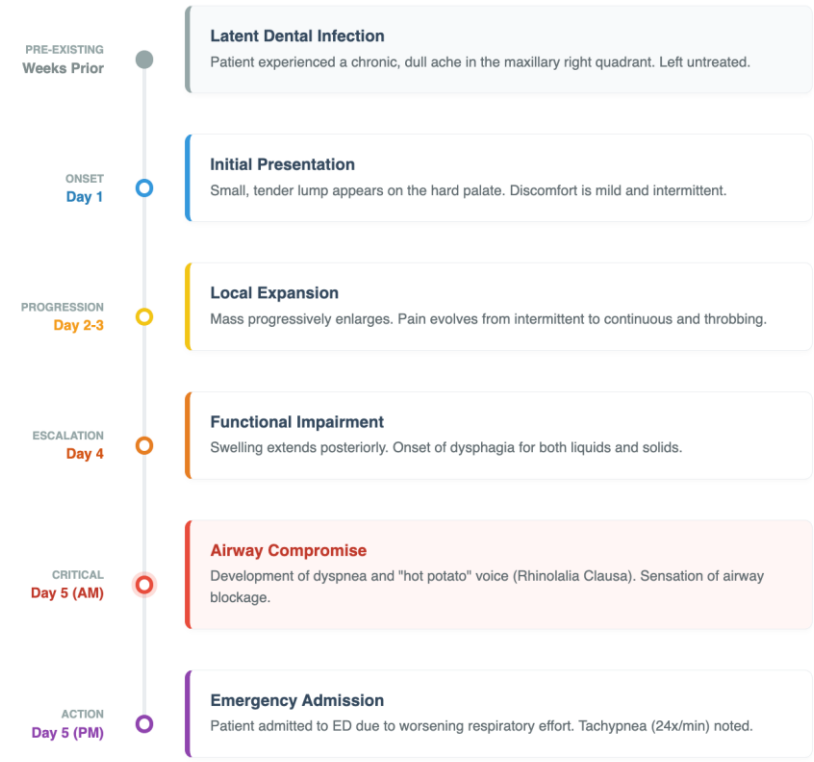



Figure 1 serves as a comprehensive clinical dashboard, synthesizing the dramatic visual evidence of the pathology with the patient's physiological status upon presentation. The core of this figure is the intraoral assessment, which reveals the immediate and formidable nature of the threat. The image graphically depicts a massive, dome-shaped swelling dominating the oral cavity. The overarching erythema and profound edema of the overlying mucosa are classical signs of acute, intense inflammation. Crucially, the visual evidence highlights the anatomical extent of the abscess; it is not confined to the anterior hard palate but shows significant posterior progression, causing a downward and backward displacement of the soft palate. This visual finding directly explains the mechanical narrowing of the oropharyngeal inlet, confirming the anatomical basis for the patient's dyspnea and dysphagia. The clinical notation of fluctuance upon palpation is a pivotal diagnostic detail, confirming the presence of a liquid collection (pus) under tension, rather than a solid neoplastic mass, and immediately guiding the therapeutic approach toward surgical drainage. Juxtaposed against this alarming anatomical presentation is the vital signs panel, which introduces the critical theme of geriatric presentation. The patient's respiratory rate of 24 breaths per minute is highlighted as a critical indicator of physiological stress. This tachypnea is a compensatory reflex driven by hypercapnia or hypoxia resulting from the increased work of breathing against the oropharyngeal obstruction. The accompanying tachycardia (96 bpm) further reflects the systemic stress response to pain, anxiety, and impending respiratory fatigue. However, the most instructionally significant data point in Figure 1 is the temperature reading of 36.8°C. The

state of being afebrile in the presence of such a massive, purulent infection is a hallmark presentation of immunosenescence in the elderly. In a younger adult, an abscess of this magnitude would typically elicit high-grade fevers and rigors driven by robust cytokine release (IL-1, IL-6, TNF-α). In this geriatric patient, the blunted innate immune response fails to reset the hypothalamic thermostat. This figure powerfully illustrates the diagnostic danger of relying on classic sepsis markers in the elderly; the lack of fever could easily lead a clinician to underestimate the severity of the infection. Therefore, Figure 1 demands that the clinician prioritize objective functional markers of airway patency—specifically respiratory rate and the visual extent of swelling—over traditional systemic markers like temperature when assessing geriatric patients with deep space head and neck infections. The combination of the visual pathology and the vital signs creates a complete clinical picture of a silent but imminent respiratory crisis.

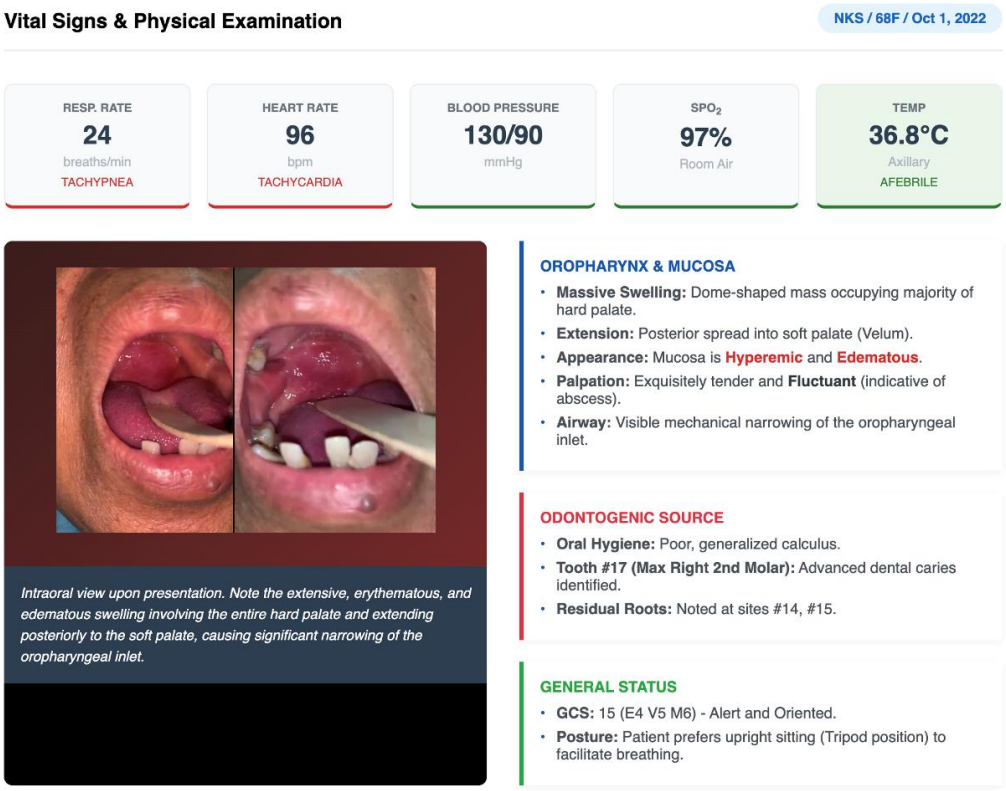



Figure 1. Vital signs and physical examination.

Table 3 provides a detailed analytical view of the diagnostic adjuncts that confirmed the clinical impression and stratified the patient's risk. The data presented here reinforces the complex interplay between severe local pathology and a deceptively muted systemic response, characteristic of geriatric patients. The hematological profile is dominated by the leukocyte count (WBC) of 10.60 x $10^9$/L. While technically falling just within the upper limit of the standard reference range, its interpretation requires significant clinical nuance. In the context of a massive, poorly contained 15cc abscess, this value represents a relative leukopenia. It signifies a blunted bone marrow response, where the elderly patient is unable to mount the substantial leukocytosis (>15.0 x $10^9$/L with a left shift) that would be expected in a younger individual

with an equivalent bacterial load. This data point serves as a critical reminder that normal laboratory values must be interpreted within the context of age and clinical severity; in this scenario, a high-normal WBC is, in fact, pathologically low relative to the stimulus. Furthermore, the hemoglobin level of 9.4 g/dL indicates a normocytic normochromic anemia, likely reflecting anemia of chronic disease, suggesting the patient's overall physiological reserve was already compromised prior to this acute episode. The microbiology section highlights the utility of immediate bedside diagnostic measures. The needle aspiration yielding foul-smelling purulent fluid provided instant, irrefutable confirmation of an abscess, ruling out other differentials such as a vascular malformation or a solid tumor, and justifying immediate surgical intervention. While the eventual culture revealed only scanty growth—likely an artifact of prior antibiotic exposure or transport limitations for anaerobes—the gross appearance and odor of the aspirate were sufficient for empirical decision-making. The radiological data focuses on the immediate priority: the airway. The lateral cervical X-ray, while less detailed than CT, provided rapid, bedside confirmation of soft tissue prominence in the retropalatal and oropharyngeal spaces. This imaging finding objectively corroborated the physical exam and the patient's subjective dyspnea, demonstrating the encroachment of the swollen soft palate upon the air column. The clear chest X-ray was a relevant negative finding, ruling out immediate complications such as aspiration pneumonia, which is a high risk in patients with dysphagia and glottic edema. Collectively, the data in Table 3 paint a picture of a patient with a confirmed severe pyogenic infection threatening the airway, whose systemic immune markers are dangerously understated due to age-related physiological changes.

Table 3. Laboratory & Radiological Profile — Oct 01, 2022

**HEMATOLOGY & MICROBIOLOGY**

| | | |
|---|---|---|
| Leukocytes (WBC) | 10.60 × 10⁹/L | Ref: 4.5-11.0 |
| ⚠ *Upper Limit of Normal:* Indicative of blunted immune response (Immunosenescence) despite severe infection. | | |
| Hemoglobin (Hb) | 9.4 g/dL | Ref: 12.0-15.0 |
| **Low:** Normocytic Normochromic Anemia. | | |
| Dx. Needle Aspiration | Purulent Fluid | |
| **Positive:** Foul-smelling pus confirmed. Definitive diagnosis of Abscess. | | |

**RADIOLOGICAL IMAGING**

| | |
|---|---|
| Cervical X-Ray (Lateral) | Soft Tissue Prominence |
| **Prevertebral/Retropalatal:** Evidence of airway narrowing at the oropharyngeal level. | |
| Chest X-Ray (PA) | Clear Lung Fields |
| **Negative:** No evidence of aspiration pneumonia, cardiomegaly, or mediastinal widening. | |
| *Note: CT Scan was deferred in favor of immediate airway management and surgical intervention based on clinical and X-ray findings. | |

Figure 2 is a composite schematic that bridges the theoretical management algorithm with the practical surgical reality of treating a life-threatening palatal abscess. The left panel visualizes a structured, stepwise clinical pathway designed to simultaneously address the microbial insult, the inflammatory response, and the mechanical obstruction. Step 1 highlights the immediate prioritization of airway and breathing. Placing the dyspneic patient in a High-Fowler's position is a critical, non-pharmacological

intervention that utilizes gravity to reduce venous congestion in the neck and maximizes diaphragmatic excursion, thereby reducing the work of breathing. The preparation of a difficult airway cart reflects the anticipation of potential glottic edema or anatomical distortion that could complicate intubation. Step 2 details the aggressive intravenous pharmacotherapy regimen. The choice of Ceftriaxone and Metronidazole represents a robust, broad-spectrum antibiotic blockade targeting the complex polymicrobial flora typical of odontogenic infections, covering Gram-positive cocci, Gram-negative rods, and, crucially, strict anaerobes. The inclusion of high-dose intravenous Methylprednisolone is a pivotal element of this protocol designed to combat airway threat. By potently inhibiting the inflammatory cascade and reducing vascular permeability, steroids act synergistically with surgery to rapidly decrease the viscoelastic edema of the soft palate, accelerating airway restoration. Step 3 and the corresponding right-hand image panel represent the definitive therapeutic juncture: surgical intervention. The protocol emphasizes the necessity of General Anesthesia to ensure a secure airway and optimal surgical field, mitigating risks of aspiration or laryngospasm associated with local anesthesia in an inflamed pharynx. The intraoperative photograph provides visceral evidence of the pathology's severity. It captures the precise moment of source control, where a hemostat is used to explore the abscess cavity after incision. The description notes the evacuation of 15cc of thick, purulent exudate. This volume is substantial within the confined anatomical space of the oro- and nasopharynx. The image visually reinforces the mechanical theory of the patient's dyspnea: the immediate release of this pressurized fluid volume, which was distending the soft palate and obstructing the airway, is the single most critical action in reversing the patient's respiratory compromise. Step 4 completes the pathway by emphasizing the role of a drain to prevent re-accumulation, ensuring continued decompression post-surgery. Together, the schematic and photograph in Figure 2 illustrate a cohesive, aggressive multidisciplinary strategy where medical therapy optimizes the patient for the definitive surgical cure.

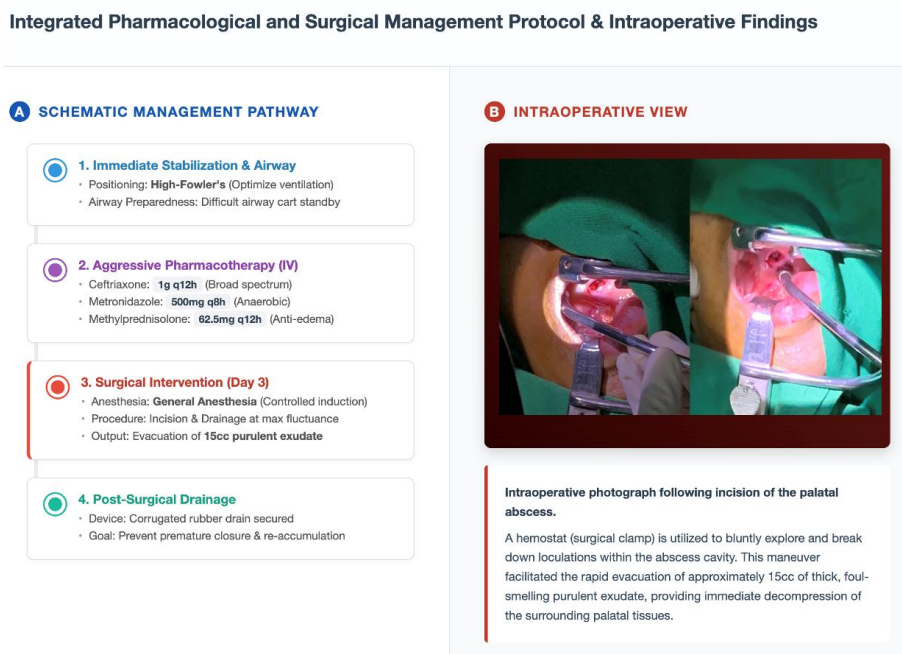


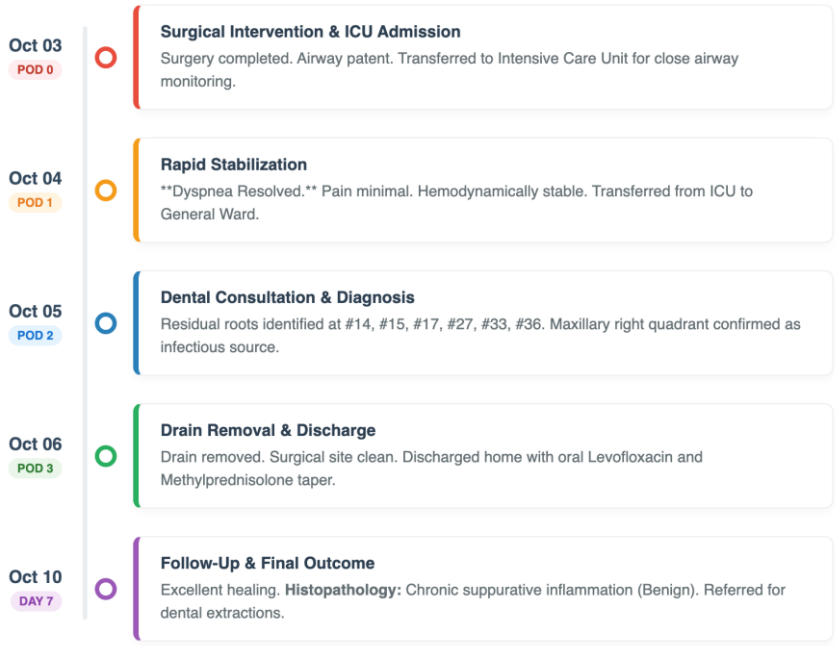

Figure 2. Integrated pharmacological and surgical management protocol and intraoperative findings.

Table 4 provides a schematic overview of the patient's rapid convalescence following definitive intervention, serving as validation for the aggressive medical and surgical management strategy employed. The timeline illustrates the dramatic reversibility of the acute airway crisis once the underlying mechanical cause was addressed. The entry for POD 0 (Post-operative Day 0) establishes the immediate post-procedural baseline: following the evacuation of 15cc of pus under general anesthesia, the patient was transferred to the ICU. This placement reflects the continued high risk of airway edema in the immediate post-extubation period, requiring vigilant monitoring despite successful surgery. The most significant clinical inflection point is recorded on POD 1. The notation that dyspnea resolved marks the successful termination of the medical emergency. The speed of this resolution—within 24 hours of surgery—underscores that the patient's respiratory distress was primarily mechanical in nature, caused by the volume and pressure of the abscess dissecting into the soft palate. Once this pressure was released via incision and drainage, and complemented by the anti-inflammatory action of high-dose corticosteroids, the airway patency was rapidly restored. The transfer from the ICU to the general ward on this day signifies a de-escalation of care based on achieved physiological stability. POD 2 highlights the transition from acute crisis management to definitive source investigation. The formal dental consultation identified multiple residual roots, specifically pinpointing the maxillary right quadrant as the origin of the infection. This milestone emphasizes that drainage of the abscess is only temporizing; identification and eventual eradication of the necrotic dental source are imperative to prevent recurrence. By POD 3, the patient met criteria for discharge. The removal of the corrugated rubber drain indicated that purulent drainage had ceased and the abscess cavity was collapsing appropriately, healing by secondary intention. The discharge plan, including oral antibiotics and a steroid taper, demonstrates a careful bridging strategy to ensure continued resolution of inflammation in the outpatient setting. Finally, the Day 7 Follow-Up confirms the favorable long-term outcome. The benign histopathology report ruled out sinister underlying pathology, and the referral for dental extractions closed the loop on the patient's care pathway, transitioning her from acute infection management to preventative oral health rehabilitation. The overall trajectory depicted in Table 4 is a testament to the efficacy of rapid surgical source control combined with supportive medical therapy in reversing life-threatening odontogenic airway obstruction.

**3. Discussion**

The case presented herein serves as a critical clinical exemplar, illustrating the capacity for a seemingly benign, localized dental pathology to metamorphose into a life-threatening airway emergency. While the existing literature copiously documents deep neck space infections—such as Ludwig's angina or parapharyngeal abscesses—as primary causes of odontogenic airway compromise, the direct obstruction of the airway by a palatal abscess is an anatomical rarity. Figure 3 serves as a comprehensive conceptual schematic, visually synthesizing the complex pathophysiological cascade that transformed a localized dental infection into a life-threatening airway emergency in this geriatric patient. This diagram is essential for understanding the case beyond its superficial clinical presentation, mapping the spatiotemporal progression of the pathology against the critical modifying influence of host physiology. The figure is structured as a sequential flow, delineating four distinct stages of anatomical spread, underpinned by a crucial modifier that explains the deceptive clinical picture.[11]

Table 4. Post-operative Recovery Milestones




The cascade initiates with Stage 1: Dental Etiology, establishing the fundamental biological driver of the process. It identifies the maxillary right second molar (FDI #17) not merely as a carious tooth, but as a nidus for systemic threat. The mechanism is identified as pulpal necrosis secondary to long-standing decay, creating a protected reservoir for a polymicrobial consortium of oral aerobes and anaerobes. This stage emphasizes that the subsequent massive pathology began with a common, often neglected, dentoalveolar condition.[12] Stage 2: Anatomical Spread illustrates the initial vector of infection, dictated by the unique microanatomy of the maxillary posterior maxilla. The figure highlights that the medial positioning of the root apex relative to the alveolar bone directed the purulent egress through the thinner palatal cortical plate, rather than the more common buccal route. At this juncture, the infection was characterized as a subperiosteal abscess confined to the hard palate. The description implies the clinical correlate of this stage: intense, localized pain due to the hydraulic pressure of pus trapped between bone and the tightly adherent, unyielding mucoperiosteum of the hard palate. Stage 3: Critical Extension marks the inflection point where the case evolved from a dental urgency to a medical emergency. The schematic visually represents the failure of the hard palate to contain the growing abscess volume. The infection dissected posteriorly, breaching the anatomical boundary between the fixed hard palate and the dynamic soft palate (velum). The significance of this transition is paramount; the soft palate is composed of muscle and loose areolar connective tissue, offering little resistance to fluid accumulation.[13] This allowed for the rapid sequestration of a substantial volume—specifically noted as 15cc of purulence—along with significant collateral inflammatory edema. This stage transformed a localized pressure problem into a space-occupying lesion in a critical area. Stage 4: Airway Compromise details the direct physiological consequence of this extension. The diagram explains the mechanical basis of the patient's dyspnea: the massive, fluid-filled soft palate was displaced inferiorly and posteriorly, acting as a physical obturator.[14] This mass effect significantly

narrowed the dimensions of the oropharyngeal inlet, the critical choke-point for airflow.[15] The figure correlates this anatomical mechanism with the presenting clinical signs of tachypnea and rhinolalia clausa (hot potato voice), demonstrating cause and effect. Crucially, the entire cascade is underpinned by the Geriatric Modifier: Immunosenescence box at the bottom. This section provides the scholarly context for why this severe anatomical progression was not met with a commensurate systemic inflammatory response. It details how age-related decline in immune vigilance led to a failure to mount classic signs of sepsis, such as high-grade fever or marked leukocytosis. This created a dangerous diagnostic dichotomy, where the patient's systemic markers suggested stability while her anatomy was critically compromised. Figure 3, therefore, not only maps the route of infection but also serves as a pedagogical tool, warning clinicians that in the elderly, anatomical pathology can progress silently and catastrophically behind a facade of physiological normalcy.[16]

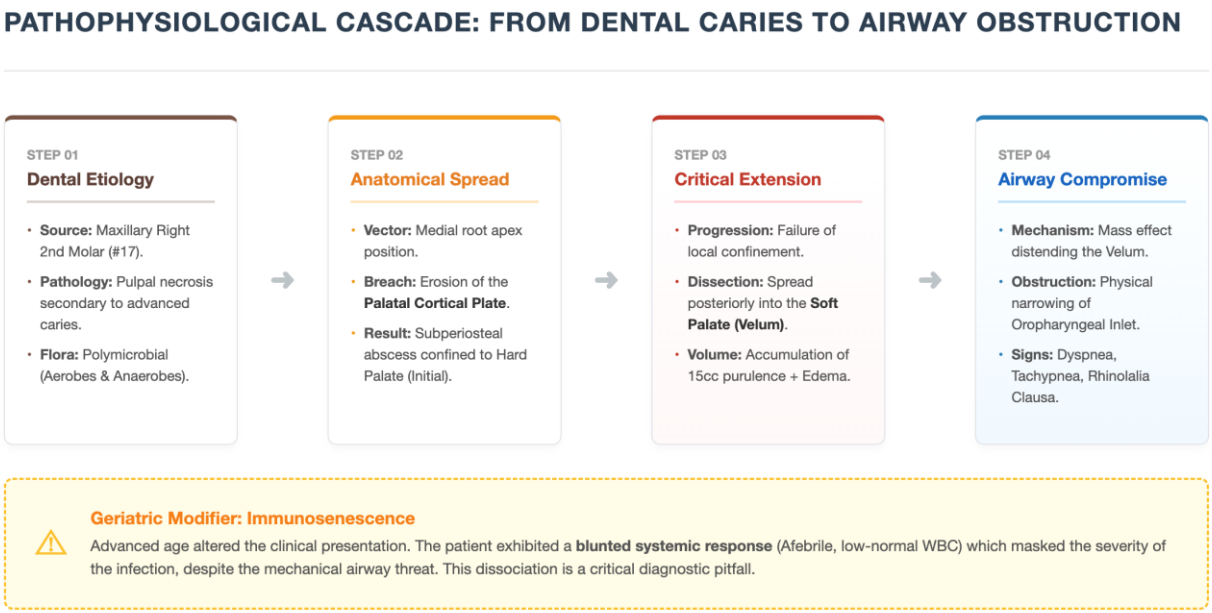



Figure 3. Pathophysiological cascade: from dental caries to airway obstruction.

The genesis of any odontogenic infection lies in the necrosis of the dental pulp, typically a sequela of untreated caries. In the maxilla, the trajectory of purulent spread is governed by the path of least resistance, which is dictated by the relationship between the root apex and the surrounding cortical bone plates.[17] As established in foundational maxillofacial literature, when the apices of the maxillary molars or premolars are positioned medially, the infection erodes through the palatal cortical plate rather than the thinner buccal plate. In the standard presentation of a palatal abscess, the infection is strictly confined. The mucoperiosteum of the hard palate lacks a submucosa in the median raphe and gingival regions and is bound tightly to the underlying bone by dense connective tissue septa.[18] This anatomical arrangement typically results in a hydraulic press effect, where the pus forms a tense,

well-demarcated hemisphere that is exquisitely painful but distinctively localized. However, the deviation in the reported case—and the primary cause of the airway threat—was the posterior extension of the abscess into the soft palate (velum). Unlike the hard palate, the soft palate is a dynamic, muscular curtain composed of the tensor veli palatini, levator veli palatini, palatoglossus, palatopharyngeus, and the musculus uvulae. Crucially, these muscles are enveloped in a matrix of loose connective tissue and glandular structures that allow for significant distension. When the purulent collection, measured at a substantial volume of 15cc, was dissected posteriorly past the junction of the hard and soft palate, it entered this distensible space. The introduction of 15cc of fluid, compounded by the inevitable reactive edema mediated by inflammatory cytokines (IL-1, IL-6, TNF-alpha), resulted in a massive increase in the volume of the soft palate. This structure forms the anterior boundary of the nasopharynx and the superior boundary of the oropharynx. As the soft palate swelled, it was displaced inferiorly and posteriorly, effectively acting as an obturator. This created a bottle-neck effect at the oropharyngeal inlet, drastically reducing the cross-sectional area available for airflow. According to Poiseuille's Law of fluid dynamics, resistance to flow is inversely proportional to the fourth power of the radius. Therefore, even a modest reduction in the airway radius caused by the bulging soft palate leads to a profound exponential increase in airway resistance, manifesting clinically as the patient's dyspnea and rhinolalia clausa.[19]

A pivotal finding in this case was the discordance between the severity of the anatomical pathology and the patient's systemic inflammatory markers. The patient presented with a massive abscess and impending airway failure, yet remained afebrile (36.8°C) and exhibited a leukocyte count of 10.60 x $10^9$/L—a value that, while at the upper limit of normal, is disproportionately low for a 15cc abscess. This phenomenon is a hallmark of immunosenescence, the gradual deterioration of the immune system associated with aging. In geriatric patients, the function of both innate and adaptive immune cells is compromised. Specifically, there is a documented reduction in the ability of macrophages and monocytes to produce endogenous pyrogens such as Interleukin-1 (IL-1) and Interleukin-6 (IL-6) in response to bacterial pathogens. Consequently, the hypothalamic set-point is not elevated, and the classic febrile response is absent. Furthermore, the bone marrow's left shift response—the rapid mobilization of neutrophils—is often delayed or blunted. The theoretical implication for the clinician is profound: in the elderly, vital signs can be vital lies. The absence of fever and leukocytosis does not exclude severe sepsis or deep-seated infection. Instead, functional parameters become the gold standard for assessment. In this case, the respiratory rate of 24 breaths per minute was the true vital sign. Tachypnea is a compensatory mechanism to maintain oxygenation in the face of increased airway resistance and metabolic demand. Had the clinical team relied solely on the lack of fever or the normal white count, the severity of the airway threat might have been drastically underestimated. This silent progression puts the geriatric patient at high risk for sudden decompensation, as their physiological reserves to maintain the work of breathing are significantly lower than in younger cohorts.

The management of this patient followed a strict, evidence-based algorithm designed to address both the microbial load and the mechanical obstruction. The selection of Ceftriaxone and Metronidazole was grounded in the polymicrobial nature of odontogenic infections. The oral cavity harbors a complex biome of over 700 bacterial species. Odontogenic abscesses are typically dominated by facultative anaerobes (*Streptococcus viridans* group) and strict anaerobes (*Fusobacterium*, *Prevotella*, *Porphyromonas*). Ceftriaxone, a third-generation cephalosporin, provides potent coverage against Gram-positive cocci

and Gram-negative rods, disrupting bacterial cell wall synthesis. Metronidazole is specifically targeted at the obligate anaerobes, functioning by reducing its nitro group within the bacterial cell to form cytotoxic free radicals that disrupt DNA. This combination offers a synergistic blockade that covers the vast majority of odontogenic pathogens. The administration of Methylprednisolone (62.5 mg) is a critical discussion point. The theoretical risk of corticosteroids in infection is immunosuppression. However, in the context of acute upper airway obstruction, the immediate threat is mechanical, not microbial. Corticosteroids act by inhibiting the phospholipase A2 pathway, thereby blocking the production of arachidonic acid and its potent inflammatory metabolites, prostaglandins and leukotrienes. This results in a rapid reduction of vascular permeability and vasodilation, directly reducing the viscoelastic edema of the soft palate. The rapid resolution of the patient's dyspnea by Post-operative Day 1 suggests that the steroids played a vital synergistic role with surgical drainage in restoring airway patency. The decision to perform incision and drainage under general anesthesia was non-negotiable. The theoretical risk of attempting this procedure under local anesthesia includes the potential for aspiration if the abscess ruptures uncontrolled into the pharynx, and the risk of laryngospasm precipitated by pain or fluid in the glottic inlet. General anesthesia with a secure endotracheal tube protected the distal airway. The evacuation of 15cc of pus accomplished the primary goal of surgery: source control. By removing the pressurized fluid, the tension on the tissue was released, restoring microvascular perfusion to the ischemic mucosa and allowing the host immune system and antibiotics to effectively reach the site of infection. The successful outcome, with discharge on Day 3, validates the aggressive multidisciplinary approach. The histological finding of chronic suppurative inflammation without malignancy confirmed the odontogenic etiology and ruled out neoplastic differentials such as adenoid cystic carcinoma or non-Hodgkin lymphoma, which can mimic palatal abscesses. The culture result showing scanty Gram-negative rods is a common finding in pre-treated abscesses, reinforcing the importance of empirical broad-spectrum coverage when culture sensitivity is compromised by prior interventions.[20]

## 4. Conclusion

This case report documents a rare and life-threatening complication of a palatal abscess in an elderly patient, challenging the paradigm that palatal infections are inherently localized. The progression of the infection into the soft palate created a mechanical airway obstruction, a sequela that was nearly masked by the blunted systemic immune response typical of geriatric immunosenescence. A palatal abscess is not merely a dental issue; posterior extension into the soft palate can rapidly compromise the retropalatal airway. The absence of fever and significant leukocytosis is a deceptive and dangerous marker of stability in the elderly. Objective functional metrics, such as respiratory rate and work of breathing, must take precedence over traditional markers of sepsis. The management of airway-threatening odontogenic infections requires a low threshold for securing the airway, the use of adjuvant corticosteroids to reduce edema, and immediate surgical drainage under controlled conditions. Ultimately, this case serves as a stark reminder of the continuity between oral health and systemic survival. Neglected dental pathology in the vulnerable geriatric population is not a static quality-of-life issue but a dynamic reservoir for potential mortality that demands acute clinical vigilance.

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