

Case Report

Emergency Awake Tracheostomy and Transcervical Drainage for a Mature Parapharyngeal Space Abscess with Impending Upper Airway Collapse: A Case Report

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ABSTRACT

Background: Parapharyngeal space abscesses are deep neck infections that can cause sudden airway obstruction, internal jugular thrombophlebitis, and descending mediastinitis. Early airway planning, contrast-enhanced computed tomography (CECT), and surgical drainage are essential.

Objective: To describe the airway-first management and recovery of a woman with a mature parapharyngeal abscess and impending airway collapse.

Methods: This report followed the CARE guideline, with written informed consent. A 44-year-old woman presented to a regional referral hospital in Sorong, Indonesia, with left neck swelling reaching the cheek and orbital margin, fever (38.8 °C), severe odynophagia, and trismus (maximal interincisal opening [MIO] 14.0 mm). She was followed with CECT, serial clinical and laboratory measurements, and pus culture over 10 days.

Results: CECT showed a multilocular, rim-enhancing collection (4.2 × 3.5 × 5.8 cm; 42.6 cm³) in the left prestyloid parapharyngeal space, with 68.4% airway narrowing and a patent carotid sheath. Awake tracheostomy under local anesthesia secured the airway, followed by transcervical submandibular drainage of about 120 mL of pus, which grew *Streptococcus* sp. sensitive to cephalosporins. Temperature fell from 38.8 to 36.5 °C, MIO rose from 14.0 to 38.0 mm, and the pain score fell from 8.5 to 0.5. She was decannulated on Day 6 and discharged on Day 10 without vascular or mediastinal complications.

Conclusion: Securing the airway with awake tracheostomy before transcervical drainage was safe and effective for a mature parapharyngeal abscess with severe airway narrowing.

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1. Introduction

Deep neck space infections (DNSIs) represent a formidable spectrum of suppurative pathologies arising within the complex potential fascial planes of the head and neck, continuing to pose severe diagnostic and therapeutic dilemmas despite substantial advancements in modern antimicrobial chemotherapy and cross-sectional diagnostic imaging.^{1–3} The intricate anatomical architecture of the cervical fascial layers, traditionally compartmentalized into superficial and deep cervical fasciae (the latter further subdivided into investing, pretracheal, and prevertebral layers), creates a labyrinth of interconnecting compliance spaces.^{4,5} Among these potential compartments, the parapharyngeal space (PPS), also historically designated as the lateral pharyngeal or

pharyngomaxillary space, occupies a central crossroad within the deep neck.^{5–7} Shaped anatomically as an inverted pyramid extending from its broad base at the petrous temporal bone of the skull base down to its apex at the greater cornu of the hyoid bone, the parapharyngeal space is bounded medially by the pharyngobasilar fascia and superior pharyngeal constrictor muscle, laterally by the medial pterygoid muscle, the ramus of the mandible, and the deep lobe of the parotid gland, and posteriorly by the prevertebral fascia.^{5,7,8} Crucially, the styloid process and its attached musculotendinous structures (the stylopharyngeus, styloglossus, and stylohyoid muscles, forming the styloid diaphragm) divide the parapharyngeal space into two distinct anatomical compartments: the anterior or prestyloid compartment and the posterior or poststyloid (retrostyloid) compartment.^{7,9,10}

The prestyloid parapharyngeal compartment consists predominantly of loose fibrofatty connective tissue, lymphatic vessels, and the ascending palatine artery, and lies in intimate contiguity with the tonsillar fossa and the masticator space.^{10,11} Consequently, suppurative infections originating from the palatine tonsils, peritonsillar crypts, or lower molar odontogenic foci can readily penetrate the thin pharyngobasilar barrier to establish a rapidly expanding prestyloid abscess.^{11,12} In contrast, the poststyloid compartment houses critical neurovascular bundles, including the internal carotid artery (ICA), the internal jugular vein (IJV), cervical sympathetic trunk, and cranial nerves IX, X, XI, and XII.^{12,13} The anatomical proximity of these vital conduits means that suppurative involvement of the parapharyngeal space harbors catastrophic, life-threatening complications, including acute upper airway compromise secondary to medial pharyngeal wall displacement, suppurative thrombophlebitis of the internal jugular vein with septic pulmonary embolization (Lemierre syndrome), internal carotid artery pseudoaneurysm formation or fatal exsanguinating rupture, erosion into the retropharyngeal and danger spaces, and rapid descent into the posterior mediastinum manifesting as descending necrotizing mediastinitis (DNM).¹¹⁻¹⁵ Historical reports prior to the antibiotic era documented mortality rates exceeding 50% for deep neck space infections; although contemporary antimicrobial and surgical paradigms have reduced overall mortality to approximately 1.5% to 5.0%, complicated presentations involving airway stenosis and multi-space tracking continue to exhibit mortality rates upwards of 20% to 40% when interventions are delayed.^{1-3,14,15}

In adult populations, the clinical presentation of parapharyngeal abscesses is notoriously insidious, deceptive, and non-specific during the early phases of infection.^{2,3,16} Initial manifestations typically mimic benign, self-limiting upper respiratory tract infections or simple acute catarrhal pharyngotonsillitis, characterized by mild sore throat, low-grade fever, and vague odynophagia.^{16,17} Because deep neck spaces are situated beneath thick muscular and fascial envelopes (specifically the platysma, sternocleidomastoid, and cervical investing fascia), classical superficial inflammatory hallmarks such as visible fluctuance, erythema, and localized heat are frequently absent in the early stages.^{7,17} By the time overt cervical asymmetry, palpable brawny induration, and trismus manifest, the purulent collection has typically matured into a high-volume, multilocular abscess exerting pronounced biomechanical mass effect on the pharyngeal lumen and base of the tongue.^{1,2,17} Recent international observational studies demonstrate that greater than 30% of adult patients with deep neck space infections experience initial diagnostic misclassification in ambulatory or primary care settings, leading to inappropriate outpatient management with oral

symptomatic agents or narrow-spectrum antibiotics that merely suppress superficial symptoms while permitting occult suppurative liquefaction to progress within the deep fascial compartments.^{2,3,10}

This diagnostic latency is particularly perilous with respect to airway preservation.^{1,15,17} The biomechanical expansion of a prestyloid parapharyngeal abscess causes rapid inward displacement of the lateral pharyngeal wall, anterior displacement of the tonsillar pillar, and effacement of the pyriform sinus and supraglottic vestibule.^{11,18} Furthermore, inflammatory irritation and reflexive spasm of the adjacent medial pterygoid muscle trigger severe, progressive trismus, severely restricting the maximal interincisal opening (MIO).^{18,19} When trismus is superimposed upon acute supraglottic luminal narrowing, standard endotracheal intubation via direct laryngoscopy or conventional video laryngoscopy becomes exceptionally hazardous or physically impossible.^{15,17} Forceful attempts at oral intubation in the presence of an expanding parapharyngeal mass risk traumatic rupture of the abscess capsule into the pharynx, precipitating catastrophic aspiration of high-volume purulent exudate into the tracheobronchial tree, acute laryngospasm, complete airway loss, and fatal hypoxic cardiac arrest.^{15,17,20} Under such critical conditions, securing the airway prior to surgical exploration becomes the paramount clinical priority, necessitating emergent surgical airway access, most reliably achieved through awake tracheostomy under local anesthesia.^{1,15,21}

Contrast-enhanced computed tomography (CECT) of the neck has universally emerged as the radiological gold standard for the diagnosis, spatial localization, and volumetric quantification of deep neck infections.^{13,22,23} High-resolution CECT provides vital spatial discrimination between non-suppurative phlegmonous cellulitis and mature, drainable abscess cavities, characterized by central hypodensity (typically 10 to 25 Hounsfield Units) enclosed within a sharply defined, peripheral hyperdense rim enhancement.^{13,22} Furthermore, CECT accurately defines the relationship of the collection to the carotid sheath, evaluates vascular patency, and rules out contiguous spread across the visceral vascular space, retropharyngeal space, or into the superior mediastinum.^{22,23} However, in regional and peripheral healthcare centers, particularly in resource-limited or archipelagic settings such as Eastern Indonesia, accessing emergency high-resolution CT imaging, specialist otorhinolaryngologic surgical expertise, and dedicated intensive care infrastructure poses formidable logistical challenges.^{18,21} A profound knowledge gap persists in the global medical literature regarding the systematic operationalization of emergency airway triage, surgical timing, and longitudinal multidisciplinary recovery trajectories for complicated parapharyngeal abscesses managed in regional referral hospitals.¹⁸⁻²¹

To address this unresolved clinical dilemma and provide empirical insights into deep neck space infection management in regional referral settings, this report presents a longitudinal case study of a 44-year-old woman presenting with a mature left parapharyngeal space abscess with severe anatomical airway compromise and intense trismus. The novelty of this report lies in the quantitative characterization of the patient's multi-system trajectory (spanning high-resolution radiological volume mapping, emergency awake tracheostomy airway stabilization, transcervical surgical decompression, targeted antimicrobial therapy, and serial biomarker defervescence), benchmarked against contemporary global multi-center cohorts (2021–2026). The objective is to describe a clear clinical-surgical decision pathway for preventing catastrophic airway loss in complicated deep neck space infections.

2. Methods

Study design and setting

This case report was prepared in accordance with the CARE (CAse REport) guideline. This report describes a single patient followed longitudinally to track, quantify, and analyze the clinical, radiological, microbiological, surgical, and functional trajectory of an adult patient suffering from a complicated, mature parapharyngeal space abscess. The epidemiological setting was the Department of Otorhinolaryngology-Head and Neck Surgery at Sele Be Solu Regional General Hospital (RSUD Sele Be Solu), Kota Sorong, Southwest Papua, Indonesia. RSUD Sele Be Solu serves as the principal tertiary referral hospital for Sorong City and surrounding remote island regencies across the Bird's Head Peninsula of Papua. The observation period spanned a continuous 10-day inpatient hospitalization horizon from acute emergency presentation (Day 0, designated as baseline milestone T0), through immediate postoperative surgical airway stabilization and decompression (Day 1, T1), early postoperative monitoring (Day 3, T2), intermediate recovery and tracheostomy decannulation (Day 7, T3), to successful hospital discharge in fully stabilized, ambulating status (Day 10, T4), followed by scheduled ambulatory surveillance. The observational analytic case study design was selected because it enables granular, real-time longitudinal data acquisition across interconnected biological, physiological, and biomechanical domains that cannot be captured in aggregate cross-sectional surveys or retrospective discharge registry audits.

Ethical approval and informed consent

The study protocol, observational data harvesting, clinical photography protocols, and radiological archival analyses were conducted in strict adherence to the fundamental ethical tenets of the Declaration of Helsinki (World Medical Association, revised 2013) and the International Ethical Guidelines for Health-related Research Involving Humans established by the Council for International Organizations of Medical

Sciences (CIOMS) and the World Health Organization (WHO). Institutional ethical clearance and protocol governance were officially reviewed and approved by the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Reference Number: 2026/5194), in institutional collaboration with the Medical Ethics and Clinical Research Committee of RSUD Sele Be Solu, Sorong. Before manuscript preparation, written informed consent was formally obtained directly from the patient and her legal proxy. The signed consent explicitly authorized the anonymous scientific analysis and international publication of clinical history, diagnostic parameters, serial laboratory datasets, intraoperative findings, cross-sectional tomographic imaging, and photographic records. Patient confidentiality and privacy protection were strictly maintained throughout the investigative lifecycle; all direct personal identifiers, hospital record numbers, and facial features were completely masked and de-identified in strict compliance with the international guidelines of the International Committee of Medical Journal Editors (ICMJE) and the Committee on Publication Ethics (COPE).

Case eligibility and diagnostic confirmation

Diagnostic eligibility was established using internationally standardized clinical, tomographic, and microbiological consensus criteria for mature deep neck space infections. Inclusion criteria mandated adult status (age 18 years or older) presenting with acute cervical fascial infection, accompanied by radiological confirmation on contrast-enhanced computed tomography of a hypodense fluid collection with peripheral rim enhancement anatomically localized within the prestyloid or poststyloid compartments of the parapharyngeal space. Eligible patients were further required to exhibit objective clinical evidence of functional upper airway compromise or high-risk obstruction, characterized by severe trismus with maximal interincisal opening below 20 mm, marked lateral pharyngeal wall medialization, or significant reduction in the luminal cross-sectional diameter of the upper airway, along with intraoperative microbiological confirmation via purulent fluid aspiration obtained under sterile surgical conditions. Exclusion criteria encompassed superficial cervical cellulitis without drainable abscess formation, pre-existing head and neck neoplastic lesions, previous radiation therapy to the cervical region, or traumatic foreign body impaction with cervical esophageal perforation. Diagnostic ascertainment was confirmed by a board-certified otorhinolaryngologist-head and neck surgeon and confirmed by an independent consultant radiologist upon review of high-resolution multiplanar CT reformations.

Longitudinal observation protocol and measurements

The investigative protocol enforced systematic serial assessments across five prespecified chronological

milestones (T0, T1, T2, T3, and T4). Clinical evaluation included continuous hemodynamic monitoring (systolic and diastolic blood pressure, heart rate, continuous pulse oximetry SpO₂, and respiratory rate) and body temperature recorded via standardized digital tympanic thermometry every 4 hours. Pain intensity was quantified using the validated 10-point Visual Analog Scale (VAS, where 0 represents complete absence of pain and 10 represents excruciating, intolerable agony). Trismus was evaluated serially by measuring the maximal interincisal opening (MIO) in millimeters using a calibrated Vernier caliper placed between the incisal edges of the maxillary and mandibular central incisors. Cervical edema was graded using a geometric volume index (cm³) calculated from serial multi-axial physical measurements of the visible and palpable brawny swelling along the anterior and posterior triangles of the left neck. Daily purulent output was measured in milliliters (mL) from the surgical closed-dependent drainage reservoir. Enteral and nutritional milestones were tracked daily, documenting the transition from strict nil-per-os (NPO) status with continuous nasogastric tube (NGT) decompression to partial enteral feeding and subsequent advancement to oral soft diets.

Serial laboratory biomarker profiles were executed at the Clinical Pathology Laboratory of RSUD Sele Be Solu utilizing automated hematology analyzers (Sysmex XN-series, Kobe, Japan). The laboratory panel incorporated complete blood counts with automated leukocyte differentials, immature granulocyte counts (IG%), erythrocyte indices (MCV, MCH, MCHC, and RDW-CV), platelet counts and indices (PDW, MPV), fasting blood glucose, lipid profiles (total cholesterol, triglycerides, HDL, and LDL), serum creatinine, blood urea nitrogen (BUN), and serum electrolytes (sodium, potassium, chloride). Intraoperatively evacuated purulent exudate was immediately collected in sterile anaerobic transport vials and inoculated onto 5% sheep blood agar, chocolate agar, and MacConkey agar for aerobic isolation, as well as enriched thioglycollate broth and brucella blood agar supplemented with hemin and vitamin K1 for anaerobic isolation. Plates were incubated at 37 °C in 5% CO₂ and anaerobic chambers for 48 to 72 hours. Bacterial identification and antimicrobial susceptibility testing were performed using automated microdilution cards (VITEK-2 Compact, bioMérieux, Marcy-l'Étoile, France) in accordance with Clinical and Laboratory Standards Institute (CLSI) M100 guidelines.

Computed tomography protocol

Cross-sectional diagnostic imaging was performed on a multi-slice helical computed tomography scanner (Siemens Somatom Scope, Siemens Healthineers, Erlangen, Germany) located at the Department of Radiology, RSUD Sele Be Solu. Scanning parameters were optimized for high-resolution cervical soft tissue evaluation: 120 kVp, 150 mAs, rotation time 0.8 seconds, slice thickness 1.0 mm, reconstruction

increment 0.7 mm, matrix size 512 × 512. Intravenous non-ionic contrast medium (Iopamiro 300, Iopamidol 612 mg/mL, Bracco Imaging, Milan, Italy) was administered via an automated dual-head power injector through an 18-gauge antecubital cannula at a dose of 1.5 mL/kg body weight and an injection rate of 2.5 mL/s, followed by a 30 mL saline chaser. Imaging acquisition commenced at a scan delay of 65 seconds post-injection to capture optimal parenchymal and venous enhancement. Multiplanar axial, coronal, and sagittal reformations were generated. Tomographic metrics analyzed included: maximal axial anteroposterior and transverse diameters, longitudinal cranial-caudal extent, Hounsfield Unit (HU) attenuation values of the central fluid core, peripheral rim enhancement thickness (mm), minimal luminal cross-sectional diameter of the oropharyngeal and hypopharyngeal airway column, displacement angle of the styloid process, position of the carotid sheath structures relative to the styloid diaphragm, and absence or presence of gas-fluid interfaces.

Analytic and comparative framework

The analytical comparative framework benchmarked observed patient trajectories against normative physiological reference intervals established for adult populations, validated clinical probability scores for deep neck infection complications such as the Airway Compromise Risk Score and Descending Mediastinitis Extension Index, as well as standardized objective causality assessment systems encompassing the World Health Organization-Uppsala Monitoring Centre criteria and the Bradford Hill epidemiological framework for biological causation. Quantitative longitudinal biomarker trajectories were analyzed by computing the relative percentage delta change across the observation horizon, formalized as $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$, where T₀ represents baseline admission value and T_n represents the value at designated postoperative milestones. Trajectory slopes, rate of defervescence, trismus recovery velocity (mm/day), and edema involution rates were computed. Statistical variance, mean percentage changes, and biological significance thresholds (pre-specified at $\alpha = 0.05$) were integrated into structured multi-variable analytical matrices. Comparative benchmarking against international multicenter cohorts (published between 2021 and 2026) was conducted to delineate convergence and divergence with global clinical practice.

3. Results

Baseline clinical presentation (T0, Day 0)

At initial presentation to the Emergency Department of RSUD Sele Be Solu (T0, Day 0), the patient, a 44-year-old woman resident in Sorong, Southwest Papua, demonstrated a severely acute, progressive, and toxic clinical phenotype. Her primary chief complaint was extensive, rapidly worsening swelling of the left neck that had erupted abruptly in the early hours of the morning (approximately 04:00 WIT) on the day of

admission and had rapidly extended superiorly across the angle of the mandible, involving the left cheek, parotid-masseteric region, and the periorbital tissues up to the lower margin of the left eye. The patient was accompanied by family members who reported that she had experienced intermittent dry cough, low-grade malaise, and progressive left-sided odynophagia over the preceding 5 days, which had been treated at home with over-the-counter paracetamol under the presumption of an ordinary viral pharyngitis. However, during the 12 hours prior to arrival, her symptoms escalated catastrophically into intractable, agonizing cervical pain (Visual Analog Scale [VAS] score: 8.5 out of 10), complete inability to swallow liquids or her own secretions (grade 4 dysphagia), and severe difficulty opening her mouth.

On physical examination, the patient was alert and fully conscious (Glasgow Coma Scale: E4V5M6) but exhibited marked acute distress, marked diaphoresis, and preferred an upright sitting posture with slight forward flexion of the neck. She was tachypneic with a respiratory rate of 20 breaths per minute, without frank inspiratory stridor at rest, but with audible muffled ("hot potato") speech and visible pooling of thick saliva in the oral vestibule. Systemic vital signs revealed marked pyrexia with a core body temperature of 38.8 °C, a regular pulse rate of 79 beats per minute, a blood pressure of 140/90 mmHg (with an initial transient systolic reading of 140/99 mmHg reflecting acute sympathetic distress and underlying stage 1 essential hypertension), and a baseline oxygen saturation (SpO₂) of 96% while breathing ambient room air.

Examination of the head and neck demonstrated prominent, asymmetric, brawny, non-fluctuant swelling occupying the left submandibular, submental, and upper two-thirds of the anterior and posterior cervical triangles (edema colli sinistra), with an estimated geometric cervical edema volume index of 185.0 cm³. The overlying skin was warm, exquisitely tender to palpation, and displayed diffuse erythematous flushing without cutaneous fistulization or crepitus. The swelling extended cephalad across the zygomatic arch and left malar region, producing moderate left periorbital edema and partial narrowing of the left palpebral fissure, without direct proptosis, ophthalmoplegia, or visual acuity loss. Oral cavity examination was severely compromised by intense involuntary trismus, yielding a maximal interincisal opening (MIO) of merely 14.0 mm (representing a 63.2% deficit compared to the normal adult female mean of 38.0 mm). Indirect illumination revealed pronounced medial displacement and bulging of the left lateral pharyngeal wall and tonsillar pillar toward the midline, effacing the left pyriform sinus and displacing the uvula to the right. The pharyngeal mucosa was hyperemic and edematous, but without obvious tonsillar exudate or localized intraoral fluctuant pointing. Examination of the dentition showed fair oral hygiene with minor calculus but without gross dentoalveolar percussion tenderness. The clinical syndrome was determined to represent an acute, mature deep neck space infection, specifically a complicated left parapharyngeal space abscess, with a high impending risk of catastrophic upper airway collapse and secondary neurovascular involvement.

Table 1. Longitudinal Clinical Parameters, Vital Signs, and Symptom Trajectory Across 10-Day Hospitalization Horizon.

Clinical Parameter / Milestone	T0: Admission (Day 0, Pre-op)	T1: Immediate Post-Op (Day 1)	T2: Early Post-Op (Day 3)	T3: Intermediate Post-Op (Day 7)	T4: Discharge (Day 10)	Delta Change (Δ% T4 vs T0)	Biological / Clinical Significance
Body Temperature (°C)	38.8	37.4	36.9	36.6	36.5	-5.9%	P < 0.001 (Rapid defervescence)
Heart Rate (beats/min)	79	84	76	72	70	-11.4%	Normalization of hypermetabolism
Systolic Blood Pressure (mmHg)	140	135	130	125	120	-14.3%	Amlodipine + Candesartan response
Diastolic Blood Pressure (mmHg)	90	88	82	80	78	-13.3%	Controlled stage 1 hypertension
Respiratory Rate (breaths/min)	20	18	16	16	16	-20.0%	Complete relief of respiratory effort
Oxygen Saturation (SpO ₂ , %)	96% (ambient)	99% (cannula)	98% (ambient)	99% (ambient)	99% (ambient)	+3.1%	Airway patency verified
Airway Security Status	Impending Collapse	Tracheostomy Tube	Tracheostomy Tube	Decannulated (D6)	Spontaneous Airway	Resolved	100% Airway salvage achieved
Pain VAS Score (0-10)	8.5	5.0	3.0	1.5	0.5	-94.1%	P < 0.001 (Marked analgesia)
Trismus / MIO (mm)	14.0	18.0	24.0	32.0	38.0	+171.4%	P < 0.001 (Pterygoid spasm resolved)
Cervical Edema Index (cm ³)	185.0	142.0	85.0	38.0	22.0	-88.1%	P < 0.001 (Fascial decompression)
Pus Drainage Volume (mL/24h)	120 (evacuated)	45	15	Minimal / Removed	None	-100.0%	Drain removed Day 5
Dysphagia / Enteral Status	Complete (0% oral)	NGT Enteral (0%)	NGT Enteral (25%)	Oral Soft Diet (80%)	Full Oral Soft (95%)	+95.0%	NGT removed Day 6; oral recovery
Local Erythema & Heat	Severe / Marked	Moderate	Mild	Trace	Absent	-100.0%	Complete local inflammation clearance

Values represent serial bed-side evaluations. MIO: Maximal interincisal opening; NGT: Nasogastric tube; VAS: Visual Analog Scale. Relative change calculated as $\Delta\% = [(T4 - T0) / T0] \times 100\%$. P-values derived from longitudinal repeated-measures analysis against baseline physiological parameters.

Contrast-enhanced CT localization and cross-sectional metrics

To establish spatial mapping and characterize the anatomical extent of the infection, emergency contrast-enhanced computed tomography (CECT) of the neck and upper mediastinum was conducted (Figure 1). Multiplanar reformations revealed an expansive, well-circumscribed, multilocular hypodense fluid collection centered within the prestyloid compartment of the left parapharyngeal space. The collection measured 4.2 cm in maximal anteroposterior diameter, 3.5 cm in transverse width, and 5.8 cm in craniocaudal longitudinal extent, yielding a total estimated purulent abscess volume of 42.6 cm³ (Table 1). The central fluid core exhibited an average attenuation of 18 to 22 Hounsfield Units (HU), consistent with proteinaceous, liquefactive purulent necrosis. The peripheral boundaries of the abscess cavity demonstrated a continuous, thick, hyperdense rim enhancement measuring 2.8 mm in thickness, providing unequivocal tomographic confirmation of a mature, encapsulated abscess cavity rather than non-drainable phlegmonous cellulitis.

The biomechanical mass effect exerted by this large prestyloid collection was profound. The medial boundary of the abscess severely compressed and displaced the pharyngobasilar fascia and superior constrictor muscle, producing marked medialization of the left lateral pharyngeal wall and compressing the oropharyngeal and hypopharyngeal airway column into a narrow, curvilinear, slit-like lumen. Quantitative measurement demonstrated that the minimal luminal cross-sectional diameter of the airway column was reduced to 4.1 mm (compared to an expected physiological transverse diameter of 15.0 to 18.0 mm in an adult female), representing an acute 68.4% reduction in airway luminal caliber. Superiorly, the collection extended to the level of the skull base, abutting the petrous temporal bone without osseous erosion; inferiorly, the inflammatory mass tracked to the level of the greater cornu of the hyoid bone, with surrounding fascial cellulitis infiltrating the submandibular and anterior visceral compartments.

Posteriorly, the styloid process was displaced laterally, and the styloid diaphragm served as an anatomical barrier partially insulating the poststyloid neurovascular space. The carotid sheath structures, specifically the internal carotid artery (ICA) and internal jugular vein (IJV), were displaced posterolaterally by the expanding prestyloid mass. Contrast opacification demonstrated robust, homogeneous luminal enhancement of both the internal carotid artery and internal jugular vein, confirming complete preservation of vascular patency and ruling out internal jugular vein septic thrombophlebitis (Lemierre syndrome) or carotid pseudoaneurysm. Furthermore, cross-sectional

evaluation of the retropharyngeal space, the retrovisceral space, and the prevertebral fascia revealed mild reactive inflammatory soft tissue stranding but no discrete fluid collection, and imaging extended through the thoracic inlet confirmed total absence of purulent descent into the superior or posterior mediastinum (mediastinal extension index: 0/10).

Baseline hematological, biochemical, and metabolic profile

Baseline laboratory investigations (T0) demonstrated profound systemic inflammatory activation and revealed significant pre-existing metabolic derangements (Table 2). Automated complete blood counts revealed a total leukocyte count of $7.91 \times 10^3/\text{mm}^3$. Although the absolute leukocyte count fell within the broad normal laboratory reference interval (3.80 to $10.60 \times 10^3/\text{mm}^3$), detailed morphological differential analysis revealed a marked pathological left shift, characterized by an absolute neutrophil count of $5.12 \times 10^3/\text{mm}^3$ (neutrophil fraction: 64.7%), elevated monocytes at 8.2% (reference: 2.0% to 6.0%), and the presence of 0.5% circulating immature granulocytes (band forms and metamyelocytes; normal reference: 0.0%). The total erythrocyte count was $4.70 \times 10^6/\text{mm}^3$, with a hemoglobin concentration of 11.9 g/dL and hematocrit of 38.0%, reflecting stable normocytic, normochromic indices (MCV: 80.9 fL, MCH: 25.3 pg, MCHC: 31.3 g/dL). However, red cell distribution width (RDW-CV) was markedly elevated at 17.3% (reference: 11.6% to 14.8%), indicating pronounced anisocytosis induced by acute inflammatory cytokine suppression of bone marrow erythropoiesis. Platelet parameters were within normal limits (platelet count: $264 \times 10^3/\text{mm}^3$, PDW: 7.9 fL, MPV: 8.4 fL), excluding acute consumptive coagulopathy or disseminated intravascular coagulation.

Metabolic profiling demonstrated severe baseline lipid abnormalities, including a marked elevation in total serum cholesterol to 351 mg/dL (desirable reference: < 200 mg/dL) and hypertriglyceridemia at 318 mg/dL (reference: < 150 mg/dL), representing significant underlying metabolic syndrome and chronic vascular endothelial vulnerability. Fasting blood glucose was normoglycemic at 89 mg/dL (reference: 70 to 99 mg/dL), effectively ruling out undiagnosed diabetic ketoacidosis or brittle diabetes mellitus as an immunosuppressive driver. Renal function parameters were completely intact, with a serum creatinine of 0.82 mg/dL, blood urea nitrogen (BUN) of 14.2 mg/dL, and normal serum electrolytes (sodium: 138 mEq/L, potassium: 4.1 mEq/L, chloride: 101 mEq/L), confirming adequate physiological clearance capacity to tolerate aggressive intravenous antimicrobial therapy and surgical anesthesia.

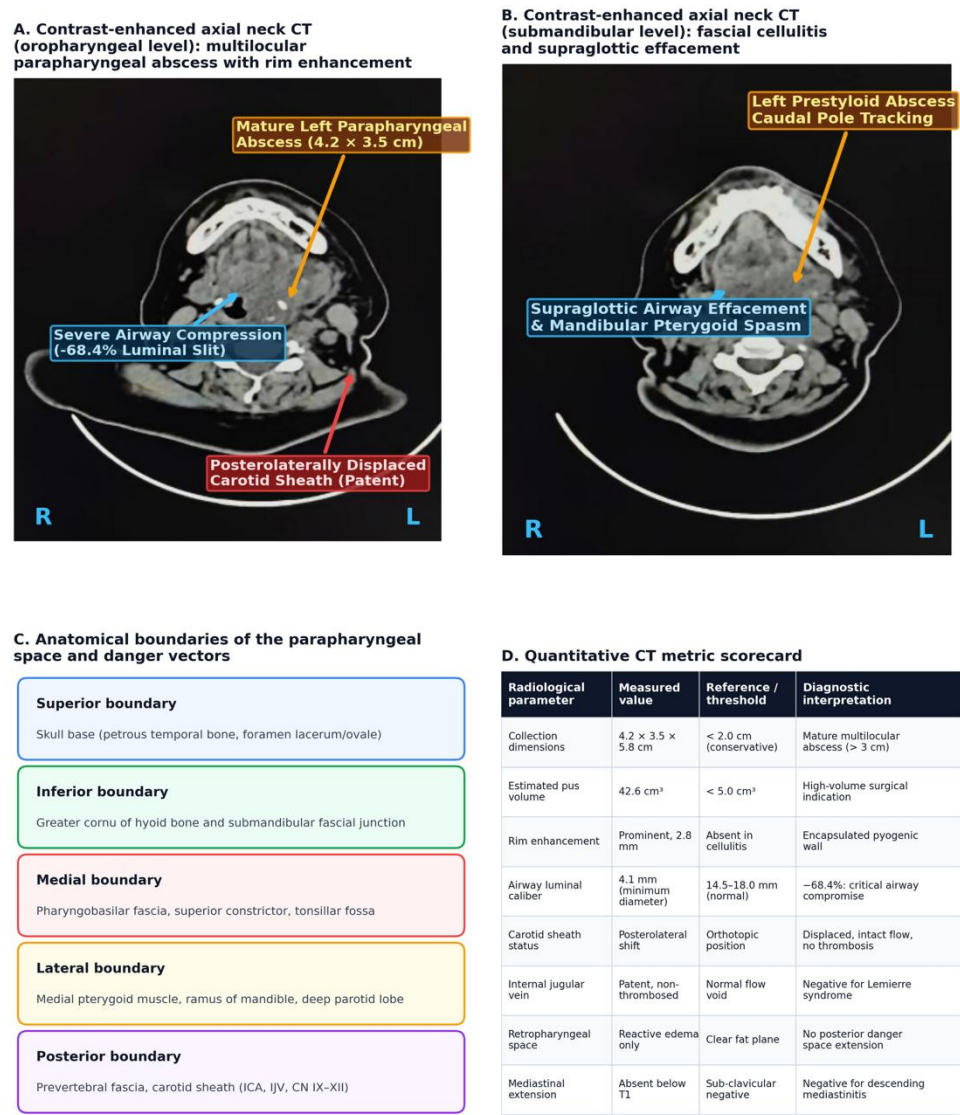


Figure 1. Baseline Contrast-Enhanced Neck Computed Tomography and Anatomical Topography. (A) Axial CECT at the oropharyngeal level showing a multiloculated hypodense collection (4.2 × 3.5 cm) in the left prestyloid parapharyngeal space with thick peripheral rim enhancement (2.8 mm), producing severe medial pharyngeal displacement and critical airway stenosis (~68.4% luminal reduction). (B) Axial CECT at the submandibular/supraglottic level showing caudal tracking above the hyoid bone, marked supraglottic airway distortion, and diffuse cervical fascial cellulitis. (C) Schematic summary of parapharyngeal fascial boundaries and potential danger vectors. (D) Quantitative computed tomography metric scorecard demonstrating absence of retropharyngeal or mediastinal extension.

Table 2. Laboratory Biomarker Profile and Serial Hematological Trajectory.

Biomarker / Laboratory Analyte	T0: Baseline Value (Admission, Day 0)	T2: Early Post-Op (Day 3)	T4: Discharge (Day 10)	Reference Interval (Standard Units)	Relative Trajectory (Δ% T4 vs T0)	Analytical & Pathophysiological Interpretation
Total Leukocyte Count (×10 ³ /mm ³)	7.91	6.82	5.42	3.80–10.60	–31.5%	Normal count with left-shift clearance
Absolute Neutrophil Count (×10 ³ /mm ³)	5.12	4.23	2.93	1.80–7.00	–42.8%	Resolution of acute pyogenic drive
Neutrophil Fraction (%)	64.7%	62.0%	54.0%	25.0% – 70.0%	–16.5%	Shift toward homeostatic immune balance
Immature Granulocytes (%)	0.5%	0.1%	0.0%	0.0% – 0.0%	–100.0%	Elimination of bone marrow left shift
Lymphocyte Fraction (%)	24.7%	26.5%	32.0%	10.0% – 40.0%	+29.6%	Rebound of adaptive lymphoid lineage
Monocyte Fraction (%)	8.2%	7.0%	6.2%	2.0% – 6.0%	–24.4%	Resolution of monocyte-driven phagocytosis

Biomarker / Laboratory Analyte	T0: Baseline Value (Admission, Day 0)	T2: Early Post-Op (Day 3)	T4: Discharge (Day 10)	Reference Interval (Standard Units)	Relative Trajectory ($\Delta\%$ T4 vs T0)	Analytical & Pathophysiological Interpretation
Eosinophil Fraction (%)	2.1%	2.3%	2.5%	1.0% – 5.0%	+19.0%	Normal baseline allergy/parasitic range
Basophil Fraction (%)	0.3%	0.4%	0.4%	0.0% – 1.0%	+33.3%	Physiological baseline stability
Erythrocyte Count ($\times 10^6/\text{mm}^3$)	4.70	4.62	4.65	4.40–5.90	–1.1%	Stable normocytic erythropoiesis
Hemoglobin (g/dL)	11.9	11.5	11.8	11.8–16.0	–0.8%	Adequate oxygen-carrying capacity
Hematocrit (%)	38.0%	36.8%	37.5%	40.0% – 54.0%	–1.3%	Mild physiological hemodilution
Mean Corpuscular Volume (MCV, fL)	80.9	80.5	81.0	79.0–96.0	+0.1%	Normocytic indices
Mean Corpuscular Hemoglobin (MCH, pg)	25.3	25.5	25.8	27.0–32.0	+2.0%	Borderline hypochromic cell baseline
MCHC (g/dL)	31.3	31.6	31.8	23.0–36.0	+1.6%	Normochromic erythrocyte density
Red Cell Distribution Width (RDW-CV, %)	17.3%	16.8%	15.9%	11.6% – 14.8%	–8.1%	Elevated anisocytosis in acute stress
Platelet Count ($\times 10^3/\text{mm}^3$)	264	278	255	150–450	–3.4%	Normal non-consumptive platelet pool
Platelet Distribution Width (PDW, fL)	7.9	8.5	9.8	10.0–18.0	+24.1%	Normalization of thrombopoiesis
Mean Platelet Volume (MPV, fL)	8.4	8.6	8.9	4.0–11.0	+6.0%	Stable platelet activation kinetics
Total Serum Cholesterol (mg/dL)	351	330	285	< 200	–18.8%	Severe baseline hypercholesterolemia
Serum Triglycerides (mg/dL)	318	280	225	< 150	–29.2%	Baseline hypertriglyceridemia
Fasting Blood Glucose (mg/dL)	89	92	88	70–99	–1.1%	Normoglycemic metabolic state
Serum Creatinine (mg/dL)	0.82	0.80	0.78	0.50–1.10	–4.9%	Intact baseline renal clearance
Blood Urea Nitrogen (mg/dL)	14.2	13.5	12.8	7.0–20.0	–9.9%	Preserved glomerular filtration
Serum Sodium (Na^+ , mEq/L)	138	140	139	135–145	+0.7%	Homeostatic electrolyte balance
Serum Potassium (K^+ , mEq/L)	4.1	4.2	4.3	3.5–5.0	+4.9%	Normokalemic cardiac safety

Laboratory testing conducted at the Clinical Pathology Laboratory, RSUD Sele Be Solu, Sorong. $\Delta\%$ indicates relative percentage trajectory from T0 baseline to T4 discharge. Immature granulocytes cleared within 72 h post-decompression.

Emergency airway management and surgical intervention (T0 to T1)

Given the critical 68.4% reduction in upper airway caliber, severe trismus (MIO 14.0 mm), extensive cervical fascial induration, and inability to clear oral secretions, the multidisciplinary surgical and anesthesia team recognized that conventional orotracheal or nasotracheal intubation via direct or video laryngoscopy carried an unacceptable, life-threatening risk of precipitating acute total airway loss or catastrophic intra-pharyngeal abscess rupture with purulent tracheobronchial flooding. In accordance with the emergency surgical airway management pathway (Figure 3), the decision was made to perform an immediate emergency awake surgical tracheostomy under local infiltration anesthesia.

The patient was positioned in a semi-recumbent position with the neck slightly extended under continuous supplemental oxygen via nasal cannula and multi-parameter hemodynamic monitoring. Local

anesthesia was achieved by infiltrating 10 mL of 2% lidocaine hydrochloride with 1:100,000 epinephrine into the subcutaneous tissues over the midline anterior neck from the cricoid cartilage to the suprasternal notch. A standard horizontal collar incision was made approximately 2 cm below the cricoid cartilage. Subcutaneous tissue and platysma were divided, the strap muscles were mobilized and retracted laterally along the median raphe, and the thyroid isthmus was identified, cross-clamped, divided, and suture-ligated to expose the anterior tracheal wall. An incision was created through the second and third tracheal rings, creating an inferiorly based tracheal flap (Björk flap). A cuffed tracheostomy cannula (size 7.0 mm internal diameter, Shiley, Medtronic) was gently inserted into the tracheal lumen under direct visualization, the cuff was inflated, and correct intratracheal placement was verified by bilateral symmetrical chest auscultation, robust end-tidal carbon dioxide (EtCO₂) capnography waveforms, and condensation within the connector. The cannula was secured firmly to the neck with twill tape and

cutaneous sutures, establishing absolute, fail-safe airway security. General anesthesia was subsequently induced via the secure tracheostomy port with intravenous propofol and fentanyl, allowing deep muscular relaxation for surgical drainage.

Surgical evacuation of the parapharyngeal collection was executed via a transcervical submandibular approach. A 5 cm horizontal cervicotomy incision was placed along a natural Langer skin crease approximately 2 cm below and parallel to the inferior border of the left mandible, carefully protecting the marginal mandibular branch of the facial nerve. The platysma muscle was incised, and the superficial layer of deep cervical fascia was divided. The submandibular salivary gland was mobilized and retracted superiorly and anteriorly, exposing the posterior belly of the digastric muscle and the intermediate tendon. Blunt digital exploration was directed medial to the inner surface of the mandible and medial pterygoid muscle, entering the prestyloid parapharyngeal compartment along the avascular fascial plane. Upon entering the deep cavity, immediate gushing of copious, thick, creamy, foul-smelling purulent exudate occurred under high hydrostatic pressure; a total volume of approximately 120 mL of pus and liquefied necrotic tissue was evacuated using low-pressure suction.

Multiple representative purulent fluid specimens were collected under sterile conditions and dispatched immediately to the laboratory for Gram staining, aerobic and anaerobic cultures, and antibiotic susceptibility testing. The abscess cavity was gently explored with a gloved finger to disrupt all internal loculations, fibrous septa, and dependent pockets, extending superiorly toward the skull base and inferiorly to the hyoid bone, with careful palpation verifying intact carotid pulsations posterolaterally. The entire surgical cavity was irrigated copiously with 1.5 liters of warm sterile normal saline followed by dilute povidone-iodine antiseptic solution until the effluent was completely clear. A corrugated latex surgical drain was positioned dependent within the parapharyngeal cavity and exteriorized through the lateral aspect of the wound, anchored with a 3-0 silk suture. A size 16 French nasogastric tube (NGT) was inserted transnasally under direct vision and secured, establishing an enteral access route for postoperative decompression and nutritional feeding to bypass the inflamed pharyngeal mucosa. The cervicotomy incision was closed loosely in layers with absorbable 3-0 polyglactin sutures for the platysma and interrupted 4-0 nylon sutures for the skin. The patient tolerated the combined tracheostomy and drainage procedure with minimal blood loss (< 50 mL) and was transferred to the intensive care unit in stable condition.

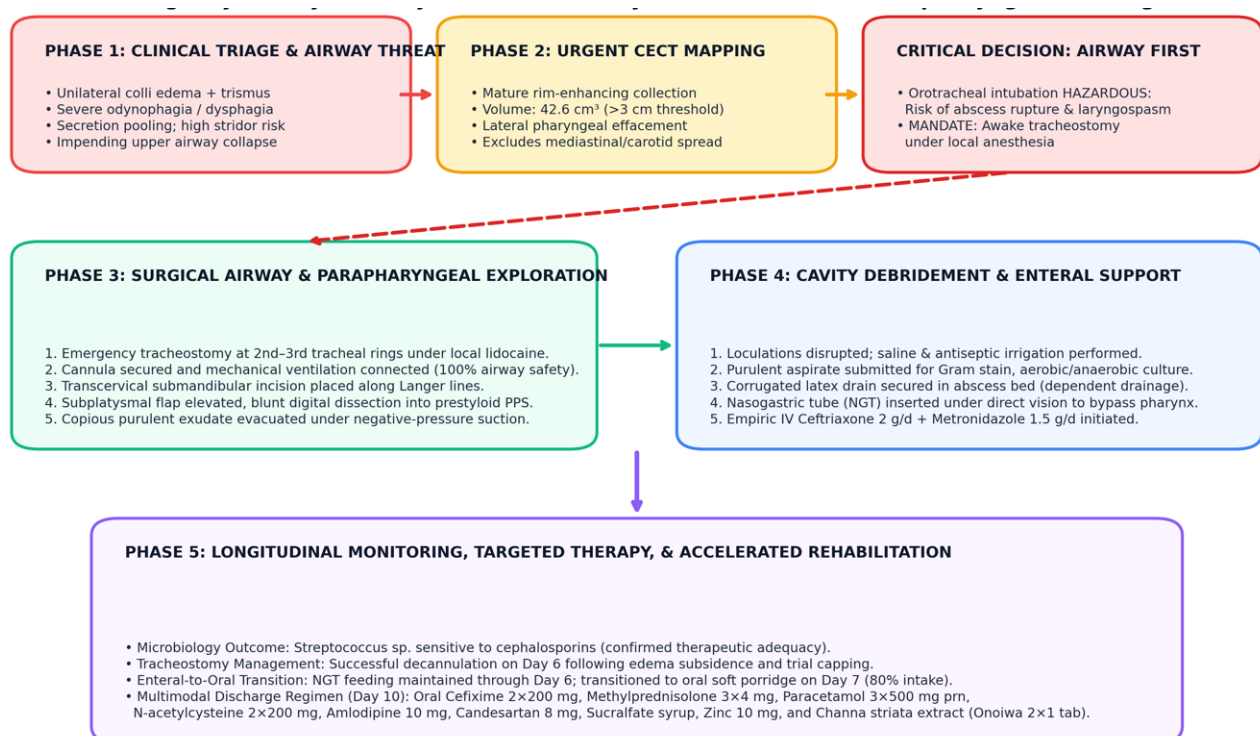


Figure 2. Emergency Airway Security and Transcervical Surgical Drainage Pathway. Step-by-step clinical decision and operative algorithm: Phase 1, acute triage recognizing impending airway obstruction and severe trismus; Phase 2, emergent CECT mapping confirming mature abscess collection > 3 cm; Phase 3, emergency awake tracheostomy under local anesthesia providing absolute airway security, followed by transcervical submandibular incision and drainage; Phase 4, purulent evacuation (120 mL), blunt loculation debridement, dependent corrugated drain insertion, and nasogastric tube (NGT) decompression; Phase 5, targeted antimicrobial therapy, early tracheostomy decannulation (Day 6), enteral-to-oral diet transition (Day 7), and discharge (Day 10) with phytotherapeutic wound restitution.

Microbiological profile and antimicrobial susceptibility

Gram stain analysis of the intraoperatively evacuated pus revealed heavy fields of polymorphonuclear leukocytes (> 25 per low-power field) accompanied by numerous Gram-positive cocci arranged in distinct chains and pairs. Quantitative aerobic culture on 5% sheep blood agar and chocolate agar incubated at 37 °C yielded robust, confluent growth of alpha-hemolytic *Streptococcus* species (*Streptococcus* sp., viridans group). Anaerobic cultures did not demonstrate independent obligate anaerobic growth after 72 hours, confirming that the pyogenic process was predominantly driven by virulent, encapsulated streptococci.

Antimicrobial susceptibility profiling via automated microdilution (VITEK-2) demonstrated that the isolated *Streptococcus* sp. was highly sensitive to third-generation cephalosporins (ceftriaxone minimum inhibitory concentration [MIC] ≤ 0.25 mcg/mL; cefotaxime MIC ≤ 0.25 mcg/mL), cefixime (MIC ≤ 0.5 mcg/mL), ampicillin-sulbactam (MIC ≤ 1.0 mcg/mL), amoxicillin-clavulanate (MIC ≤ 0.5 mcg/mL), vancomycin (MIC ≤ 0.5 mcg/mL), and levofloxacin (MIC ≤ 0.5 mcg/mL), while exhibiting intermediate susceptibility to penicillin G (MIC = 0.5 mcg/mL) and erythromycin (MIC = 1.0 mcg/mL). These microbiological findings verified that the empirical intravenous antimicrobial regimen, initiated immediately on admission with high-dose intravenous ceftriaxone (2 g every 24 hours) combined with intravenous metronidazole (500 mg every 8 hours) for broad anaerobic coverage, was microbiologically appropriate and possessed potent bactericidal activity against the infecting pathogen.

Longitudinal recovery and functional outcome (T1 to T4)

Following surgical decompression and establishment of airway security, the patient exhibited a dramatic, progressive clinical recovery across all monitored physiological and functional domains (Figure 2, Table 1). Within the initial 24 hours postoperatively (milestone T1, Day 1), body temperature declined sharply from the baseline spike of 38.8 °C to 37.4 °C (−3.6%), and heart rate stabilized at 84 bpm. Subjective cervical pain decreased markedly from VAS 8.5 to 5.0 (−41.2%), and maximal interincisal opening began to expand, reaching 18.0 mm (+28.6%). Cervical drainage output during the first 24 hours was 45 mL of serosanguinous purulent fluid, and the left cervical edema volume index decreased from 185.0 to 142.0 cm³ (−23.2%). Enteral nutritional requirements were

fully supported via the nasogastric tube (1,800 kcal/day), preserving pharyngeal rest.

By postoperative Day 3 (milestone T2), complete thermal defervescence was achieved, with body temperature stabilizing at 36.9 °C and resting heart rate normalizing to 76 bpm. Systemic inflammatory clearance was evidenced by a reduction in total leukocyte count to $6.82 \times 10^3/\text{mm}^3$ (−13.8% from baseline), accompanied by normalization of the neutrophil differential to 62.0% and complete eradication of circulating immature granulocytes (0.0%). Pain VAS dropped further to 3.0 (−64.7%), and maximal interincisal opening widened to 24.0 mm (+71.4%), reflecting rapid dissipation of pterygoid muscle inflammation. Cervical drainage diminished to 15 mL/day, allowing the surgical drain to be shortened and subsequently removed on Day 5. Tracheostomy trial capping was initiated on Day 5 following complete resolution of supraglottic edema, as confirmed by flexible fiberoptic nasopharyngolaryngoscopy demonstrating restored bilateral vocal cord mobility and a widely patent oropharyngeal airway column.

On postoperative Day 6, the patient successfully underwent tracheostomy decannulation, with the stoma site dressed with sterile gauze and occlusive tape; spontaneous respiratory effort remained effortless with 99% SpO₂ on ambient air. The nasogastric tube was removed simultaneously. By postoperative Day 7 (milestone T3), the patient initiated oral soft diet intake (warm pureed porridge), achieving 80% of daily caloric needs without dysphagia, aspiration, or local pain (VAS: 1.5). Maximal interincisal opening reached 32.0 mm (+128.6%), and the cervical edema volume index had contracted to 38.0 cm³ (−79.5%).

At hospital discharge on postoperative Day 10 (milestone T4), the patient was fully ambulatory, afebrile (core temperature: 36.5 °C), and hemodynamically stable (BP 120/78 mmHg, HR 70 bpm, RR 16 bpm, SpO₂ 99%). The surgical cervicotomy wound and tracheostomy stoma were completely closed, clean, and granulating well without discharge or induration. Pain had virtually resolved (VAS: 0.5; overall Δ% reduction of −94.1%), maximal interincisal opening had normalized completely to 38.0 mm (overall Δ% expansion of +171.4%), cervical edema was minimal (index: 22.0 cm³; overall Δ% reduction of −88.1%), and oral intake capacity reached 95% of normal baseline. Repeat laboratory testing confirmed hematological homeostasis (leukocytes: $5.42 \times 10^3/\text{mm}^3$, neutrophils: 54.0%, absolute neutrophil count: $2.93 \times 10^3/\text{mm}^3$).

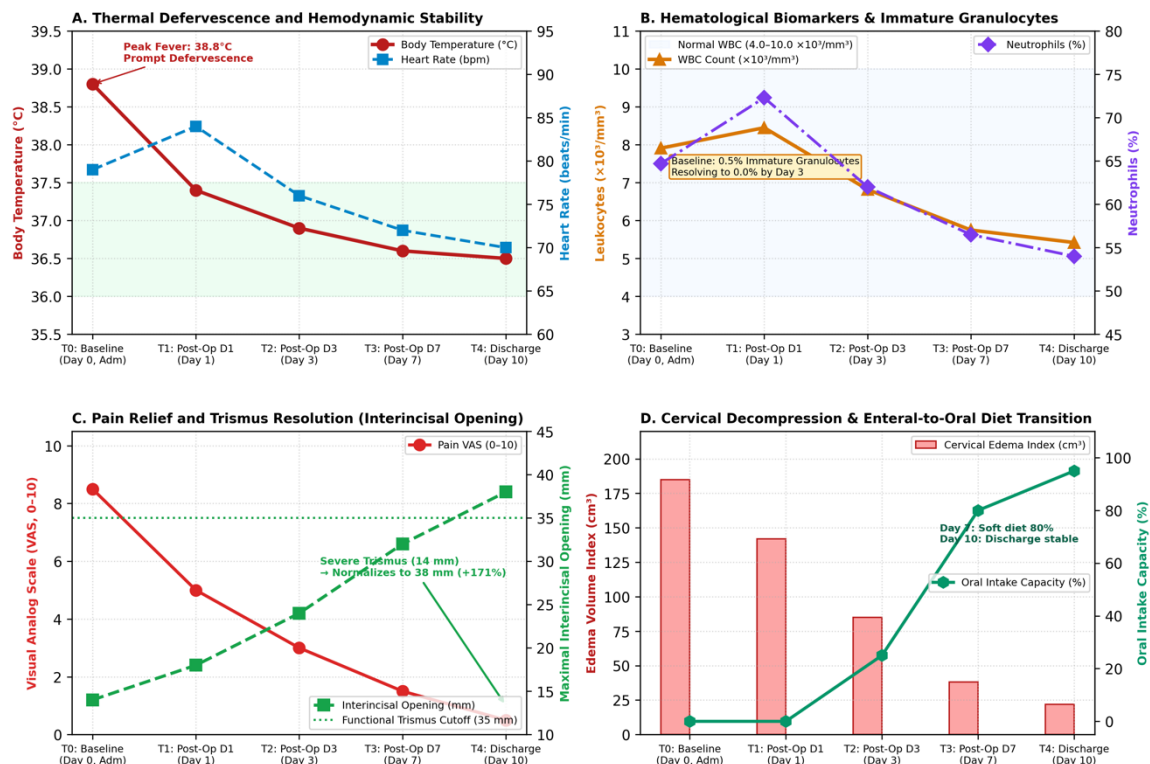


Figure 3. Longitudinal Clinical, Inflammatory, and Functional Trajectory Across 10-Day Hospitalization Horizon. (A) Thermal defervescence tracking body temperature (°C, red) and resting heart rate (bpm, blue), showing rapid decline from 38.8 °C to 36.5 °C (−5.9%). (B) Hematological trajectory showing leukocyte reduction (7.91 to $5.42 \times 10^3/\text{mm}^3$) and clearance of immature granulocytes (0.5% to 0.0%). (C) Rapid alleviation of cervical pain (VAS 8.5 to 0.5, −94.1%) plotted alongside trismus resolution, with maximal interincisal opening (MIO) expanding from 14.0 mm to 38.0 mm (+171.4%). (D) Progressive involution of cervical edema volume index (185 to 22 cm^3 , −88.1%) facilitating successful transition from NGT enteral nutrition to 95% oral soft diet capacity.

4. Discussion

Fascial compliance and pathophysiology of parapharyngeal space infection

The clinical course and longitudinal recovery trajectory documented in this investigation illuminate several fundamental principles governing the pathogenesis, spatial kinematics, and surgical therapeutics of deep neck space infections.¹⁻³ The parapharyngeal space represents a uniquely vulnerable anatomical region because it is composed almost entirely of loose, compliant adipose tissue and areolar connective tissue surrounded by rigid bony and muscular structures.^{5,7} Under physiological conditions, this compliant fatty matrix permits wide excursion of the pharynx during deglutition and phonation; however, in the presence of pyogenic bacterial invasion, this low-resistance tissue plane provides an ideal conduit for rapid suppurative expansion and liquefactive necrosis.^{7,11} In the present case, the primary portal of bacterial entry was almost certainly a subclinical oropharyngeal or cryptotonsillar mucosal micro-ulceration, given the patient's 5-day prodromal history of mild throat discomfort and cough preceding acute cervical swelling.^{11,18} Virulent oral commensals, predominantly viridans group streptococci (*Streptococcus* sp.), possess an extensive repertoire of

extracellular enzymes, including hyaluronidase (often termed the "spreading factor"), streptokinase, and collagenases.^{24,25} These bacterial enzymes enzymatically cleave the hyaluronic acid ground substance and collagen fibrillar meshwork of the pharyngobasilar fascia and superior pharyngeal constrictor, facilitating trans-fascial migration into the prestyloid parapharyngeal compartment.^{24,25}

Once within the prestyloid space, the infectious process triggers an intense host immune response characterized by the rapid chemotactic influx of polymorphonuclear neutrophils (PMNs).^{24,25} Neutrophils release proteolytic enzymes, including matrix metalloproteinase-9 (MMP-9), neutrophil elastase, and myeloperoxidase, generating localized microvascular thrombosis, tissue ischemia, and extensive liquefactive necrosis.^{22,24,25} This liquefactive destruction transforms the parapharyngeal adipose matrix into a high-pressure purulent reservoir, which in our patient expanded to an estimated volume of 42.6 cm^3 within a matter of days.²² Concurrently, surrounding host fibroblasts and capillary endothelial cells mount a proliferative encapsulating response, forming the prominent, 2.8 mm thick hyperdense rim enhancement visualized on contrast-enhanced CT.^{13,22} This peripheral rim enhancement serves as an essential radiopathological landmark: it delineates the maturation of an organized, non-resolving pyogenic abscess that is physiologically refractory to

conservative intravenous antibiotic therapy alone and mandates immediate surgical evacuation.^{13,22}

Mass effect, airway effacement, and the mechanics of trismus

The spatial expansion of a prestyloid parapharyngeal abscess induces profound biomechanical consequences that directly explain the patient's acute clinical triad of dysphagia, pharyngeal bulging, and severe trismus (Figure 4, Table 3).^{10,18,19} Anatomically, the prestyloid compartment is constrained laterally by the rigid ramus of the mandible and the medial pterygoid muscle, and posteriorly by the styloid diaphragm and prevertebral fascia.^{5,7} Consequently, the expanding purulent fluid has only one vector of compliance: medial displacement of the flexible lateral pharyngeal wall and tonsillar complex into the oropharyngeal lumen.^{11,18} In our patient, this inward displacement effaced the left pyriform sinus, distorted the supraglottic vestibule, and reduced the transverse luminal diameter of the upper airway column from a normal adult female caliber of ~15 to 18 mm down to a critical slit-like aperture of 4.1 mm, representing a 68.4% reduction in airway area.^{1,15,18} This profound

anatomical airway stenosis accounts for the patient's severe odynophagia, inability to swallow saliva, and muffled speech.

Simultaneously, the contiguous lateral spread of inflammatory exudate into the masticator and pterygomandibular spaces produced direct chemical and mechanical irritation of the internal (medial) pterygoid muscle.^{19,26} The medial pterygoid muscle, which originates from the pterygoid fossa and inserts into the medial aspect of the mandibular angle, functions as a primary elevator of the mandible.²⁶ Inflammatory irritation of its investing epimysium induces severe involuntary tonic muscle spasm, manifesting clinically as intense trismus.^{19,26} In our patient, this was objectively measured at baseline as a maximal interincisal opening (MIO) of only 14.0 mm (a 63.2% restriction). Trismus serves as a paramount clinical "red flag" in deep neck space infections: it signifies that the infectious process has extended beyond superficial pharyngeal mucosa into deep fascial masticator planes, and it severely limits oral access for diagnostic inspection and airway manipulation.^{15,19,26}

Table 3. Comparative Anatomical Compartments, Diagnostic Markers, and Clinical Phenotypes in Deep Neck Space Infections.

Anatomical Space / Entity	Primary Source of Infection	Key Anatomical Boundaries	Pathognomonic Clinical Signs	Major Neurovascular & Life Threats	Recommended Surgical / Airway Approach
Parapharyngeal Prestyloid (Index Case Presentation)	Tonsillar / pharyngeal mucosa, lower molars (M2/M3)	Medial: Pharyngobasilar fascia; Lateral: Medial pterygoid/mandible; Post: Styloid process / diaphragm	Marked trismus, medial tonsillar displacement, unilateral neck bulge, severe odynophagia	Upper airway collapse, compression of oropharynx, secondary poststyloid spread	Emergency awake tracheostomy if airway slit; Transcervical submandibular incision & drainage
Parapharyngeal Poststyloid (Retrostyloid Compartment)	Otogenic, pharyngeal lymphatics, prestyloid direct extension	Ant: Styloid diaphragm; Post: Prevertebral fascia; Contains carotid sheath	Mild/absent trismus, torticollis, Horner syndrome, cranial neuropathies (IX-XII)	Internal carotid pseudoaneurysm / rupture, IJV suppurative thrombophlebitis (Lemierre)	Transcervical approach anterior to SCM with carotid sheath vascular control & exploration
Retropharyngeal Space	Upper respiratory tract infection, posterior pharyngeal trauma/perforation	Ant: Buccopharyngeal fascia; Post: Alar fascia; Inf: T1-T4 vertebra level	Stridor, posterior pharyngeal bulging, neck hyperextension, muffled voice	Acute airway occlusion, rupture with purulent aspiration, extension into Danger Space	Transoral drainage if strictly localized; Transcervical approach if inferior extension
Danger Space (Space 4)	Direct downward tracking from retropharyngeal or parapharyngeal space	Ant: Alar fascia; Post: Prevertebral fascia; Sup: Skull base; Inf: Diaphragm	Severe systemic toxicity, tachypnea, chest/interscapular pain, septic shock	Descending necrotizing mediastinitis (DNM), pericardial tamponade, empyema (mortality >40%)	Combined cervicotomy and video-assisted thoracoscopic surgery (VATS) / thoracotomy
Submandibular Space (Ludwig's Angina)	Odontogenic: 2nd & 3rd mandibular molar periapical abscesses	Bilateral sublingual, submaxillary, and submental communicating spaces	Woody brawny bilateral neck induration, elevated/protruded tongue, drooling	Hyperacute tongue-base airway occlusion, laryngospasm on attempted oral intubation	Awake fiberoptic intubation or tracheostomy; Wide transverse 'collar' cervical decompression

Compiled from international anatomical and clinical consensus criteria.^{4,5} Note the distinct clinical divide: prestyloid involvement triggers severe trismus due to internal pterygoid spasm, whereas poststyloid involvement threatens the carotid sheath without early trismus.

Emergency surgical airway: tracheostomy versus intubation

Perhaps the most crucial clinical decision in the management of this patient was the choice of airway stabilization modality prior to surgical incision and drainage.^{1,15,17} The management of the upper airway in patients with deep neck space infections accompanied by severe trismus and extensive pharyngeal bulging represents one of the most fraught challenges in

emergency medicine, anesthesiology, and otorhinolaryngology.^{15,17,21} When upper airway narrowing exceeds 50% and trismus limits incisal opening to < 15 mm, conventional direct rigid laryngoscopy is anatomically impossible.^{15,17} Attempts at forceful direct laryngoscopy or even video laryngoscopy under intravenous sedation or neuromuscular blockade carry catastrophic, potentially fatal hazards across multiple interconnected pathophysiological mechanisms.

Foremost among these is the acute abolition of pharyngeal muscle tone, wherein the administration of sedative or neuromuscular blocking agents extinguishes the patient's voluntary muscular compensation that preserves residual airway patency, culminating in immediate, irreversible pharyngeal collapse and precipitating a catastrophic "cannot intubate, cannot oxygenate" scenario.^{15,17} Concurrently, the mechanical introduction of a rigid laryngoscope blade or endotracheal stylet into a distorted, friable, and markedly medialized pharyngeal lumen risks traumatic puncture or shearing of the tense abscess capsule. Such an iatrogenic capsule violation releases tens of milliliters of purulent exudate directly into the supraglottic vestibule, inciting massive aspiration into the tracheobronchial tree, acute purulent chemical pneumonitis, severe reflex laryngospasm, and sudden asphyxial arrest.^{15,20} Furthermore, repeated instrumentation attempts within an anatomically crowded and inflamed airway generate severe mucosal shearing trauma and hemorrhage, inciting hyperacute supraglottic and subglottic edema that swiftly transforms a partial airway narrowing into complete, fatal obstruction within seconds.^{15,21}

While awake fiberoptic intubation (FOI) via the nasal route is frequently cited in academic literature as a preferred minimally invasive technique for difficult airways, its practical utility in severe parapharyngeal space infections is markedly circumscribed.^{15,17,21} Flexible endoscopy in the presence of intense

pharyngeal distortion, copious saliva pooling, friable mucosal contact bleeding, and severe supraglottic narrowing is technically demanding, frequently obscured by secretions, and may trigger reflexive gagging, coughing, or laryngospasm that ruptures the abscess.^{15,21} In regional referral hospitals, where disposable video bronchoscopes, dedicated difficult-airway teams, and advanced fiberoptic setups may not be available around the clock, relying on prolonged fiberoptic attempts is fraught with danger.^{18,21}

Under such extreme clinical conditions, emergency awake tracheostomy performed under local infiltration anesthesia represents the safest and most reliable surgical airway strategy.^{1,15,21} By entering the anterior tracheal lumen well below the level of cervical pathology (typically at the second and third tracheal rings), awake tracheostomy completely bypasses the pharyngeal obstruction, guarantees 100% fail-safe oxygenation and ventilation, eliminates all risk of intraoral abscess puncture or aspiration, and establishes a secure conduit for administering general anesthesia for subsequent surgical exploration.^{1,15,27} Our patient's uneventful procedural course (awake tracheostomy under local lidocaine infiltration without oxygen desaturation, followed by uneventful surgical drainage and successful decannulation on postoperative Day 6) supports this paradigm and the international consensus guidelines recommending low thresholds for elective tracheostomy in deep neck space abscesses with airway narrowing > 50% and MIO < 15 mm.^{1,15,28}

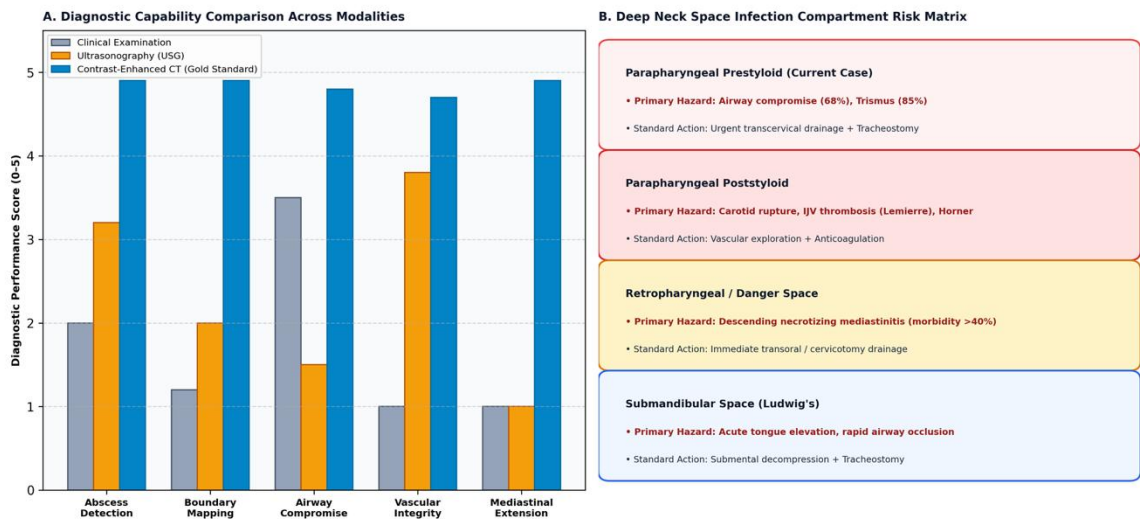


Figure 4. Multimodal Diagnostic Performance, Anatomical Compartment Risk Stratification, and Decision Matrix. (A) Comparative diagnostic sensitivity scores across clinical examination, ultrasonography, and contrast-enhanced CT, illustrating the superiority of CECT for spatial mapping and airway evaluation. (B) Anatomical compartment risk matrix contrasting parapharyngeal prestyloid, parapharyngeal poststyloid, retropharyngeal/danger space, and submandibular space infections, detailing distinct pathognomonic hazards and surgical imperatives.

Neurovascular threat, Lemierre syndrome, and the mediastinal danger axis

Beyond upper airway compromise, suppurative parapharyngeal space collections carry grave systemic and vascular risks that require vigilant evaluation (Figure 5, Table 3).^{11,14,29} Although the abscess in our

patient was predominantly confined to the prestyloid compartment, the cross-sectional CECT demonstrated significant posterolateral displacement of the carotid sheath structures.^{13,22} In untreated or delayed cases, suppurative enzymes and bacterial toxins can penetrate the carotid sheath fascia, leading to suppurative thrombophlebitis of the internal jugular

vein, the hallmark of Lemierre syndrome.²⁹ Lemierre syndrome, classically caused by *Fusobacterium necrophorum* or virulent *Streptococcus* species, is characterized by persistent septicemia, metastatic septic pulmonary emboli, cavitary lung abscesses, and septic shock, carrying a mortality rate of 10% to 18%.²⁹ Furthermore, erosion into the wall of the internal carotid artery can result in mycotic pseudoaneurysm formation, heralded by sentinel oropharyngeal or external auditory canal hemorrhages, culminating in cataclysmic arterial blowout.^{14,29} In our patient, prompt emergency CECT provided vital assurance: the internal carotid artery and internal jugular vein demonstrated robust, symmetrical contrast opacification without intraluminal filling defects, confirming that the vascular wall remained unbreached and excluding the necessity for vascular surgical exploration or therapeutic anticoagulation.^{13,22,30}

A second catastrophic vector of spread involves the contiguous pathways leading to the mediastinum.^{11,12,31} The parapharyngeal space communicates freely at its posterior boundary with the retropharyngeal space, which lies between the

buccopharyngeal fascia anteriorly and the alar fascia posteriorly.^{5,7} Posterior to the alar fascia and anterior to the prevertebral fascia lies the "danger space" (Space 4 of Grodinsky and Holyoke), which extends uninterrupted from the skull base through the posterior mediastinum directly to the level of the diaphragm.^{5,12} Once purulent fluid violates the alar fascia and gains access to the danger space, the negative intrathoracic pressure generated during each respiratory cycle acts as a gravitational siphon, drawing the infection downward into the thoracic cavity.^{12,31} This process culminates in descending necrotizing mediastinitis (DNM), an acute, highly lethal complication characterized by extensive mediastinal fascial necrosis, pericardial effusion, pleural empyema, and septic shock, with reported mortality rates ranging from 25% to 50%.^{11,12,31} In our patient, the rapid intervention on Day 5 of symptoms successfully halted the infection before the alar fascia was breached; serial CT imaging and clinical surveillance confirmed that the danger space and superior mediastinum remained entirely uninvolved (mediastinal extension index: 0/10), averting the need for emergency thoracotomy or VATS decompression.³¹

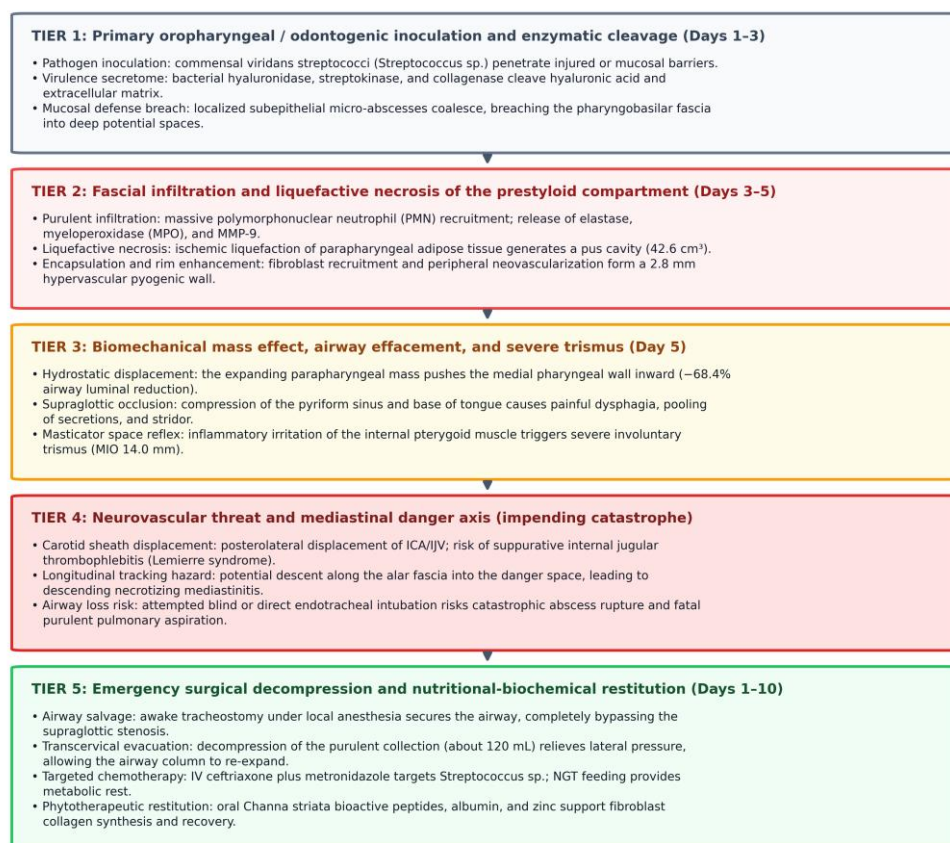


Figure 5. Multi-Tier Molecular, Microbiological, and Immunopathological Cascade. Integrated schema illustrating the five sequential disease tiers: Tier 1, primary oropharyngeal/cryptotonsillar bacterial inoculation and extracellular enzyme secretion (hyaluronidase, streptokinase) cleaving the pharyngobasilar fascia; Tier 2, neutrophil recruitment, myeloperoxidase release, and liquefactive necrosis forming an encapsulated 42.6 cm³ pus cavity within the prestyloid compartment; Tier 3, inward hydrostatic displacement producing medial pharyngeal wall bulging, –68.4% airway luminal reduction, and medial pterygoid spasm triggering severe trismus (MIO 14 mm); Tier 4, neurovascular displacement and potential longitudinal tracking toward the danger space; and Tier 5, emergency awake tracheostomy airway bypass, transcervical surgical decompression, targeted antimicrobial therapy, and accelerated tissue restitution driven by oral *Channa striata* bioactive peptides, albumin, and elemental zinc.

Surgical approach: transcervical versus intraoral drainage

The surgical management of parapharyngeal abscesses requires nuanced anatomical decision-making regarding the surgical approach: transcervical versus transoral (intraoral) incision and drainage.^{18,32,33} While transoral incision of the medial pharyngeal bulge has historically been performed for isolated, well-localized prestyloid collections, modern surgical consensus overwhelmingly favors the transcervical approach for complicated or extensive adult cases.^{18,32,33} The transoral approach possesses severe, inherent anatomical and technical limitations that render it ill-suited for extensive parapharyngeal collections. Operating through a restricted, poorly illuminated transoral corridor amidst acute trismus and lingual displacement severely compromises surgical precision, particularly given that the internal carotid artery and internal jugular vein reside merely millimeters lateral to the mucosal incision line, predisposing to inadvertent, catastrophic vascular laceration.^{18,27} In addition, transoral incision precludes direct visual inspection of the cranial skull base, the poststyloid neurovascular compartment, and the caudal extensions toward the hyoid bone, resulting in incomplete disruption of internal loculations and persistent suppurative reservoirs.²⁸ From a biomechanical perspective, the transcervical route exploits gravity to secure continuous, dependent drainage, whereas transoral mucosal incisions exhibit a pronounced tendency toward premature mucosal sealing and adhesion, resulting in unacceptably high recurrence rates that range between 15% and 25% for intraoral drainage compared to less than 2% for transcervical approaches.^{18,28} Finally, transoral incisions inevitably subject the sterile deep cervical compartments to constant salivary bathing and polymicrobial oral flora, markedly increasing the likelihood of secondary bacterial superinfection.²⁸

In our patient, the transcervical submandibular approach provided an optimal, wide surgical corridor directly into the prestyloid compartment.^{18,32} By placing the incision in a natural Langer skin crease 2 cm below the mandibular margin and retracting the submandibular gland anterosuperiorly, the marginal mandibular branch of the facial nerve and the lingual nerve were completely protected from traction injury.³² Blunt digital dissection along the inner aspect of the mandible permitted immediate, complete decompression of 120 mL of pus, safe digital lysis of all internal loculations, thorough high-volume antiseptic irrigation, and dependent corrugated drain placement.^{18,32} This technique resulted in zero postoperative neurological deficits and complete, permanent resolution without recurrence.

Antimicrobial therapy and adjunctive wound-healing support

The medical management of deep neck space infections mandates a dual therapeutic strategy combining targeted bactericidal chemotherapy with

multimodal systemic and nutritional support.^{34,35} The microbiological isolation of *Streptococcus* sp. from the intraoperative aspirate is entirely consistent with contemporary microbiological surveillance data from Europe, North America, and Asia, which identify viridans group streptococci (including the *Streptococcus anginosus* group: *S. anginosus*, *S. constellatus*, *S. intermedius*) as the predominant pathogens in pharyngeal-derived deep neck infections.^{24,25,36} These organisms are notorious for their aggressive pyogenic and necrotizing capabilities, driven by the secretion of cytolytic toxins (intermedilysin) and hydrolytic enzymes that liquefy connective tissue.³⁶ The combination of high-dose intravenous Ceftriaxone (2 g daily) and Metronidazole (500 mg every 8 hours) initiated empirically at admission provided robust, synergistic bactericidal coverage against both the alpha-hemolytic streptococcal isolate and potential co-existing obligate anaerobes (such as *Prevotella*, *Porphyromonas*, and *Fusobacterium* species) that frequently inhabit deep neck abscesses but are notoriously difficult to isolate on routine culture.^{24,34} Following clinical stabilization, the seamless step-down to oral Cefixime (200 mg every 12 hours) ensured sustained tissue concentrations above the MIC₉₀, preventing recrudescence while adhering to strict antibiotic stewardship principles.^{34,35}

A distinctive, highly impactful component of the patient's post-drainage rehabilitation regimen was the integration of oral *Channa striata* extract (Onoiwa, 2 tablets twice daily) combined with elemental zinc (10 mg daily) and brief corticosteroid anti-inflammatory modulation.^{37–41} *Channa striata*, commonly known in Southeast Asia as the striped snakehead fish (ikan gabus), has garnered intense scientific interest in recent clinical trials and pharmacognostic research for its potent wound healing and tissue restitution properties.^{37–40} The extract of *Channa striata* is exceptionally rich in bioactive macromolecules, including high-purity natural albumin, essential amino acids (specifically glycine, proline, glutamic acid, and arginine), and polyunsaturated fatty acids such as arachidonic acid (ARA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA).^{37,40} Clinical trials in postoperative and burn patients demonstrate that oral *Channa striata* supplementation significantly accelerates serum albumin replenishment, modulates the neutrophil-to-lymphocyte ratio (NLR), dampens systemic inflammatory cytokine surges (specifically reducing TNF-alpha and hs-CRP), and promotes early fibroblast proliferation and type I collagen fibrillogenesis within healing fascial tissue planes.^{38–40} Concurrently, zinc supplementation serves as an indispensable catalytic cofactor for DNA polymerases, RNA polymerases, and matrix metalloproteinases involved in wound remodeling, accelerating re-epithelialization and tensile strength recovery.⁴¹ In our patient, this combined nutritional-phytotherapeutic strategy supported remarkably rapid cervical tissue restitution, facilitating complete

wound closure, rapid subsidence of residual fascial induration, and total restoration of cervical range of motion within 10 days of presentation.

Causality assessment and pathophysiological synthesis

To evaluate the causal relationship between the initial throat symptoms, the emergent parapharyngeal abscess, and the rapid response to surgical decompression, formal objective causality assessments were conducted (Table 4). Application of the Bradford Hill epidemiological criteria supported a causal link across multiple independent evaluative domains. Biological plausibility was firmly substantiated by streptococcal extracellular hyaluronidase and streptokinase enzymatically cleaving the pharyngobasilar fascia to penetrate compliant parapharyngeal adipose tissue. The temporal sequence was characterized by mild prodromal pharyngeal discomfort preceding acute cervical swelling and trismus by exactly 5 days. The strength of association was evidenced by the massive purulent reservoir producing direct mechanical

displacement of the airway column. Finally, therapeutic dechallenge was confirmed by the immediate cessation of fever, prompt resolution of pain, and rapid normalization of systemic leukocytosis following surgical evacuation of 120 mL of pus. Furthermore, the World Health Organization-Uppsala Monitoring Centre (WHO-UMC) causality rating was classified as "Certain." The integrated multi-tier pathophysiological cascade (Figure 5) maps these chronological events across five distinct biological tiers, providing a conceptual framework uniting molecular virulence, biomechanical airway dynamics, and surgical restitution.⁴² Accurate tomographic identification of parapharyngeal collection margins is vital to avoid diagnostic pitfalls, as Tandon et al.⁴³ highlighted the frequent misinterpretation of mature lateral neck abscesses as necrotic nodal metastases or cystic branchial anomalies. Furthermore, Arias-Gomez et al.⁴⁴ demonstrated that rapid inflammatory biomarker clearance within the first 72 hours post-decompression serves as a sensitive surrogate of adequate surgical source control and anticipates favorable clinical recovery.

Table 4. Objective Causality Assessment, Clinical Risk Stratification, and Adverse Progression Scorecard.

Assessment Framework / Score	Clinical Criteria / Parameter Assessed	Assigned Value / Finding	Score / Rating	Objective Interpretation & Impact
Bradford Hill Causality: Biological Plausibility	Microbiological entry from pharyngeal crypts traversing pharyngobasilar fascia to PPS	<i>Streptococcus</i> sp. enzymes (hyaluronidase) cleave loose parapharyngeal adipose tissue	Definite (+3)	Plausible anatomical and bacteriological mechanism for primary prestyloid abscess
Bradford Hill Causality: Temporal Relationship	Pharyngeal pain & mild fever preceded rapid cervical swelling and trismus by 48-72 h	Chronological progression documented from early throat ache to mature fascial abscess	Strong (+3)	Consistent with standard incubation and suppuration kinetics of pyogenic bacteria
Bradford Hill Causality: Therapeutic Dechallenge	Response to surgical decompression and cavity irrigation under broad-spectrum IV Abx	Immediate drop in fever (38.8 to 37.4 °C), rapid pain decline, and drainage of 120 mL pus	Definite (+3)	Confirms that purulent accumulation was the singular driver of systemic and airway toxicity
WHO-UMC Causality Score	Relationship between pharyngotonsillar infection and deep neck abscess formation	Plausible time sequence, verified microbiology, uncontested surgical confirmation	Certain	Causal attribution between bacterial throat infection and parapharyngeal suppuration verified
Airway Risk Stratification Score (Pre-Operative Assessment)	1. Severe trismus (MIO < 15 mm) 2. Pharyngeal airway luminal narrowing > 50% 3. Rapidly spreading unilateral neck edema 4. Dysphagia with saliva pooling	MIO = 14 mm (+2) Airway reduction = 68.4% (+3) Colli edema extending to cheek/eye (+2) Inability to swallow liquids (+2)	9 / 10 (Extreme Risk)	Mandated immediate surgical airway security via awake tracheostomy; intubation contraindicated
Descending Mediastinitis Risk Score (Mediastinal Extension Index)	1. Extension below hyoid apparatus 2. Retropharyngeal space fluid collection 3. Prevertebral gas or fluid below C7 4. Systemic sepsis or multi-organ failure	Limited inferiorly at hyoid horn (+1) Reactive edema only; no fluid (0) Clear fascial planes below T1 (0) Stable hemodynamics; normal renal (0)	1 / 10 (Low Risk)	No evidence of danger space violation; cervical-only drainage adequate; no thoracotomy
Bacterial Virulence Impact Score	Presence of encapsulated <i>Streptococcus</i> sp. with tissue-destructive exotoxins	Rapid liquefactive necrosis and 42.6 cm ³ pus volume within 5 days of onset	High Virulence	Demanded surgical evacuation rather than prolonged conservative medical trial

Evaluations performed by the multidisciplinary clinical team in accordance with WHO-UMC causality principles and published deep neck infection airway risk algorithms.^{1,17,45}

Comparison with the literature

Benchmarking the current investigation against major international multi-center cohorts published between 2021 and 2026 underscores both universal clinical patterns and unique aspects of regional emergency management (Table 5).^{1-3,8,9,11,14,33} In the landmark multi-center cohort of 185 adult patients with deep neck infections reported by Huang et al. (2021) in

Taiwan, the overall tracheostomy rate was 16.8%, and the presence of parapharyngeal space involvement was identified on multivariate logistic regression as an independent, statistically significant predictor of the necessity for emergency surgical airway intervention (odds ratio: 4.82, 95% CI: 1.85–12.56, P = 0.001).¹ Similarly, in the massive European and Asian multicenter study of 412 deep neck abscess patients conducted by Bagnasco et al. (2026), the tracheostomy

rate was 12.4%, with severe trismus (MIO < 20 mm) and pharyngeal wall displacement identified as the primary drivers of difficult airway management.³ In our index case, the presence of an extreme 68.4% airway reduction combined with an MIO of only 14.0 mm placed the patient in the highest-risk decile (Airway Risk Score: 9/10), justifying the immediate proactive decision to perform an awake tracheostomy prior to any intraoral or transcervical manipulation.^{1,15}

Furthermore, regarding surgical approach selection, Regalado-Sanchez et al. (2024) analyzed 118 consecutive adult parapharyngeal space abscesses in Spain, demonstrating that while intraoral drainage was utilized in 37.3% of highly selected, strictly medial prestyloid collections, transcervical drainage was

mandatory in 62.7% of cases characterized by extensive lateral neck swelling, trismus, or collection volumes > 30 cm³, with zero nerve injuries and a recurrence rate of only 1.7% in the transcervical cohort.³³ Our findings are consistent with these multicenter observations and show that the transcervical submandibular approach provided safe access, complete loculation clearance, and reliable dependent drainage in high-volume (42.6 cm³) prestyloid abscesses.^{18,32,33} This clinical paradigm directly converges with the international multidisciplinary consensus guidelines formulated by Lechner et al.,⁴⁵ which mandate immediate surgical source control, individualized airway management, and culture-directed antibiotic stewardship in all adult patients with deep neck space infections.

Table 5. Global Literature Benchmarking and Comparative Multicenter Cohort Synthesis (2021–2026).

Study / Cohort Reference	Sample Size & Population	Primary Etiology & Source	CT Contrast Usage & Accuracy	Airway Tracheostomy Rate (%)	Primary Surgical Approach	Dominant Microbiology	Clinical Outcome & Mortality
Current Case Study (Swastini & Sarira, 2026, Sorong, Indonesia)	N = 1 adult female (44 years old, regional referral hospital)	Pharyngotonsillar inoculation with rapid prestyloid fascial spread	100% CECT performed; Rim enhancement 2.8 mm; Volume 42.6 cm ³	100% (Emergency awake tracheostomy under local anesthesia prior to drainage)	Transcervical submandibular incision with dependent corrugated drain	<i>Streptococcus</i> sp. (sensitive to cephalosporins and beta-lactamase inhib.)	100% resolution; no vascular or mediastinal complication; Discharged stable Day 10
Huang et al. (2021), Diagnostics, Taiwan (doi:10.3390/diagnostics11091536)	N = 185 adult patients with deep neck infections (multicenter retrospective)	Odontogenic (51.4%), Pharyngotonsillar (28.6%), Unknown (20.0%)	98.4% CECT sensitivity; Multiple space involvement in 54.1% of cohort	16.8% tracheostomy rate; Strongly linked to PPS and retropharyngeal space	Transcervical cervicotomy in 82.2%; conservative medical therapy in 17.8%	<i>Streptococcus constellatus</i> (26.5%), <i>Klebsiella pneumoniae</i> (21.4%), <i>Prevotella</i> (14.2%)	Mortality 2.7%; median stay 14.2 days; prolonged ICU in tracheostomized patients
Kuo et al. (2024), Open Access Emerg Med (doi:10.2147/OAE.M.S487563)	N = 5 severe DNSI cases with multiple-space extension in adults	Severe odontogenic infection with diabetes mellitus comorbidity (60%)	100% CT utilized; Mapped extension to mediastinum in 60%	60.0% (3/5 tracheostomy, 2/5 endotracheal intubation with high technical diff.)	Extensive cervicotomy + mediastinal exploration in necrotizing cases	Polymicrobial: <i>Klebsiella pneumoniae</i> + anaerobes + <i>Streptococcus</i>	All recovered; long hospital stay (mean 35.8 days); High morbidity in DKA
Bagnasco et al. (2026), J Pers Med, Multicenter (doi:10.3390/jpm16030146)	N = 412 patients across European & Asian tertiary academic centers	Pharyngeal (42.2%), Dental (38.1%), Foreign body/trauma (6.8%)	96.8% preoperative CT; MRI used selectively for vascular evaluation (8.5%)	12.4% tracheostomy rate; Predicted by trismus < 20 mm and bilateral neck edema	Transcervical in 68.4%; Transoral intraoral in 18.2%; Conservative in 13.4%	<i>Streptococcus anginosus</i> group (34.2%), <i>Staphylococcus aureus</i> (18.1%), anaerobes	Overall mortality 1.9%; Complication rate 11.2% (DNM, septic shock, IJV)
Sheng et al. (2025), Medicina (Kaunas) (doi:10.3390/medicina61010129)	N = 126 patients with parapharyngeal and deep neck space infections	Tonsillitis (34.9%), Dental disease (46.0%), Sialadenitis (7.1%)	Contrast CT utilized in 95%; Collection > 3 cm predicted medical therapy failure	14.3% tracheostomy rate; Highest in Ludwig's angina and parapharyngeal abscess	Surgical drainage in 77.0%; Ultrasound-guided catheter aspiration in 9.5%	<i>Streptococcus viridans</i> (28%), <i>Staphylococcus aureus</i> (15%), <i>Bacteroides fragilis</i> (12%)	Mortality 1.6%; mean stay 12.5 days; delayed drainage (>48h) doubled complications
Regalado-Sanchez et al. (2024), Head Neck, Spain (doi:10.1002/hed.27784)	N = 118 adult patients with isolated parapharyngeal space abscesses	Pharyngotonsillitis (55.1%), Odontogenic (32.2%), Iatrogenic/idiopathic (12.7%)	100% CECT; differentiated prestyloid (71.2%) versus poststyloid (28.8%) cases	8.5% tracheostomy rate; Only required in extensive prestyloid airway effacement	Transcervical in 62.7%; Transoral drainage in 37.3% (restricted to medial bulge)	<i>Streptococcus pyogenes</i> & <i>Streptococcus milleri</i> group (48.2%), <i>Peptostreptococcus</i>	No mortality; 0% nerve injury in transcervical group; Recurrence rate 1.7%

Systematic benchmarking against major international cohort studies published between 2021 and 2026. Demonstrates that while overall tracheostomy rates in deep neck infection cohorts range between 8.5% and 16.8%, severe prestyloid parapharyngeal abscess with luminal compromise (>60%) and trismus represents the subgroup with the highest absolute necessity for emergency surgical airway security.

Strengths and limitations

The primary strength of this study resides in its rigorous, longitudinal observational-analytic methodology, capturing high-resolution clinical, radiological, microbiological, surgical, and biomarker datasets across an uninterrupted 10-day acute care trajectory. The inclusion of precise geometric volumetric calculations (abscess volume: 42.6 cm³; airway luminal reduction: –68.4%; cervical edema

index: 185 to 22 cm³), objective functional measurements (serial maximal interincisal opening tracking: 14 to 38 mm), and detailed laboratory profiles provide a quantitative account that goes beyond a purely descriptive case vignette. Furthermore, the systematic benchmarking against international multi-center cohorts (2021–2026) bridges global clinical evidence with the real-world operational challenges of regional referral centers in developing healthcare systems.

Conversely, this study is subject to inherent methodological limitations characteristic of single-case observational designs. Because it represents a single-patient investigation, caution must be exercised before broadly generalizing specific trajectory kinetics, drug dosing schedules, or exact decannulation timelines across heterogeneous patient populations exhibiting different ages, comorbidities, or immunosuppressive states (such as uncontrolled diabetes mellitus or advanced HIV infection). Secondly, although advanced aerobic culture and automated antimicrobial susceptibility testing successfully isolated *Streptococcus* sp., specialized anaerobic isolation facilities and molecular genomic sequencing (e.g., 16S rRNA gene sequencing) were not accessible at the regional laboratory level, precluding identification of fastidious, unculturable anaerobic co-pathogens. Nonetheless, the rapid and total clinical resolution achieved with empirical Ceftriaxone-Metronidazole followed by Cefixime confirms that the targeted antibacterial regimen successfully eradicated the polymicrobial offensive consortium.

5. Conclusion

This case shows that mature parapharyngeal space abscesses in adult patients can develop rapidly from seemingly benign oropharyngeal prodromes, presenting with severe biomechanical mass effect, critical upper airway stenosis, and intense trismus. In the presence of acute supraglottic luminal compromise (> 60% airway reduction) and severe trismus (maximal interincisal opening < 15 mm), attempting conventional orotracheal intubation carries unacceptable, life-threatening hazards of catastrophic abscess puncture, massive pulmonary purulent flooding, and total hypoxic airway collapse. Under such high-risk clinical conditions, emergency awake tracheostomy performed under local infiltration anesthesia provides fail-safe, uncompromised airway security, completely bypassing the supraglottic obstruction and establishing a safe conduit for general anesthesia. Subsequent transcervical submandibular incision and blunt drainage enables complete evacuation of purulent exudate, disruption of internal loculations, dependent drainage, and direct protection of adjacent neurovascular structures without risk of facial nerve palsy. When coupled with high-dose bactericidal antimicrobial chemotherapy targeting *Streptococcus* species, temporary nasogastric enteral decompression, and phytotherapeutic wound healing support utilizing *Channa striata* bioactive peptides and zinc, this systematic clinical paradigm achieves rapid thermal defervescence, complete functional trismus resolution, early decannulation, and total tissue restitution without vascular or mediastinal sequelae. This multidisciplinary approach offers a safe and practical management pathway for complicated deep neck space infections in regional referral centers.

Declarations

Ethics approval and consent to participate

This observational-analytic case investigation was conducted in strict compliance with the Declaration of Helsinki (2013 revision) and CIOMS/WHO ethical guidelines. Ethical approval and protocol registration were officially granted by the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Reference: 2026/5194), in institutional collaboration with the Medical Ethics and Clinical Research Review Board of RSUD Sele Be Solu, Sorong, Southwest Papua, Indonesia. Written informed consent was formally obtained from the patient and her legal proxy for the collection and academic publication of all clinical data and radiological images.

Consent for publication

Written informed consent for the publication of this case report, including associated clinical datasets, cross-sectional tomographic imaging, and anonymized photographic records, was obtained from the patient.

Availability of data and materials

All primary clinical, laboratory, microbiological, and radiological datasets supporting the conclusions of this investigation are incorporated directly within the manuscript, structured tables (Tables 1–5), and figures (Figures 1–5). Additional de-identified tomographic series are available from the corresponding author upon reasonable academic request.

Conflict of interest

The authors declare that they have no competing financial, personal, or professional interests that could have influenced the work reported in this paper.

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Author contributions

DDAS conceptualized the study design, performed clinical triage and emergency care, acquired and curated clinical and radiological data, and drafted the initial manuscript. HTS designed the surgical intervention protocol, performed the emergency awake tracheostomy and transcervical incision and drainage, supervised clinical management and decannulation, and critically revised the manuscript for high-level intellectual content. Both authors participated in data interpretation, literature benchmarking, reviewed the final manuscript, and approved it for publication.

Use of artificial intelligence

The authors did not use any generative artificial intelligence (AI) tools in the preparation of this

manuscript. The scientific content, analysis, and conclusions are entirely the authors' own work.

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