

Deep Neck Infections and Descending Necrotising Mediastinitis : Multidisciplinary Approach. A Review

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Abstract

Untreated or non-responsive to treatment deep neck infections, often arising from odontogenic, oropharyngeal or cervical foci, can result in life-threatening complications of airway obstruction, systemic sepsis and descending necrotising mediastinitis (DNM). DNM is a severe, potentially fatal infection, characterized by high morbidity. It is commonly polymicrobial, spreading downward from head and neck sources through the fascial planes into the mediastinum. Despite advances in diagnosis and treatment, the fulminant course, rarity, and subtle presentation at the beginning of DNM still contribute to mortality rates of 11 % - 40 %. Survivors require prolonged hospital stay. Earlier diagnosis is essential to allow more timely airway management and aggressive transcervical and transthoracic debridement and drainage, involving imaging surveillance and repeated intervention(s) to drain residual or persistent collections, all within a multidisciplinary care framework to reduce mortality and morbidity in DNM patients. The aim of the study was to provide a comprehensive review of current knowledge on these still underrecognised conditions, raise clinical awareness, and emphasize the importance of rapid diagnosis and vigorous treatment.

Keywords: Deep Neck Infection, Cervical Necrotising Fasciitis, Descending Necrotising Mediastinitis, Aggressive Management, Cervicotomy, Thoracotomy.

Introduction

Deep neck infection (DNI) most often arise from dental or tonsillar bacterial sources, and may have a subtle onset developing in the potential spaces and fascial planes of the head and neck [1, 2]. A critical concern in the progression of purulent DNI is the possible obstruction of the airway passage (e.g., from deep neck abscess / DNA) and the rapid downward spread of infection from the neck into the mediastinum [3]. This latter condition - descending necrotising mediastinitis (DNM) - is extremely acute, highly lethal, and represents a major complication of odontogenic, oropharyngeal or cervicofacial infections. It spreads through the contiguous deep spaces of the neck and may involve the mediastinum, pleural spaces, pericardium, and even the abdominal cavity [4-9].

DNM, typically polymicrobial and synergistic, can lead to dramatic clinical deterioration within hours causing necrosis, sepsis, shock, multi-organ failure (MOF), and death. If timely

surgical treatment is not achieved, the evolution of disease leads to death [3, 4, 7, 10, 11]. Despite advances in anaesthesia, critical care, antimicrobial therapy, imaging diagnostics, and surgery, overall mortality of DNM remains high at 11 %-40 %, and even higher in certain patient populations [10- 13]. Early diagnosis and multidisciplinary management are crucial to reduce the fatality associated with DNM [14].

Current evidence on risk factors, etiology and outcome varies widely, likely because most studies include very small cohorts (fewer than 10 cases). Due to the aggressive nature of DNM, the optimal management strategy is still debated [15]. Medical responsibility may be scrutinized postmortem, and medicolegal implications may arise [16].

Methods

A comprehensive current literature review on DNI and DNM was conducted using MEDLINE, PubMed and Google Scholar.

ar. Search terms included “oropharyngeal infection”, “deep neck infection”, “cervical necrotising fasciitis”, and “descending necrotising mediastinitis”. Data regarding pathophysiology with anatomical considerations, diagnostic approaches, risk factors, treatment strategies, and survival rates were evaluated.

Discussion

Epidemiology

In the United States, the incidence of necrotising fasciitis (NF) is approximately 0.4 cases per 100,000 individuals [8]. An estimated 3-10 % of all NF cases involve the head and neck region [17, 18]. Around 1 % of severe odontogenic infections progress to cervical NF (CNF), and 1.5 %-3.6 % of DNIs evolve into DNM [19]. DNM can affect all age groups, and there is a moderate male predisposition.

Anatomical Considerations and Pathophysiology

DNIs tend to remain localized (for longer times in immunocompromised, diabetics, corticosteroid-recipients, elderly), and then, if not timely recognized and treated, may turn into DNA formation or extend into more diffuse infection. An exceptional and extremely severe subset of diffuse suppurative infections is the CNF, a polymicrobial infection characterized by rapid destructive spread along the deep fascial planes of the neck. In such cases, commensal bacteria of the oropharyngeal and dental flora may become highly invasive and virulent, triggering the systemic inflammatory response syndrome (SIRS). Synergistic interactions between multiple microorganisms often produce clinically significant symptoms within 24-48 hours after the onset of infectious process. Suppurative descending mediastinitis is differentiated from mediastinitis complicating sternotomy for cardiothoracic surgery, esophageal perforation or trauma, ingestion of foreign body, or neoplastic processes, as the latter originates primarily within the mediastinum itself. A localized DNI may spread through contiguity, haematogenously, or via lymphatics, and even with inoculation (e.g., in case of cerebral abscess) [20].

The deep cervical fascia, composed of three layers, creates multiple compartments, crypts, and spaces, while enclosing the contents of the neck. These fascial planes are continuous with those in the mediastinum. The complex fascial framework of the neck can confine and guide the spread of suppurative infection, unless its natural resistance is overcome [21]. Although the value of this anatomical containment has been debated, since spread of infection does not always follow predictable tissue planes, these considerations remain clinically relevant for treatment planning and for guarding against potential complications of DNIs.

DNIs and DNMs may originate from any oropharyngeal or cervical structure, with dental and tonsillar sources being most common [22]. Odontogenic infections, particularly those involving mandibular molars, provide bacteria with direct access from the oral cavity into the deep neck spaces. Recent literature indicates a shift from odontogenic to (oro)pharyngeal sources, and in up to 50 % of cases the primary DNI origin remains unknown [23]. The causative organisms vary, depending on the origin, although most infections are polymicrobial involving both aerobes and anaerobes.

Before reaching the mediastinum, DNIs can disseminate within muscles of the neck, submandibular and thyroid glands, and the neurovascular bundle, causing pharyngeal and tongue edema. These changes can result in early airway compromise and tracheal deviation / obstruction. Ludwig's angina, a complication of severe oropharyngeal infection, mostly odontogenic, may involve the bilateral submandibular and sublingual spaces as well as the submental space.

Major anatomical pathways allow suppurative material and ruptured abscesses to descend into the mediastinum: (i) pretracheal route (→ anterior mediastinum); (ii) lateral pharyngeal route (→ middle mediastinum); and (iii) retropharyngeal route -“danger space” (→ posterior mediastinum). Additional spread can occur through the prevertebral and carotid sheath spaces which also provide direct extension into the mediastinum [24]. In DNMs, necrotic infectious process, using the contiguous deep spaces as portals of entry, may extend beyond the mediastinum and pleural spaces to involve even the abdomen [4]. Spreading downward is promoted by the negative intrathoracic pressure, respiration, and gravity [25]. The development of DNM is further facilitated by reduced tissue oxygenation and impaired immune responses.

Pathological features of DNM include tissue edema, fluid accumulation, abscess formation, and tissue liquefactive necrosis, similar to necrotic infectious process (NF) in other regions of the body. Pleural and pericardial effusions, empyema's, as well as pneumonitis are common. Infection may involve blood vessels, leading to thrombosis or catastrophic haemorrhage, such as jugular vein thrombosis, disseminated intravascular coagulation, carotid or jugular rupture, cardiac tamponade, and intrathoracic bleeding. Patients may experience acute respiratory distress (ARDS), severe sepsis, multiple organ dysfunction (MOD), MOF, and ultimately death.

Classification of DNM

DNM is classified according to the extent of infectious spread as demonstrated on computed tomography (CT) [26]. In 1999, Endo and colleagues proposed the following classification of the necrotic infection: (i) Type I: infection confined to the superior mediastinum above the tracheal carina (localized DNM); (ii) Type IIA: infection extending into the lower anterior mediastinum (diffuse); and (iii) Type IIB: infection involving both anterior and posterior compartments of the lower mediastinum (diffuse). This classification does not distinguish infections isolated solely to the posterior mediastinum [27].

A more recent system, introduced by Guan in 2021, divides the mediastinum into superior and inferior mediastinum at the level of the tracheal bifurcation: (i) Type Ia, anterosuperior mediastinal infection; (ii) Type I, anterior mediastinal infection; (iii) Type II, posterior / posterosuperior mediastinal infection; and (iv) Type III, infection involving the entire mediastinum. Sugio and colleagues analysed 225 DNM patients from 131 institutions, and proposed another classification involving type I for infections above the carina and types IIA (anterior), IIB (anterior and posterior), and IIC (posterior) for the lower infections [28].

Microbiology

Neck purulent collections and abscesses are produced by bacteria normally present in the pharynx and oral cavity. DNIs that progress to fulminant NF and DNM typically contain mixed polymicrobial flora, including both aerobic and anaerobic species. Monomicrobial infections are less common - 4 % in Freeman et al series [4] (all, *b*-haemolytic *Streptococcus*), but surprisingly 35.5 % in the recent large series by Bandol et al [29].

In purulent DNIs, the most commonly isolated pathogens include *Streptococcus* / *Peptostreptococcus* species (in non-diabetic patients), and *Staphylococcus*, *Bacteroides*, and *Klebsiella* species (in diabetics). A growing proportion of DNI and DNM cases yield negative bacterial cultures, largely due to empiric antibiotic therapy before hospital presentation. Samples for bacteriological cultures and sensitivities may include blood, pus accumulations, pleural fluid or bronchial lavage, and can be collected pre-, intra-, and post-operatively.

Determinants of Morbidity and Mortality

Comorbidities and predisposing factors have been proven to promote the downward spread of infection from the neck by impairing immune response, worsening prognosis despite treatment.

Diagnostic delays play a very important role in the high mortality rates seen in some studies on DNM. Two recent large series described prolonged infection below the carina in 80 % of cases, with 86.6 % of patients requiring mechanical ventilation, and noted excessive mean elapsed time of around two days between diagnosis and treatment.

In the early 2000, Freeman and colleagues proposed an algorithm incorporating CT scan findings for diagnosis and surveillance, combined with the results of serial operative debridement's and drainage to reduce DNM mortality. Recently, Gehrke et al, reporting on DNIs, and Kim et al in a meta-analysis on head and neck NF, identified predictive inflammatory markers linked to CT-scan findings and / or other clinical measures (Gehrke), and proposed algorithms aiming at earlier detection and improved outcomes [30, 31]. The use of such algorithms for DNM converge on the conclusion that, the time factor is the major concern for diagnosis, and prompt combined medical and surgical management is more decisive for survival than the presence of comorbidities.

Several predisposing factors have been implicated in the worse or lethal course of DNM, such as the high pathogen virulence, individual immune response, poor oral or dental hygiene, abscess extending below the hyoid bone, multiple-space involvement, and dyspnea or impaired oxygenation on admission. Septic shock at presentation is widely reported as an independent predictor of mortality or associated with a higher morbidity. In a large series, patients requiring tracheostomy for airway obstruction had a 6.51-fold higher risk of long-term hospitalization compared to those without airway obstruction.

Elevated C-reactive protein (CRP) levels are also considered a risk factor, as non-survivors have higher mean CRP levels than those who survived. Additional predictors include neutrophil

and lymphocyte percentages and ratios, low serum albumin, elevated blood glucose, and haemoglobin, sodium chloride and creatinine levels. SOFA (Sequential Organ Failure Assessment) and SAPS-3 (Simplified Acute Physiology Score-3) are commonly used to evaluate mortality risk in such critically-ill patients.

Diabetes mellitus (DM) is the most frequently cited comorbidity in DNI / DNM studies (~30 % of cases). Chronic or uncontrolled hyperglucemia impairs neutrophil function and complement activity / cytokine production, enhancing pathogen virulence [8, 21]. The resultant immune dysfunction limits the ability to confine the infection, which is evident in the increase incidence of multi-space involvement in diabetic patients with DNI.

Several other comorbidities, especially systemic conditions with reduced tissue oxygenation, have been described, such as chronic obstructive pulmonary disease, cardiovascular disease, chronic renal disease, liver cirrhosis, obesity, malnutrition, malignancy, and advanced age (> 55 years) [32]. Nonetheless, DNM can also occur in young healthy individuals with no medical history.

Active Mortality in DNM

Mortality associated with DNIs is generally reported to be low. Marioni et al documented zero deaths in a series of 233 consecutive DNI cases. In a recent study involving 46 adults with purulent DNI, including 18.8 % with multiple abscesses, two patients died from complications.

The mortality rate of DNM reached 60 % -70 % during the mid-late 20th century, and, even with therapeutic advances, remained 40 % -50 % through the 1990s [33]. Deaths can occur either early due to the airway obstruction or airway management mishaps, or later as a result of prolonged disease and sepsis.

Mortality among DNM patients admitted with sepsis remains exceptionally high. When DNM evolves, the risk of developing septic shock increases more than threefold. In a comprehensive review, Sarna and colleagues reported mortality rates of up to 20 % for CNF confined to the neck, 41 % for CNF complicated by DNM of odontogenic origin (nearly double the mortality associated with mediastinitis in cardiothoracic surgery), and 64 % for cases involving CNF, DNM and sepsis. Some patient deaths following surgical treatment for DNM may result from the challenges of achieving adequate debridement and maintaining effective drainage, as NF continues its spread across the fascial compartments of the neck and chest. Postoperative morbidity in DNM is also considerable, including ARDS, cardiac tamponade, acute renal failure, stroke, pneumonia and chylo-thorax.

Suppurative cervicomedastinitis frequently affects young people, hence the substantial medico-legal consequences and implications of these cases. Frequently, questions arise regarding possibly missed or delayed diagnosis, or delayed, inappropriate, or insufficient treatment contributing to dissemination of the infection.

However, the vast improvements in an aesthesia and critical care

, modern intravenous (I.V.) antibiotics, the advent of CT imaging, and the more active surgery and multidisciplinary management are reflected in improved outcomes and lower mortality in recent selected studies. Currently, mortality rates for DNM range from 11 % to 40 % [1, 4, 6-11, 13, 15, 19, 26], and a latest review reports a mortality rate of 17.5 % [34].

Diagnosis

Evaluation of DNIs is challenging because the infection is located deep within a complex anatomical region closely associated with vital structures. Many studies focused on NF do not include DNI / DNM cases, despite their pathophysiological similarities to necrotising infections in other regions of the body. Early recognition of DNI / DNM remains difficult, partly due to the rarity of the disease and the non-specific early symptoms. In many cases, the severity and extent of infection do not correlate clearly with the patient's symptoms. Clinicians should always be aware, as disease progression can be very rapid. Urgent recognition of respiratory distress or impending airway compromise is essential.

Clinical symptoms may indicate early and progressive deep-seated severe condition. However, the clinical manifestations of CNF do not accurately correspond to the timeline of the underlying pathology, as severe symptoms typically appear once the infection has already been advanced.

The diagnostic criteria for DNM were first established by Estrera and colleagues in 1983, and incorporate clinical, radiographical, and intraoperative findings: (i) clinical evidence of severe infection (in the oropharyngeal region); (ii) demonstration of specific radiographic findings (on CT imaging); (iii) intraoperative and / or autopsic confirmation of mediastinal infection; and (iv) establishment of strict etiopathogenetic relationship between oropharyngeal or cervical infection and the development of DNM [35].

A thorough history, physical examination, laboratory tests, and diagnostic imaging may provide important information when evaluating suspected DNI / DNM.

Imaging and Laboratory Findings

Chest x-rays in certain DNI cases may be helpful in detecting complications such as mediastinitis (e.g., mediastinal widening, tracheal displacement, air-fluid level), pneumonia or pleural effusion [11, 14]. In mild or localized dental infections, ultrasound (US) has been successfully used for image-guided percutaneous drainage [36].

Contrast-enhanced CT scan of the neck and chest is the preferred imaging modality, allowing accurate assessment of the location and extent of the infection, aiding scheduling therapeutic strategy, evaluating treatment response and result, and monitoring disease progression. Cervicothoracic CT scan surveillance is recommended 48 hours after surgical intervention, or routinely every 48-72 hours until clinical improvement, or sooner if postoperative deterioration occurs.

An abdominal CT scan should be performed if the patient's clinical condition fails to improve or worsens in the presence of a normal postoperative cervico-thoracic CT scan or if the

patient has symptoms or signs positive for intraabdominal infection. Of course, many of these patients are intubated at that time making clinical evaluation difficult.

Magnetic resonance imaging (MRI) may serve as an initial or sole imaging modality for acute neck infection. Compared to CT scan, MRI offers superior soft-tissue contrast, but it is more susceptible to motion artifacts, more expensive and more time-consuming. In the event of vascular complication, MRI angiography is recommended. Pharyngoscopy / laryngoscopy or nasopharyngolaryngeal videofibrosopic examination may help visualize the upper aerodigestive tract when needed.

Microbiological culture results often reveal aerobic / anaerobic coinfections corresponding to pharyngeal or odontogenic origins. White blood cell count and CRP level are commonly used inflammatory markers, but their diagnostic value is not always unequivocal.

Early bedside tissue testing, routinely used in diagnosing other necrotising infectious conditions (e.g., necrotising soft-tissue infections), is typically not performed in DNI or DNM cases because of priorities and inherent procedural limitations.

Recent studies have examined the predictive utility of the LRINEC (Laboratory Risk Indicator for Necrotising Fasciitis) score in CNF [37]. Its accuracy in this setting appears limited and in need for further refinement. With recent advancements, Deep Learning (DL) methods within Artificial Intelligence (AI) has been applied in some studies, such as predicting the need for tracheostomy in DNI patients [38].

Clinical Symptomatology

In the early stages, the infection is often clinically silent and may be veiled by analgesic use delaying the diagnosis. Some patients wait 3 to 4 days before seeking medical help, experiencing delayed diagnosis and treatment. Initial symptoms may include neck pain (following toothache), fever, and swallowing and speaking difficulties (sore throat, dysphagia, dysphonia). If left untreated or not treated properly, the extremely rapid progress of the infection to cervicomediatinitis may additionally include manifestations such as malaise, dyspnea, retrosternal pain, high fever with chills, cervical swelling or erythema, lymphadenopathy, neck stiffness, stridor, trismus and otalgia. At this stage, the extent of infection is disproportionate to the signs and clinical symptoms. According to the literature, the clinical manifestations of DNM occur between 12 hours and 2 weeks after initial presentation (onset of oropharyngeal infection), although they often arise within the first 24-48 hours [39]. A severe presentation is marked by the development of sepsis accompanied by MOD and MOF.

Respiratory function is compromised at the time of localized confined DNI. In fewer than 20 % of cases, airway obstruction - the most feared and deadly complication - occurs very early due to the excessive swellings and purulent accumulations in the neck. In Ludwig's angina cases, the life-threatening edema underneath the tongue and in the neck makes breathing and swallowing difficult, requiring prompt intervention; up to 75 % of patients may require surgical opening of the airway. Established suppurative mediastinitis is commonly associated

with the hallmarks of deteriorating respiratory distress, haemodynamic instability, chest pain, dysphagia and fever.

DNM should be strongly suspected in patients whose general condition is rapidly worsening by severe dyspnea and septic shock. Septic shock at the time of DNM diagnosis is present frequently, with percentages ranging from 13 % in De Palma's series, to 55.5 % in the series by Ramos-Hinojosa.

Major complications associated with the most severe course include ARDS, acute renal failure, MOF, disseminated intravascular coagulation, internal jugular vein thrombophlebitis (Lemierre's syndrome, septicaemia), carotid artery erosion, and more rarely, vertebra osteomyelitis, brain abscess, cranial nerve paresis, and meningitis. Orbital involvement significantly increases morbidity and mortality.

Physical findings may include neck masses (e.g., peritonsillar abscess) in DNI, erythema or pallor, excessive edema on lower neck and upper chest, subcutaneous emphysema (crepitus) or an audible "crunch" on chest auscultation, and skin necrosis in advanced DNM.

Post-mortem autopsy diagnosis of infected neck tissues and DNM carries important medico-legal implications.

Treatment

Because DNMs are rare, complex, and highly aggressive, and because DNIs can rapidly progress into DNM, the therapeutic standard and the optimal management protocol are still controversial. Formal guidelines are lacking, though various recommendations for treatment and monitoring algorithms have been proposed. There is general consensus that treatment must be early, aggressive and multidisciplinary, provided by a group of specialists such as thoracic surgeons, neck / head surgeons or otorhinolaryngologists, oromaxillofacial surgeons, anaesthesiologists, intensivists, specialists in infectious diseases, as well as pathologists, general surgeons, immunologists, and radiologists. Treatment is tailored to the individual patient.

Medical management and surgical intervention should be provided promptly and often simultaneously. DNI / DNM patients often require temporary care in the Intensive Care Unit.

Medical Treatment

Airway assessment is the highest priority in order to ensure airway patency and adequate respiratory function. Emergency airway protection is mandatory in the presence of signs and findings of respiratory distress, including: (i) Upward displacement of the tongue (as seen in Ludwig's angina); (ii) airway obstruction caused by retropharyngeal / parapharyngeal or anterior visceral abscesses; and (iii) respiratory tract edema or multiple-space involvement.

Both orotracheal / nasotracheal intubation (often with fiberoptic video-assisted laryngoscopy, combined flexible bronchoscopy) and tracheostomy / cricothyroidotomy are used when necessary to secure the airway. Intubation is preferred when feasible, as it offers rapid airway control, enables ventilation, and allows direct visualization of the airway, while avoiding the risks of a surgical procedure (e.g., bleeding); disadvantages are

the accidental rupture of pharyngeal abscess, airway injury, and accidental extubation, all leading to catastrophic consequences if re-intubation is not immediately successful. Tracheostomy (or cricothyroidotomy converted to tracheostomy within 24-48 hours) is recommended when formal intubation is not possible or when extrinsic compression or profound local edema obstructs the airway tract; it is avoided when infection involves the anterior cervical or pretracheal space. Tracheostomy provides a more secure airway, and allows easier suctioning of secretions. However, a difficult tracheostomy carries risks such as bleeding, esophageal injury, aspiration of pus, airway loss, arterial dissection, or death, and may lead to postoperative tracheal stenosis (~30 % of survivors).

Immediate symptomatic treatment includes I.V. fluid resuscitation to correct hypovolaemia secondary to sepsis and third-space losses, and combating septic shock (vasopressors, cardiac support, oxygen therapy). Concurrent treatment of comorbidities, such as DM, is essential for optimizing immune function and overall outcomes.

Empiric antibiotic treatment must be initiated within the first hours after the appearance of infection signs in the cervical / oropharyngeal region, before culture results are available. Treatment is typically broad-spectrum, targeting likely pathogens, and should cover gram-positive and gram-negative aerobic and anaerobic microorganisms. Recent studies recommend combinations of penicillin / beta-lactamase inhibitor plus clindamycin or metronidazole, carbapenems, 3rd or 4th generation cephalosporins, aminoglycosides, vancomycin for methicillin-resistant *Staphylococcus aureus*, neutropenia or immune dysfunction. Antifungal agents (e.g., fluconazole, caspofungin) are added when fungal infection is suspected.

Once culture and sensitivity results are available (usually within 48-72 hours), antibiotic therapy should be adjusted accordingly. The duration of targeted therapy depends on microbiological results from serially collected secretions.

Recent evidence suggests that, selected cases of suspected uncomplicated DNI (small abscess or cellulitis) may be effectively treated with antibiotics alone, with or without needle aspiration, under close monitoring. This approach is more often used in paediatric populations [40].

Nutritional support, physiotherapy, and logopedic support (speech therapy to reduce aspiration risk), become important components of recovery.

Surgical Treatment

A thorough understanding of the oropharyngeal and cervical anatomy, as well as the suspected origin, location, and extent of infection, is essential. The degree of vital organ dysfunction (MOD, MOF) is a guide for the specific surgical plan. Surgery is performed with the patient intubated under mechanical ventilation. Generally, all DNM cases require continuous postoperative positive airway pressure.

Surgical indications for DNIs include: airway compromise, critical systemic condition, septicaemia, abscesses greater than 3 cm or involving more than two anatomical spaces or compli-

cated, descending infection, and failure to respond to parenteral antibiotic therapy within the first 24 to 48 hours. In certain situations (difficult infection site, DM), immediate surgical intervention is mandatory at presentation.

Surgery should be directed towards the primary source of infection. In DNIs, the initial goals are to prevent further contamination of the mediastinum and ensure adequate drainage. The operative field may include the oral cavity, neck, chest-mediastinum, and even the abdominal cavity when necessary. The surgical strategy in the infected areas involves incision, extensive debridement of necrotic tissues, and effective drainage of all infected areas. Intraoperative cultures must always be obtained. In most cases, some wounds are laid open or partially closed, and are often irrigated daily. Key approaches of surgical techniques are summarized below.

When the infection is exclusively in the oral cavity or oropharynx, intraoral drainage incisions or an external submandibular approach may be required. Approximately 20 % of cases need tooth extraction.

For cervical infection, additional cervical exploration (external) and drainage become compulsory. Transcervical mediastinal drainage is considered adequate for DNM confined to the upper mediastinum (Endo's Type I, Guan's Type Ia). Patients with CNF associated with DNM should undergo extensive cervicectomy (unilateral or bilateral) combined with mediastinal drainage. The approach to mediastinum depends largely on whether the infection is supracardinal or intracranial. However, due to anatomical constraints and the progressive nature of DNM, there is ongoing debate as to whether a transthoracic approach to achieve optimal debridement and drainage should universally be required. Many investigators advocate mandatory transthoracic mediastinal access regardless of the level of mediastinal involvement. Failure to perform adequate drainage and radical debridement significantly increases mortality.

Infectious spread below the carina should aggressively be managed through a thoracomedial access immediately after the cervicectomy. Posterolateral thoracotomy provides standard access for drainage and radical debridement in DNM. It allows full exposure of the hemithorax, ipsilateral mediastinum, pericardium, and prevertebral / paraesophageal planes. Treatment through thoracotomy is required for prolonged or advanced cases, such as Endo's Types IIA-B or Guan's Types I-III.

Serial operative drainage and debridement, the mainstay of therapy in other necrotizing infections, are seldom described in the literature on the surgical treatment of DNM [41]. However, the accumulation of fluid or air, or the identification of a new abscess in the neck, chest, or abdomen on CT imaging, or clinical deterioration after initial surgery, are indications for repeat debridement and drainage (revision surgery). Serial transcervical or transthoracic re-operations may be necessary to eradicate sepsis, and can even include pulmonary decortications. In a 10-DNM case series, three patients required abdominal exploration.

As alternative to formal thoracotomy, subxiphoid approach with transpleural tube drainage combined with drainage from above

may be used for lower infections (Endo's Type IIa, Guan's Type I). Sternotomy and clamshell thoracotomy have also been rarely described; sternotomy offers limited access to the posterior mediastinum and is associated with risk of osteomyelitis.

There is debate in the literature regarding non-surgical management or minimally invasive intervention for selected, well-localized, unilocular, small collections without airway compromise. In this context, US- or CT-guided percutaneous needle aspiration, sometimes supplemented by mediastinoscopy or video-thoracoscopy, are potential therapeutic alternatives.

Video-assisted thoracoscopic surgical approach (VATS) has been used for drainage and debridement in DNI and DNM cases, including infections extending below the carina (Guan's Type I). Benefits include improved visualization-reduced invasiveness, while disadvantages include a high risk of pleural or pulmonary contamination (due to changes in patient positioning) and significant limitations related to one-lung ventilation, which is often contraindicated due to severe desaturation.

The use of adjunct therapies - such as hyperbaric oxygen or I.V. immunoglobulin (IVIG) - commonly reported for NF / necrotizing soft-tissue infections of trunk, extremities, and external genitalia, remains controversial in the treatment of DNI / DNM because they may disturb other procedures in the acute phase (hyperbaric oxygen) or have been reported only rarely (IVIG). Vacuum-assisted closure systems may support wound healing and have shown benefit in selected cases.

Conclusions

DNM is severe fulminant infection and remains life-threatening. A better understanding of relevant anatomy and of the natural history of the infectious process should play a prominent role in promoting improvements in therapy and outcomes. The excessive mortality associated with DNM should be reduced with adopting treatment strategies incorporating early / timely diagnosis and surveillance with CT imaging and serial transcervical and transthoracic operative drainage and debridement applied by a multidisciplinary team of surgeons, intensivists, and specialists in infectious diseases.

Conflict of Interest

The Authors declare no conflict of interest.

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