



Comprehensive Clinical Insights into the Diagnosis, Management, and Interprofessional Approaches to Deep Neck Infections for Healthcare Professionals



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Abstract

Background: Deep neck infections (DNIs) are serious suppurative processes within the fascial planes of the neck, often originating from odontogenic or oropharyngeal sources. They pose a significant risk due to their potential for rapid progression, airway compromise, and life-threatening complications like mediastinitis and sepsis.

Aim: This article provides a comprehensive clinical overview of DNIs for healthcare professionals, covering etiology, epidemiology, pathophysiology, diagnostic evaluation, and evidence-based management strategies to improve patient outcomes.

Methods: The review synthesizes current medical knowledge, highlighting that DNIs are typically polymicrobial, involving oral flora such as streptococci, staphylococci, and anaerobes. Diagnosis relies on a high clinical suspicion based on history and physical exam, confirmed primarily with contrast-enhanced computed tomography (CECT) to localize abscesses and define anatomical extent. Laboratory markers like elevated neutrophil-to-lymphocyte ratio (NLR) and C-reactive protein (CRP) aid in severity assessment.

Results: Management is multidisciplinary. The cornerstone treatment involves prompt administration of broad-spectrum intravenous antibiotics and timely surgical or image-guided drainage of established abscesses. Securing the airway is the paramount initial priority in cases of potential compromise.

Conclusion: DNIs represent a medical emergency requiring swift diagnosis and aggressive management. A coordinated interprofessional approach is essential for successful outcomes, emphasizing early imaging, appropriate antimicrobial therapy, source control, and vigilant airway management to prevent morbidity and mortality.

Keywords: Deep Neck Infection, Retropharyngeal Abscess, Parapharyngeal Abscess, Airway Compromise, Contrast-Enhanced CT, Surgical Drainage..

1. Introduction

Deep neck infections involve suppurative processes within the deep fascial planes of the neck. They arise when bacteria extend into the deep cervical spaces from nearby sites such as the tonsils, parotid gland, cervical lymph nodes, or teeth. These infections progress rapidly in some cases and can produce severe local and systemic consequences. Local mass effect frequently impairs airway patency or interferes with swallowing and phonation. Patients are commonly present with neck swelling, pain, fever, difficulty swallowing, a change in voice, or limited mouth opening. Trismus often indicates involvement of the masticator or parapharyngeal space. [1][2] Anatomical knowledge underpins clinical reasoning about deep neck infections. The neck contains multiple potential spaces delineated by fascial layers. Infection confined to a single compartment behaves differently from infection that tracks across fascial planes into contiguous spaces. For example, extension into the retropharyngeal or prevertebral space may carry risk of mediastinal spread. The parapharyngeal space lies lateral to the pharynx and communicates with other deep compartments. These anatomical relationships explain why a seemingly localized odontogenic or pharyngeal infection can evolve into a diffuse cervicothoracic process. [3][4]

Host factors alter disease course. Immunosuppression, diabetes, chronic illness, recent trauma, or recent instrumentation increases the likelihood of rapid progression and unusual pathogens. Intravenous drug use also predisposes to aggressive infections. In contrast, healthy hosts often localize infection more readily. The microbial milieu is typically polymicrobial in odontogenic sources and more likely to include aerobic streptococci in oropharyngeal sources. Empiric treatment choices must reflect these patterns and local resistance data. [5][6] Clinical presentation varies with the space involved. Masticator space disease produces jaw pain and trismus. Parapharyngeal infections manifest with lateral neck swelling and bulging of the oropharyngeal wall. Retropharyngeal collections may present with neck stiffness, difficulty handling secretions, and respiratory distress. Submandibular and submental infections classically cause notable submandibular

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swelling and elevation of the tongue, which threaten the airway. Signs may be subtle early on because the abscess lies deep beneath intact skin. Fever and leukocytosis support infection but absence of fever does not exclude a deep collection. [1][2]

Imaging is essential when deep infection is suspected. Contrast enhanced computed tomography supplies detailed information on the location, size and extent of fluid collections. CT also delineates gas within tissues and evaluates for mediastinal extension. Ultrasonography offers value for superficial or accessible collections and can guide aspiration in select cases. Magnetic resonance imaging provides superior soft tissue contrast and is useful when CT findings are equivocal or when neural or vascular involvement is suspected. Timely imaging reduces diagnostic uncertainty and informs decisions about airway management and the need for surgical drainage. [3][4] Airway assessment and protection are primary priorities. Patients with submandibular or odontogenic infections or with signs of airway compromise require early involvement of anesthesia and otolaryngology or surgery. Securing the airway may dictate the timing and setting of operative drainage. Empiric broad spectrum antibiotics should begin promptly after obtaining cultures when feasible. Antibiotic regimens should target likely aerobic and anaerobic organisms and be adjusted according to culture results and clinical response. [5][6]

Surgical drainage remains the definitive therapy for established abscesses. Indications include radiographic evidence of a drainable collection failure to improve with antibiotics or evolving airway compromise. Drainage techniques depend on the space involved and may require intraoral approaches, external neck incisions or combined procedures. Repeated drainage may be necessary for multiloculated disease. Adjunctive measures include supportive care analgesia fluid resuscitation and targeted treatment of comorbid conditions. [1][2] Outcomes depend on prompt recognition and coordinated care. Delayed diagnosis increases the risk of mediastinitis sepsis thrombophlebitis of cervical veins and cranial nerve deficits. An interprofessional approach that integrates emergency medicine otolaryngology radiology anesthesia and infectious disease specialists optimizes airway safety and reduces morbidity. Early imaging and timely surgical consultation improve the likelihood of recovery without major complications. [1][2][3][4][5][6]

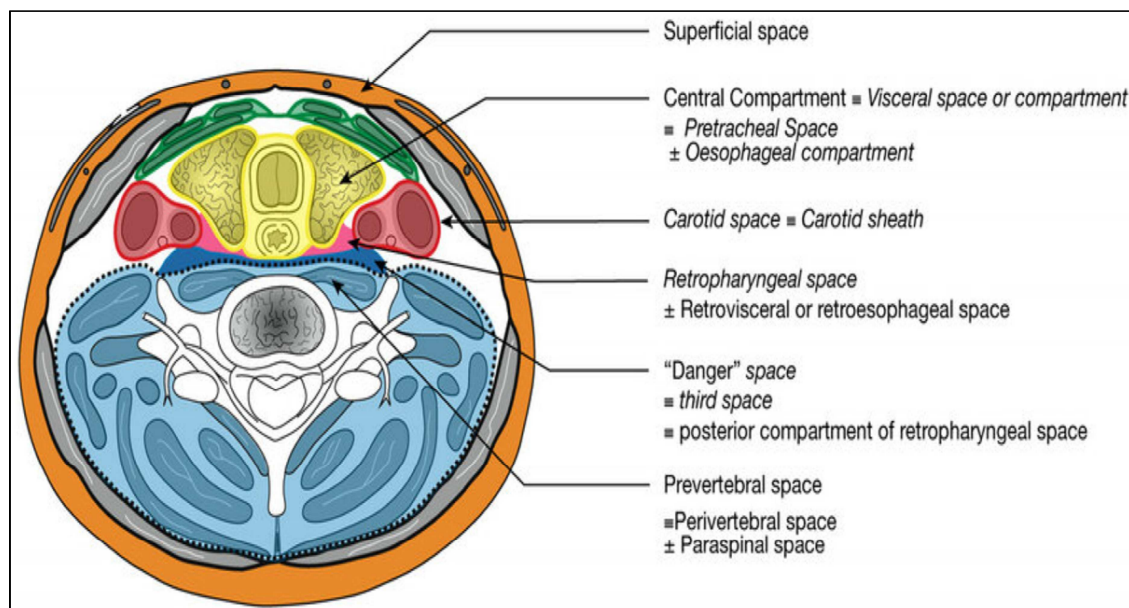


Figure 1: Anatomy of Deep Neck structures.

Etiology

Deep neck space infections typically reflect a polymicrobial process. Bacteria that normally colonize the oral cavity and upper respiratory tract breach local barriers and proliferate within the potential spaces of the neck. In adults the most frequent point of entry is the dental apparatus and periodontium. Odontogenic infections extend along fascial planes and through lymphatic channels to seed the deep cervical compartments. In adults the next most common source is infection of the tonsils and peritonsillar tissues. In children the pattern differs; pharyngeal and tonsillar infections predominate as initiating events. The anatomic origin therefore informs both the likely microbial mix and the route of spread [7][8].

The microbiology of these infections mirrors the resident flora of the mouth and throat. Aerobic streptococci, including *Streptococcus pyogenes* and members of the viridans group, are frequent pathogens. *Staphylococcus aureus* is also commonly isolated, sometimes in combination with gram-negative bacilli such as *Klebsiella* species. Obligate and facultative anaerobes contribute substantially to the infectious burden; *Fusobacterium* species and other anaerobes are major players in polymicrobial collections. The resulting flora often represents overgrowth of expected commensals rather than invasion by unusual organisms. Nevertheless, less common pathogens may be implicated in select settings. *Actinomyces* species can produce indolent, chronic infections with sinus tract formation. Mycobacterial infection and fungal pathogens may cause deep neck disease in immunocompromised hosts or in areas with specific exposure risks. Host factors shape both susceptibility and the microbiologic profile. Patients with diabetes mellitus, impaired cellular immunity, or other immunocompromised states face increased risk of rapid progression and of infection with atypical organisms. Intravenous drug use introduces distinct

pathogens and can produce aggressive, multiloculated collections. Recent oropharyngeal procedures, dental work, penetrating trauma, or foreign bodies alter local defenses and create portals of entry. The site of origin also matters odontogenic sources favor mixed aerobic and anaerobic flora, whereas pure oropharyngeal infections may show a predominance of streptococci and fewer gram-negative organisms. Geographic and institutional patterns of antimicrobial resistance further influence which organisms dominate clinical cultures [7][8].

Microbial synergy underlies the pathogenesis of many deep neck collections. Aerobes and anaerobes interact metabolically and immunologically to promote abscess formation. Anaerobic metabolism lowers local oxygen tension, favors survival of obligate anaerobes, and generates metabolites that impair host phagocytic function. Biofilm formation on devitalized tissue or foreign material can sustain a nidus of infection that resists host defenses and antimicrobials. In chronic or recurrent presentations organisms such as *Actinomyces* can form dense colonies encased in sulfur granules that require prolonged therapy and often surgical debridement. The clinical context directs empiric therapy because cultures may not capture all causative agents. Initial antibiotic choices must cover gram-positive cocci, gram-negative rods, and anaerobes pending culture data. Cultures of aspirated pus and blood cultures, when positive, refine therapy and permit narrowing of agents based on susceptibility. In immunocompromised patients or after prior antibiotic exposure clinicians should anticipate resistant organisms and atypical pathogens and tailor empirical regimens accordingly [7][8].

Epidemiologic shifts and local healthcare factors also influence etiology. In regions with high prevalence of dental disease or limited access to dental care, odontogenic sources rise in frequency. Likewise, increasing prevalence of diabetes and other chronic illnesses changes the host landscape and may shift pathogen patterns toward more virulent or resistant strains. Intravenous drug use and rising rates of hospitalization for procedures increase exposure to nosocomial organisms that can seed deep neck spaces. In summary, deep neck infections arise mainly from the normal oral and pharyngeal flora, with odontogenic and tonsillar sources most common in adults and children respectively. The infections are polymicrobial, typically involving streptococci, staphylococci, gram-negative rods, and anaerobes such as *Fusobacterium*. Uncommon organisms including *Actinomyces*, mycobacteria, and fungi appear in selected patients, often those with compromised immunity. Host comorbidities, recent procedures, and the anatomic origin of the infection shape the microbial mix and the clinical course and thus dictate empiric therapeutic choices and the need for drainage and targeted antimicrobial therapy. [7][8]

Epidemiology

Retropharyngeal and parapharyngeal abscesses represent two of the most significant forms of deep neck infections due to their anatomical location, potential for rapid spread, and association with severe complications. Retropharyngeal abscesses originate in the retropharyngeal space, which is situated between the pharyngeal constrictor muscles anteriorly and the prevertebral fascia posteriorly. This space contains lymphatic tissue that drains the nasopharynx, adenoids, and posterior paranasal sinuses, making it vulnerable to infectious processes in early childhood. In contrast, parapharyngeal abscesses usually occur as a result of direct spread of infection through the lateral pharyngeal wall into the parapharyngeal space. Both forms share similar clinical risks, yet their epidemiologic distribution and underlying causes highlight key differences. Although considered relatively rare in the general population, deep neck infections demonstrate a non-negligible impact on public health. The annual incidence is estimated at 0.22 cases per 10,000 individuals, underscoring their rarity but also their clinical importance given the potential for life-threatening complications. Recent research indicates that the incidence of these infections has been increasing over the past decade, which raises concerns for clinicians and healthcare systems alike. Several factors may contribute to this upward trend, including changes in antibiotic prescribing patterns, increased antibiotic resistance, and improved diagnostic imaging that allows earlier and more accurate detection of cases that might previously have been missed [9].

Epidemiologically, the burden of retropharyngeal and parapharyngeal abscesses falls most heavily on children, particularly those under the age of 5 years. This age group is disproportionately affected because of the presence of retropharyngeal lymph nodes, which serve as conduits for infections of the upper respiratory tract. These lymph nodes typically regress by the age of 5, thereby reducing the risk of retropharyngeal infections in older children and adults. Consequently, in young children, respiratory infections such as tonsillitis, pharyngitis, or sinusitis may extend into these lymphatic tissues, leading to abscess formation. Parapharyngeal abscesses in children may also arise secondary to tonsillar or pharyngeal infections, reinforcing the close anatomical and functional relationship between upper respiratory tract infections and deep neck abscess development [10]. The clinical symptoms commonly associated with these infections—dysphagia, odynophagia, fever, neck stiffness, and respiratory distress—reflect the involvement of vital structures within the deep neck compartments. The potential for complications arises from the close proximity of these spaces to critical vascular and mediastinal structures. Both retropharyngeal and parapharyngeal spaces communicate with the posterior mediastinum through fascial planes, creating a direct pathway for infection to extend downward, resulting in necrotizing mediastinitis. In addition, the anatomical relationship of the parapharyngeal space to the carotid sheath places the internal jugular vein and carotid artery at risk of septic thrombophlebitis, aneurysm formation, or vascular occlusion. These associations emphasize why timely recognition and management of such infections are crucial to prevent morbidity and mortality [9][10].

The epidemiologic landscape of deep neck infections is shaped not only by age distribution but also by broader health and social factors. Children with limited access to healthcare, delayed diagnosis, or untreated respiratory tract infections are at higher risk of developing complications. Geographic variation may also influence incidence, as regions with higher prevalence of untreated dental disease or limited antibiotic availability may experience greater rates of deep neck infections. Furthermore, the increase in resistant bacterial strains may complicate management and contribute to the observed rising incidence in recent years. In summary, retropharyngeal and parapharyngeal abscesses, though relatively rare, remain clinically important deep neck infections with a distinct epidemiologic profile. They occur most frequently in children younger than 5 years due to the persistence of retropharyngeal lymph nodes, often after upper respiratory tract infections.

Their rising incidence over the past decade underscores the importance of vigilance, timely diagnosis, and effective management to prevent severe complications such as airway compromise, mediastinitis, and vascular injury [9][10].

Pathophysiology

The pathophysiology of deep neck infections is closely tied to the complex anatomy of the cervical fascial compartments and interfascial spaces. The cervical fascia is broadly categorized into superficial and deep layers, each of which plays a critical role in defining the spread and containment of infections. The superficial fascia corresponds to the subcutaneous tissue of the neck and contains the platysma muscle. It envelops the head and neck in continuity, serving as the most external fascial covering. Beneath this, the deep cervical fascia is organized into three principal layers: superficial, middle, and deep. Understanding these layers is essential to appreciating how infections spread within the neck and beyond [11][12][13][14][15].

The superficial layer of the deep cervical fascia, also known as the investing layer, encircles the submandibular and parotid glands as well as major muscles including the trapezius, sternocleidomastoid, and strap muscles. Infections originating from odontogenic or submandibular sources typically involve this space, most notably the submandibular and masticator compartments. Odontogenic infections of molar teeth, in particular, are a frequent source due to their anatomical drainage pathways into these fascial planes. When left untreated, these infections may extend laterally and inferiorly, causing swelling, pain, and functional impairment of jaw mobility. The middle layer of the deep cervical fascia encases several critical structures of the aerodigestive tract. These include the pharynx, larynx, trachea, upper esophagus, thyroid, and parathyroid glands. Consequently, infections of pharyngeal, tonsillar, and laryngeal origin are most likely to involve this layer, giving rise to pathologies within the parapharyngeal and retropharyngeal spaces. Importantly, odontogenic infections from the second and third molars can also extend inferiorly beyond the dentate line of the mandible, penetrating the middle layer and resulting in retropharyngeal or parapharyngeal abscesses. Such infections pose significant clinical risks due to the proximity of vital structures like the airway, major vessels, and neural elements [11][12][13][14][15].

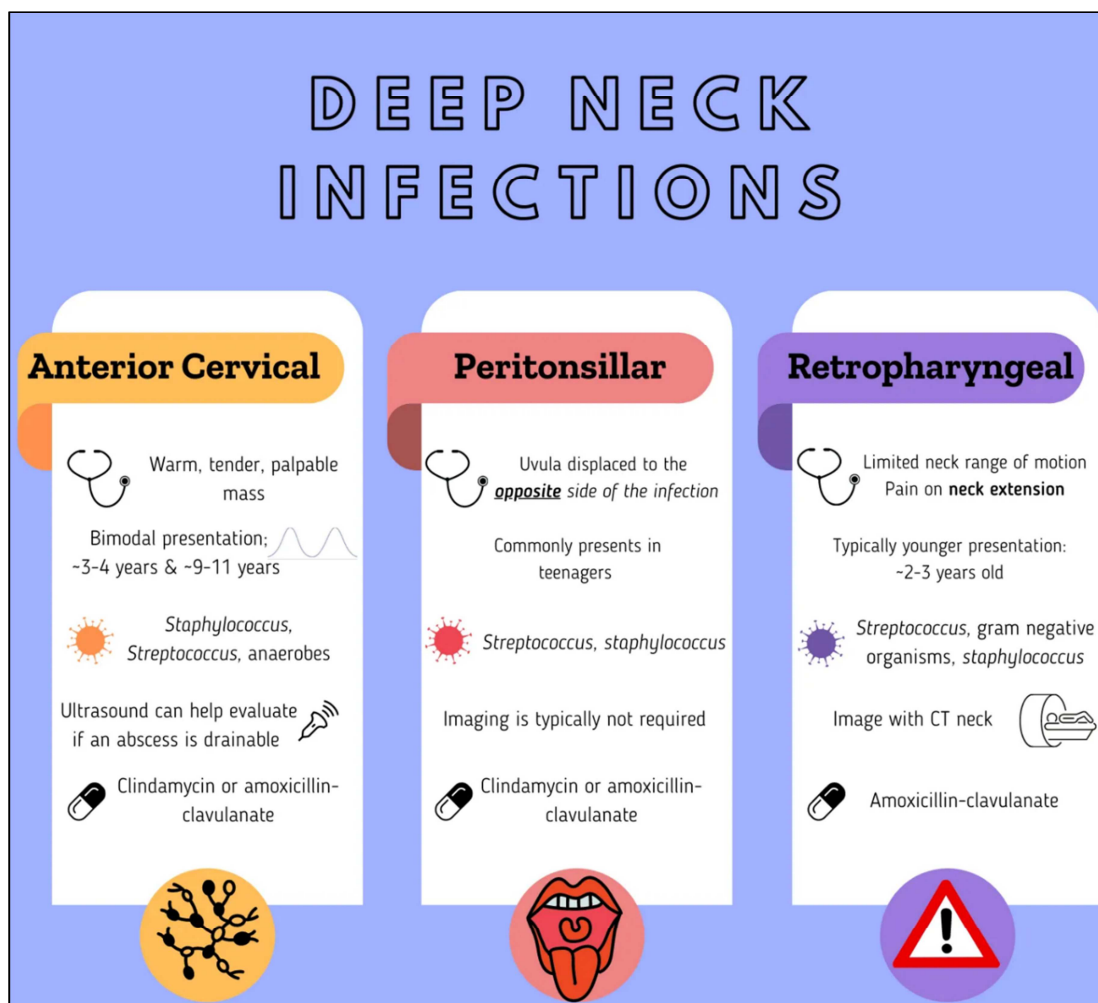


Figure-2: Types of Deep neck infections.

The deepest component of the cervical fascia, known as the prevertebral fascia, surrounds the vertebral column and associated spinal musculature. This layer also encompasses the alar fascia, which lies between the middle layer and the prevertebral fascia proper. The retropharyngeal space terminates at this alar fascia, while the region between the alar and prevertebral layers constitutes the so-called "danger space." This anatomical corridor is of particular clinical importance, as it is in direct continuity with the mediastinum. Infections arising in the upper aerodigestive tract can therefore spread inferiorly through this space into the thoracic cavity, leading to mediastinitis, a potentially fatal complication. Additionally, infections can track into the vertebral column and prevertebral muscles either through direct extension or by hematogenous seeding, particularly in patients with risk factors such as intravenous drug use. This process may result in vertebral osteomyelitis or prevertebral abscesses, conditions that often require aggressive medical and surgical management. Involvement of these fascial spaces is frequently associated with regional lymphadenopathy. The anterior and posterior cervical lymphatic chains are most commonly affected, reflecting the drainage patterns of the head and neck. Host-related factors significantly influence the course and severity of infection. Immunocompromised individuals, such as those with poorly controlled diabetes, HIV infection, or patients receiving immunosuppressive therapy, are particularly vulnerable. Likewise, recent trauma, surgical procedures, or dental interventions can facilitate bacterial entry into deeper fascial planes. Intravenous drug use introduces an additional risk by providing both a source of bacteremia and a direct inoculation route for pathogens into deep tissues [11][12][13][14][15].

A unique manifestation of deep neck infection pathophysiology is Lemierre syndrome, a condition that exemplifies how localized pharyngeal infection can evolve into a systemic and life-threatening illness. The pathogenesis begins with bacterial invasion of the pharyngeal mucosa, often compromised by preceding viral or bacterial pharyngitis. The infection extends into the lateral pharyngeal space, where it involves the internal jugular vein, resulting in septic thrombophlebitis. *Fusobacterium necrophorum* is the most frequently implicated pathogen, though *Fusobacterium nucleatum* has also been identified as a causative agent. The thrombophlebitis allows septic emboli to disseminate hematogenously, with the lungs being the most common site of metastatic infection. Patients may develop pneumonia, pleural empyema, or septic pulmonary emboli. Other less frequent but severe manifestations include ear involvement, cervical abscesses, and systemic septicemia. Given its rapid and aggressive course, Lemierre syndrome requires early recognition and the prompt initiation of appropriate antibiotic therapy, often alongside surgical intervention to control the primary infection focus [16]. In summary, the pathophysiology of deep neck infections is governed by the interplay between cervical fascial compartments and host-related factors. The anatomical continuity of fascial planes allows for rapid extension of infections from odontogenic, pharyngeal, or laryngeal origins into spaces that directly communicate with the mediastinum and vascular structures. The presence of immunosuppression, diabetes, recent procedures, or intravenous drug use exacerbates this process, contributing to more severe and complicated disease courses. Lemierre syndrome represents one of the most striking examples of these infections progressing to systemic disease, reinforcing the need for early clinical suspicion and aggressive management strategies. Understanding these mechanisms remains crucial for preventing morbidity and mortality in patients with deep neck infections [16].

History and Physical

Clinical presentations of deep neck infections vary according to the anatomic origin, the fascial compartment involved, the degree of inflammatory change, and the presence or absence of a discrete abscess. Most patients report fever and localized neck pain. Additional symptoms frequently provide topographic clues. Dental pain, difficulty swallowing, noisy breathing, changes in voice, reduced mouth opening, pain with neck motion, and escalating respiratory distress each help localize the process. A careful history should probe for factors that raise the index of suspicion for deep extension. Recent dental care, oropharyngeal procedures, penetrating trauma, and neck surgery create portals of entry. Systemic conditions such as poorly controlled diabetes, immune suppression, and intravenous drug use predispose to rapid spread and atypical pathogens. The clinician must also ask about the timing, severity, and progression of symptoms, because early recognition improves outcomes [17][18][19][20][16].

On examination many patients appear systemically unwell. Vital signs commonly show fever and tachycardia. Inspection of the neck often reveals asymmetry, swollen soft tissues, erythema, and localized induration. Regional lymphadenopathy is common and may follow the lymphatic drainage of involved structures. Torticollis can occur when pain limits neck motion or when spasm develops in adjacent musculature. Because the implicated spaces lie beneath firm fascial layers and muscular envelopes, fluctuance is often absent even when a sizeable collection exists. This feature reduces the utility of palpation alone and increases reliance on imaging for definitive diagnosis. Clinicians should maintain a low threshold for cross-sectional imaging so that patients can be evaluated supine and measures to secure the airway are instituted when necessary. Proximal deep neck infections, including peritonsillar, parapharyngeal, parotid, and submandibular processes, tend to produce oropharyngeal symptoms. A sore throat is frequent. Trismus develops when inflammation involves the muscles of mastication or when adjacent masticator spaces are involved. Visible swelling of the lower face or neck, local redness, tenderness to touch, and ipsilateral lymphadenitis are typical exam findings. Peritonsillar collections characteristically displace the soft palate and uvula toward the midline; asymmetry of the tonsillar pillars heightens suspicion for a peritonsillar abscess. Medial bulging of the lateral pharyngeal wall suggests parapharyngeal space involvement. Local pressure effects produce odynophagia and dysphagia. Inflammation may involve the cricoarytenoid joints and thereby impair vocal fold motion. When the vagus nerve is affected, dysphonia or a muffled "hot potato" voice may be evident [17][18][19][20][16].

Infections that involve the submandibular or sublingual spaces often trace to molar dental sources or salivary gland disease. These presentations can progress rapidly and carry a high risk of airway compromise. Ludwig angina denotes a diffuse cellulitis of the submandibular space classically originating from infected lower molars. The hallmark features include firm elevation of the floor of the mouth, limited tongue mobility, drooling, severe dysphagia, and progressive induration without a well-circumscribed fluctuant mass. Trismus and respiratory compromise may follow. The absence of fluctuance and

the deep location of the infection require a high index of suspicion and urgent airway planning. Peritonsillar abscesses localize between the tonsillar capsule and superior constrictor muscle. Patients present severe unilateral throat pain, painful swallowing, high fever, and difficulty handling secretions. Trismus complicates airway management and can make oropharyngeal access difficult. Although group A beta-hemolytic streptococci are commonly isolated, microbiology does not typically alter the immediate diagnostic or procedural approach. Drainage, airway assessment, and empiric antimicrobial therapy directed against aerobic streptococci and anaerobes constitute the initial steps [17][18][19][20][16].

Retropharyngeal and parapharyngeal abscesses are more frequent in younger children and commonly follow upper respiratory infections that spread to regional lymph nodes. These infections often present difficulty swallowing, drooling, neck stiffness, and stridor. The risk of airway compromise is real and can develop insidiously. The retropharyngeal compartment communicates with the danger space and the mediastinum, so downward extension can produce life-threatening mediastinitis. Clinicians must monitor signs of chest involvement and systemic sepsis. When the danger space is involved, the loose areolar tissue allows rapid propagation of infection, so a stable appearance at presentation may deteriorate quickly. Parapharyngeal infections may precipitate grave vascular and neurologic complications. Septic thrombosis of the internal jugular vein, septic emboli to the lungs, disseminated intravascular coagulation, and carotid artery pseudoaneurysm have been described. Extension to cervical vertebrae can produce osteomyelitis, epidural abscess, atlanto-axial subluxation, spinal cord abscess, and meningitis. These severe sequelae underscore the need to evaluate neurologic deficits, altered mental status, and focal signs that might indicate deep extension beyond the neck [17][18][19][20][16].

Lemierre syndrome exemplifies the potential for localized pharyngeal infection to seed distant sites. Classically following an antecedent pharyngitis, the infection invades the lateral pharyngeal space and causes septic thrombophlebitis of the internal jugular vein. *Fusobacterium necrophorum* is the organism most frequently implicated, with *Fusobacterium nucleatum* also reported. Septic emboli commonly seed the lungs, producing necrotic cavitory lesions, multifocal infiltrates, pleural effusions, empyema, or lung abscesses. Systemic spread can also produce soft tissue abscesses, hepatic or splenic lesions, endocarditis, renal abscesses, and brain abscesses. Clinicians should recognize that *Fusobacterium* species often show resistance to macrolides, fluoroquinolones, tetracyclines, and aminoglycosides, which has implications for empiric antibiotic selection. Early recognition, blood culture acquisition, and prompt initiation of appropriate antimicrobials are essential to reduce morbidity and mortality. Through evaluation the clinician must pair focused history and directed physical examination with timely imaging. The depth and complexity of these infections frequently mask overt surface findings. Complete assessment requires attention to airway patency, the trajectory of symptom progression, and the presence of systemic toxicity. Documentation should include dentition status, recent dental or oropharyngeal interventions, and any immunosuppressive conditions. Early involvement of surgical and anesthetic teams is appropriate when airway compromise or rapid progression is suspected. Prompt drainage of accessible collections, combined with targeted antimicrobial therapy, mitigates the risk of the life-threatening complications described above and improves the likelihood of full recovery. [17][18][19][20][16]

Evaluation

Assessment of deep neck infections requires integration of clinical, laboratory, and imaging data to define disease severity, identify drainable collections, and detect complications early. Inflammatory biomarkers provide objective measures of host response and have prognostic and triage value in this setting. A typical laboratory pattern includes leukocytosis with a left shift; however, more refined indices derived from the complete blood count have gained traction. Ratios such as the neutrophil to lymphocyte ratio (NLR) and the platelet to lymphocyte ratio (PLR) reflect the balance between innate inflammatory activation and adaptive immune reserve. In deep neck infections an elevated NLR corresponds with greater inflammatory burden and a higher likelihood of requiring incision and drainage; a cut-off value exceeding 8.02 has been proposed as a predictor for operative management. Additional inflammatory markers provide complementary information: elevated C-reactive protein (CRP) above 41.25 mg/L and an erythrocyte sedimentation rate (ESR) above 56.6 mm/h have been associated with more severe disease and the need for procedural intervention. These markers assist clinicians in identifying patients at risk of progression to sepsis or of harboring established abscesses that will not respond to antibiotics alone. [21]

A complete diagnostic evaluation also incorporates basic metabolic testing because dehydration and electrolyte disturbances are commonly complicate presentation when oral intake is poor due to pain and dysphagia. Blood cultures should be obtained in patients showing systemic toxicity or sepsis physiology before initiating broad-spectrum therapy, and specimens of any purulent discharge should be sent for gram stain, aerobic and anaerobic culture, and sensitivity testing to refine antimicrobial selection. Although cultures may be negative after prior antibiotic exposure, positive results direct narrowing of empiric regimens and identify resistant or unusual pathogens in immunocompromised hosts. Historically, plain radiography had a role in initial assessment, particularly in children, where lateral neck films could suggest retropharyngeal swelling when prevertebral soft tissue measurements exceed standard thresholds. A prevertebral soft tissue width greater than 7 mm at C2 or 14 mm at C6 in children, and greater than 22 mm at C6 in adults, raises suspicion for a retropharyngeal process on lateral radiographs. Plain films may also reveal foreign bodies or subcutaneous emphysema and maintain a role in screening when history is unreliable. Nevertheless, the diagnostic yield of radiographs is limited compared with cross-sectional imaging, and their routine use has declined in favor of modalities that more accurately distinguish cellulitis from abscess and delineate anatomic relationships for safe drainage. Chest radiography retains value when extension to the mediastinum, pneumomediastinum, or lower thoracic complications such as empyema are concerns. [17][22]

Ultrasonography offers rapid bedside evaluation for superficial collections and is particularly useful for peritonsillar abscesses and cervical adenitis. Its advantages include immediacy, absence of ionizing radiation, and the ability to guide percutaneous aspiration or drainage in real time. Ultrasound can reduce the need for contrast-enhanced CT in select pediatric patients, thereby limiting radiation exposure. However, ultrasound is operator dependent and limited by the depth of the lesion; it cannot reliably visualize retrodeep spaces such as the danger space or determine mediastinal extension. When ultrasound findings are equivocal or when deep space involvement is suspected, cross-sectional imaging is indicated. [23][24]

Contrast-enhanced computed tomography (CECT) is the imaging standard for defining the presence, size, and extent of deep neck abscesses. The addition of intravenous contrast markedly improves discrimination between cellulitis and frank pus collections and demonstrates relationships to critical structures such as the carotid sheath, internal jugular vein, airway, and mediastinum. CECT detects gas within a collection, identifies multiloculated pockets, and maps extension along fascial planes, information that directly informs surgical approach and the need for multidisciplinary involvement. Non-contrast CT lacks this discriminative power and should be avoided when the clinical question is whether an abscess is present and amenable to drainage. In unstable patients or when iodinated contrast is contraindicated, CT remains useful for gross anatomical evaluation but with acknowledged limitations. [25]

Magnetic resonance imaging provides superior soft tissue contrast and can better differentiate abscess from phlegmon, lymphadenopathy, and venous thrombosis without ionizing radiation exposure. MRI is particularly helpful when vascular or neural involvement is suspected, when clinical deterioration persists despite negative or equivocal CT, or when delineation of extension into the skull base or craniovertebral junction is required. The principal drawbacks of MRI in the acute setting are limited availability, longer acquisition times, higher cost, and logistical challenges such as the need for sedation in uncooperative children or in claustrophobic adults. Consequently, MRI serves as a problem-solving tool and adjunct rather than the first-line emergency study. [26][25][27][25] Imaging interpretation must be contextualized with clinical findings. Radiographic evidence of a rim-enhancing collection with central low attenuation on CECT correlates with pus and favors immediate drainage. Conversely, ill-defined soft tissue stranding without a discrete fluid collection suggests cellulitis that may respond to medical therapy. Measurements of collection size, proximity to vascular structures, and presence of septations influence the choice between percutaneous image-guided drainage and open surgical approaches. When vascular involvement is suspected, additional studies such as venous duplex ultrasound or CT venography may be required to detect internal jugular vein thrombosis that would alter both antimicrobial strategy and anticoagulation considerations [26][25][27][25].

Decision pathways that integrate biomarker thresholds, clinical severity, and imaging results improve triage and therapeutic planning. Elevated NLR, CRP, or ESR in conjunction with fever, toxemia, airway compromise, or radiologic evidence of abscess should lower the threshold for operative intervention. In contrast, patients with modest inflammatory markers, stable airway, and imaging consistent with phlegmon without a drainable collection may be managed initially with broad-spectrum antibiotics and close serial reassessment, including repeat imaging when clinical response is suboptimal. Finally, ongoing monitoring of inflammatory markers and serial imaging guides the response to therapy. Declining CRP and NLR values parallel clinical improvement and may support de-escalation of antimicrobials or avoidance of delayed intervention. Persistent or rising biomarkers despite appropriate source control mandate re-evaluation for missed loculations, inadequate drainage, vascular complications, or atypical infectious agents. This iterative process—combining clinical judgment, biomarkers, and targeted imaging—optimizes outcomes and reduces the risk of catastrophic complications in patients with deep neck infections [26][25][27][25].

Treatment / Management

Management of deep neck infections combines targeted antimicrobial therapy, timely drainage of purulent collections, and intensive supportive care. Empiric therapy should begin promptly after obtaining cultures when feasible. Regimens must cover the spectrum of likely pathogens originating from the oral cavity and upper airway, including gram positive cocci, gram negative rods, and anaerobes. Common initial intravenous options include nafcillin or vancomycin when staphylococcal coverage is required, paired with an aminoglycoside such as gentamicin or tobramycin for gram negative coverage. Broader single agent options that provide mixed aerobic and anaerobic activity include ampicillin-sulbactam and clindamycin. In settings with a high prevalence of methicillin resistant *Staphylococcus aureus* empiric coverage should include agents active against MRSA such as vancomycin or linezolid, often combined with a broad spectrum beta lactam such as cefepime. Carbapenems and piperacillin-tazobactam also serve as effective empiric choices for severe or polymicrobial disease. Local resistance patterns and individual risk factors such as prior antibiotics, immunosuppression, and intravenous drug use must guide selection and early de-escalation when culture results permit [28][16][4].

Duration of antimicrobial therapy depends on the anatomic site, the presence of a drainable abscess, and the patient response. For parapharyngeal, retropharyngeal, and prevertebral infections the usual intravenous course lasts two to three weeks, with extension for complications such as mediastinitis, vertebral osteomyelitis, or persistent bacteremia. Step down to oral agents may occur once clinical improvement is clear, oral intake is reliable, and the infecting organisms display oral-suitable susceptibility. Infectious disease consultation improves regimen selection and duration decisions in complex cases or when unusual pathogens are recovered. Cultures from aspirated pus and blood refine therapy and permit narrowing to focused agents to minimize toxicity and resistance. Surgical management is dictated by the presence of a discrete fluid collection, the anatomic location, and the clinical trajectory. Abscesses that are radiologically defined and clinically significant typically require drainage because antibiotics alone rarely penetrate a mature purulent cavity. Drainage can be performed by percutaneous image guided aspiration when the collection is accessible, unilocular, and in a safe corridor relative to vascular structures. Deep or multiloculated collections, or those adjacent to critical neurovascular elements, often mandate open or endoscopic drainage performed by an experienced head and neck surgeon. The choice between intraoral and transcervical approaches rests on abscess position, surgeon expertise, and airway considerations. Recurrent or multiloculated disease may require repeat procedures and prolonged drainage with indwelling catheters until the cavity collapses and resolves [28][16][4].

Airway control remains the first priority when upper airway compromise is suspected. Indications for early elective airway protection include progressive stridor, severe or worsening odynophagia with inability to manage secretions, and rapid neck swelling that threatens the laryngopharyngeal lumen. Awake fiberoptic intubation is frequently the safest approach because distorted anatomy and trismus may render direct laryngoscopy unsafe. When intubation fails or is judged unsafe, surgical airway access should be planned in coordination with anesthesia and surgical teams. Securing the airway before transport to imaging or to the operating room prevents catastrophic obstruction during diagnostic or therapeutic maneuvers.

The role of adjunctive therapies warrants careful judgment. Corticosteroids may reduce edema and temporize airway compromise but lack robust outcome data and may mask clinical signs of progression. Their routine use is not supported by high quality evidence; they remain an option in select cases after multidisciplinary discussion. Analgesia, aggressive hydration, and nutritional support are essential components of early care. Patients with sepsis require standard resuscitation and organ support as indicated, including vasopressors and ventilatory assistance [28][16][4].

Lemierre syndrome requires prolonged, coordinated therapy. Empiric regimens should include agents active against anaerobes and beta lactamase producing organisms. Metronidazole remains a preferred agent for *Fusobacterium* species because of its bactericidal activity and tissue penetration. Carbapenems and beta lactam-beta lactamase inhibitor combinations provide alternative choices. Typical treatment duration ranges from three to six weeks, tailored to clinical course and radiologic resolution. Surgical drainage of identified foci aids source control. Management may also require imaging guided or surgical intervention for septic emboli and pulmonary complications. The role of anticoagulation for internal jugular vein thrombosis remains controversial; decisions should be individualized and involve vascular medicine and infectious disease specialists. Interventional radiology plays a central role in both diagnosis and therapy. Image guided needle aspiration can confirm the diagnosis, obtain cultures, and provide therapeutic decompression for select collections. Catheter drainage under CT or ultrasound guidance offers a less invasive alternative to open surgery in appropriately selected cases. Radiologic guidance is also valuable for repeat drainage, for accessing deep or posterior collections, and for therapeutic irrigation when loculations are present [28][16][4].

Management pathways must incorporate strategies to prevent recurrence and to address predisposing sources. Dental evaluation and prompt treatment of odontogenic foci are essential in adults with tooth related infections. Tonsillar and adenoidal sources in children should be addressed when present. Removal of foreign bodies, debridement of necrotic tissue, and correction of anatomic contributors such as sialadenitis or salivary duct obstruction reduce the risk of relapse. Optimization of underlying medical disorders such as diabetes improves host defense and reduces reinfection risk. Monitoring and follow up require serial clinical assessment and selective repeat imaging. Clinical improvement should be evident within 24 to 48 hours of adequate drainage and targeted antibiotics. Failure to improve mandates reassessment with contrast enhanced CT or MRI to identify residual loculations, new collections, vascular complications, or extension into the mediastinum or skull base. Laboratory markers such as white blood cell count, CRP, and procalcitonin may assist in tracking response but should not replace clinical judgment [28][16][4].

Prevention strategies include public health measures to improve dental access and to reduce untreated or poorly managed oropharyngeal infections. Inpatients, adherence to protocols that limit indwelling devices and prompt review of head and neck instrumentation reduce iatrogenic risk. Education of clinicians to recognize early signs and to involve airway and surgical teams reduces delay to definitive therapy and improves outcomes. In summary, care of deep neck infections integrates prompt empiric antimicrobials, timely source control via drainage when indicated, airway protection, and aggressive supportive care. Choice of specific agents should reflect likely organisms and local susceptibility patterns. Surgical and radiologic interventions are tailored to anatomic factors and patient stability. Multidisciplinary collaboration among otolaryngology, infectious disease, anesthesia, interventional radiology, and critical care optimizes timing and modality of interventions and reduces morbidity and mortality. [28][16][4]

Differential Diagnosis

The differential diagnosis of deep neck infections is often challenging because their clinical presentation overlaps with numerous other conditions that also cause fever, neck pain, and airway or swallowing difficulties. Establishing a clear distinction is essential, as delay in identifying the true underlying disorder can lead to severe complications. The presence of fever with neck pain, for instance, is not specific to deep neck infections. Such symptoms can also indicate meningitis, where associated features such as photophobia, altered mental status, and nuchal rigidity are more prominent. Similarly, apical pneumonia can present with referred pain to the neck due to diaphragmatic irritation, while subarachnoid hemorrhage must also be considered, especially when accompanied by acute severe headache, neurologic deficits, or signs of meningeal irritation [28].

Acute neck pain with visible asymmetry or deformity should raise suspicion for traumatic causes such as cervical fractures, dislocations, muscle hematomas, or strains. These conditions typically present a history of trauma or sudden onset after exertion, but without the systemic signs of infection. Radiologic assessment is crucial in differentiating structural and traumatic etiologies from infectious processes. In patients who present with stridor, deep neck infections must be distinguished from acute epiglottitis, bacterial tracheitis, and viral croup. These conditions can all result in upper airway obstruction but differ in progression and therapeutic approach. Acute epiglottitis, for example, is often abrupt in onset and rapidly progressive, requiring immediate airway protection, while croup is usually more gradual and viral in nature. Dysphagia and odynophagia, which are hallmark features of deep neck infections, are also shared with other important conditions. Foreign body ingestion can mimic infection, particularly when impaction occurs in the pharynx or esophagus. Acute esophagitis, either infectious or chemical in origin, may also present with painful swallowing and should be excluded with appropriate endoscopic evaluation. Finally, clinicians must consider malignancy as part of the differential diagnosis when evaluating a patient with a neck mass or swelling. Neoplasms of the oropharynx, larynx, thyroid, or cervical lymph nodes can mimic deep neck abscesses by producing progressive enlargement, pain, or secondary infection. Failure to recognize an underlying tumor can delay definitive oncologic management [28].

Prognosis

Outcomes for deep neck infections vary widely and hinge on host defenses, the anatomic site and extent of disease, and the promptness and appropriateness of intervention. Immunocompetent patients with localized infections that are recognized early and treated with targeted antibiotics and timely drainage usually recover without long-term sequelae. In contrast, patients with compromised immunity, uncontrolled diabetes, advanced age, delayed presentation, or infections that

involve threatening fascial planes carry substantially higher risk. Reported case fatality rates in the literature vary dramatically, ranging from approximately 1 percent in series of promptly treated, limited infections to as high as 25 percent when disease presents late, is extensive, or is complicated by mediastinitis, septic shock, or other systemic consequences. Thus, prognosis should be framed as a continuum shaped by individual patient factors, microbiology, and the rapidity of source control [29]. Clinical trajectory often unfolds over days, with deterioration driven by local complications—airway compromise, vascular erosion, or extension into the mediastinum—and by systemic processes such as bacteremia and sepsis. Even when initial therapy appears effective, residual loculations, undrained collections, or unrecognized vascular complications can prolong illness and increase morbidity. Functional recovery depends on resolution of infection and on the preservation or restoration of critical structures; injuries to cranial nerves, osteomyelitis of the cervical spine, or scarring of soft tissues may produce persistent deficits that affect swallowing, phonation, or neck mobility. Long-term follow up should therefore assess for both infectious resolution and the functional consequences of deep infection and of any necessary interventions [29].

Complications

Deep neck infections may produce a cascade of local, regional, and systemic complications. Lateral pharyngeal space infections may extend into the carotid sheath, causing septic thrombophlebitis of the internal jugular vein and local vascular erosion; this process underlies Lemierre syndrome and predisposes to septic embolization. Retropharyngeal or danger-space infections may track inferiorly into the posterior mediastinum, producing acute mediastinitis; mediastinal involvement frequently heralds a fulminant course with potential progression to empyema and pericarditis. Airway obstruction remains a common and immediate threat whenever submandibular, parapharyngeal, or retropharyngeal collections produce mass effect; progressive compromise can culminate in respiratory failure and the need for urgent airway intervention. Systemic spread with bacteremia and sepsis may lead to multiorgan dysfunction. Spread to bone and central nervous system structures can produce cervical osteomyelitis, epidural abscess, meningitis, or brain abscess. Pulmonary complications from septic emboli or direct extension include necrotizing pneumonia, pleural empyema, and acute respiratory distress syndrome. Vascular sequelae such as carotid artery pseudoaneurysm or rupture are rare but catastrophic. The full spectrum of complications underscores the need for vigilant monitoring and rapid multidisciplinary response [29].

Consultations

Surgical consultation with head and neck specialists is integral to management because many patients ultimately require operative drainage or debridement. Early engagement of otolaryngology or head and neck surgery expedites decisions about the feasibility of intraoral versus transcervical approaches, the need for repeat procedures, and strategies for source control when complex anatomy or mediastinal extension is present. Anesthesia and critical care teams should be involved preemptively in patients who display any signs of airway compromise or systemic instability, since elective planning for awake fiberoptic intubation or for a surgical airway can be lifesaving. Infectious disease consultation refines empiric antimicrobial choices and the duration of therapy, particularly in the setting of unusual pathogens, resistant organisms, or syndromes such as Lemierre disease. Interventional radiology provides important options for image-guided aspiration or catheter drainage, especially for deep or posterior collections. Thoracic surgery consultation becomes necessary when mediastinal extension, empyema, or cardiac complications occur. Effective care relies on early, coordinated input from these disciplines [29].

Patient Education

Prevention centers on limiting the antecedent sources that seed deep neck spaces. Routine oral hygiene, timely dental care, and early management of dental caries and periodontal disease reduce the incidence of odontogenic seeding. Prompt evaluation and appropriate treatment of pharyngitis, tonsillitis, and upper respiratory tract infections limit spread to regional lymphatics and fascial planes. Patients should be counseled to seek immediate care for progressive throat pain, fever, dysphagia, voice changes, or respiratory symptoms. Education should stress the importance of completing prescribed antibiotic courses for oropharyngeal infections when indicated and of adhering to follow-up instructions after dental procedures or head and neck instrumentation. For high-risk populations, including persons with diabetes or immunosuppression, clinicians should emphasize close monitoring and low thresholds for early evaluation of concerning symptoms [29].

Other Issues

Clinicians managing deep neck infections must remember several practical points. Most deep neck infections originate from adjacent foci such as odontogenic, tonsillar, or pharyngeal sources; identifying and addressing the primary source is essential to prevent recurrence. These infections are typically polymicrobial, reflecting oral and oropharyngeal flora; empiric antimicrobial regimens should therefore cover aerobic streptococci, staphylococci, gram-negative rods, and anaerobes until culture data permit de-escalation. Thorough knowledge of neck fascial anatomy and potential spaces is indispensable for predicting routes of spread and planning safe drainage. Contrast-enhanced CT is the preferred emergency imaging modality because it discriminates abscess from cellulitis and defines relationships to major vessels and the airway; non-contrast scans have limited utility in this context. Small or equivocal collections may respond to aggressive medical therapy, but most established abscesses require drainage—either percutaneous, intraoral, or external. Finally, clinicians should not underestimate the disease's potential severity; symptoms that suggest airway involvement, mediastinal extension, or systemic toxicity warrant urgent escalation of care [29].

Enhancing Healthcare Team Outcomes

Optimal outcomes follow from structured interprofessional collaboration. Emergency physicians and primary care clinicians play a pivotal role in early recognition and stabilization. Otolaryngologists and head and neck surgeons provide expertise in airway management and definitive surgical drainage. Anesthesiologists contribute critical airway planning and execute complex intubations or surgical airway access. Infectious disease specialists guide empiric and culture-directed antimicrobial strategies and determine treatment duration. Radiologists support diagnosis and procedural planning through timely interpretation of contrast-enhanced CT and guidance for image-guided drainage. Interventional radiology offers minimally invasive drainage alternatives that can reduce operative morbidity in selected cases. Intensivists and critical care nurses manage organ support when sepsis or respiratory failure ensues. Pharmacists optimize antimicrobial dosing, monitor interactions, and assist with stewardship decisions. Dietitians and rehabilitation specialists support recovery, particularly when prolonged hospitalization or deconditioning occur. Efficient communication among team members is essential. Regular multidisciplinary case review enables real-time adjustment of the care plan, early identification of complications, and coordinated transitions from parenteral to oral therapy and from inpatient to outpatient follow-up. Standardized clinical pathways that integrate biomarker thresholds, imaging criteria, and airway management algorithms can reduce delays to source control and limit variability in practice. Education and simulation training for emergent airway scenarios strengthen team performance. Finally, engaging patients and families in understanding warning signs, the rationale for interventions, and the importance of dental and medical follow-up reduces the risk of recurrence and supports timely presentation for future symptoms. Together, these measures enhance patient safety, shorten time to definitive care, and improve the overall prognosis, which remains contingent on patient factors, infection severity, and the rapidity of effective intervention [29].

Conclusion:

Deep neck infections remain a formidable clinical challenge due to their complex anatomy and potential for rapid deterioration. A successful outcome hinges on early recognition, precise diagnostic evaluation, and aggressive, multidisciplinary management. The initial priority must always be a thorough and continuous assessment of the airway, with a low threshold for elective, controlled intubation to prevent catastrophic obstruction. Contrast-enhanced computed tomography is the imaging modality of choice, providing critical details on the location, size, and extent of the infection, thereby guiding therapeutic decisions. While broad-spectrum antibiotics targeting the polymicrobial oral flora are essential, the definitive treatment for most established abscesses is timely drainage, either surgically or percutaneously. The prognosis varies significantly, with immunocompromised patients or those with delayed presentations facing higher risks of severe complications like mediastinitis, septic thrombophlebitis, and sepsis. Therefore, a coordinated effort involving emergency physicians, otolaryngologists, anesthesiologists, radiologists, and infectious disease specialists is paramount. This collaborative approach ensures prompt source control, optimal antibiotic stewardship, and vigilant supportive care, ultimately reducing morbidity and mortality associated with these serious infections.

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