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Descending necrotizing mediastinitis in patients with deep neck phlegmon due to oropharyngeal infection

Zstępujące martwicze zapalenie śródpiersia u pacjentów z głęboką ropowicą szyi przy infekcji jamy ustnej i gardła

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ABSTRACT:

Introduction: Among the diseases leading to deep neck infection (DNI) and descending necrotizing mediastinitis (DNM), odontogenic causes are the most prevalent, accounting for 40–70% of cases. Tonsillo-pharyngeal causes follow at 16–30%, while other etiologies do not exceed 6%.

Materials and methods: A retrospective analysis was conducted, examining the clinical course, diagnostic measures, and treatment strategies of 139 patients with DNI due to oropharyngeal inflammatory diseases. These patients were treated at the Poltava Regional Clinical Hospital from 2012 to 2023. Based on the primary source of infectious/purulent inflammation, patients were divided into two groups. Group I included 25 patients with tonsillo-pharyngeal origin of DNI; Group II consisted of 114 patients with dental origin of DNI. The study assessed the course, diagnosis, and treatment outcomes based on the parameters of gender, age, etiological factor, underlying pathology, disease duration before hospitalization, initiation of antibiotic therapy, type of DNM based on spread pattern, causative agent, severity of condition upon admission, signs of sepsis, type of surgical intervention, postoperative complications, duration of treatment in the intensive care unit, overall hospitalization duration, and mortality.

Results: Oropharyngeal infection is the predominant cause of DNI and DNM. Notably, odontogenic DNI is more frequently observed than tonsillopharyngeal DNI (82.0% *vs*. 18.0%). Its progression is characterized by lower mortality (13.2% *vs*. 24.0%) and a reduced risk of DNM onset (7.0% *vs*. 44.0%). However, odontogenic DNM is more often associated with adverse outcomes (50.0% *vs*. 9.1%).

Conclusions: Thus, frequency and severity of DNM in patients with DNI depends on the etiology of the disease, but the adverse consequences of treatment are largely influenced by weak concomitant pathology and sepsis during hospitalization.

KEYWORDS: deep neck infection, descending necrotizing mediastinitis, oropharyngeal infection

STRESZCZENIE:

Wprowadzenie: W strukturze schorzeń, które powodują głębokie zakażenie szyi (ang. *deep neck infection*; DNI) i zstępują martwicze zapalenie śródpiersia (ang. *descending necrotizing mediastinitis*; DNM), dominującymi są zębopochodne (40–70%) i migdałkowo-gardłowe (16–30%), pozostałe przyczyny nie przekraczają 6%.

Materiały i metody: Retrospektywną analizę przebiegu schorzenia przeprowadzono u 139 pacjentów z DNI, które spowodowane było chorobami zapalnymi jamy ustnej i gardła, leczonymi w Połtawskim Obwodowym Szpitalu Klinicznym im. N.W. Sklifosowskiego w latach 2012–2023. Pacjentów podzielono na dwie grupy: I grupa – 25 pacjentów z migdałkowo-gardłową genezą DNI i II grupa – 114 pacjentów z zębopochodną genezą DNI. Przebieg i wyniki leczenia pacjentów oceniano według następujących parametrów: płeć, wiek, czynnik etiologiczny, patologia współistniejąca, czas trwania choroby przed hospitalizacją, rozpoczęcie leczenia przeciwbakteryjnego, częstość występowania DNM, rodzaj patogenu, ciężkość stanu podczas hospitalizacji, objawy sepsy, rodzaj interwencji chirurgicznej, powikłania pooperacyjne, czas leczenia na oddziale intensywnej terapii, terminy hospitalizacji lub śmiertelności.

Wyniki: Infekcja jamy ustnej i gardła jest dominującą przyczyną rozwoju DNI i DNM. Jednocześnie zębopochodne DNI obserwuje się częściej niż migdałkowo-gardłową (82,0% *vs*. 18,0%), jej przebieg charakteryzuje się mniejszą śmiertelnością (13,2% *vs*. 24,0%) i mniejszym ryzykiem wystąpienia zstępującego martwiczego zapalenia śródpiersia (7,0% *vs*. 44,0%). Jednak to zębopochodnemu DNM, częściej towarzyszy niekorzystny przebieg (50,0% *vs*. 9,1%).

Wnioski: Częstotliwość i nasilenie DNM u chorych na DNI zależy od etiologii choroby, jednak na niekorzystne skutki leczenia duży wpływ mają niewyrównane współistniejące patologie i sepsa w czasie hospitalizacji.

Słowa kluczowe: głębokie zakażenie szyi, infekcja jamy ustnej i gardła, zstępujące martwicze zapalenie śródpiersia

ABBREVIATIONS

CT – computer tomography
DNI – deep neck infection
DNM – descending necrotizing mediastinitis
ENT – otolaryngologist
ICU – intensive care unit
SIRS – systemic inflammatory response syndrome

RELEVANCE

DNI is a typical complication of bacterial diseases in the oropharynx, primarily with an odontogenic or tonsillo-pharyngeal origin. Dental origins predominate (40–70%) within the spectrum of diseases causing DNI, followed by tonsillo-pharyngeal sources (16–30%). Other causes, including traumatic and iatrogenic injuries to the throat and esophagus, thyroiditis, and diverticula, contribute to just 6% of cases. The genesis of DNI often remains undetermined [1–8]. In the majority of cases, sanitizing the infection source and the affected neck spaces in conjunction with antibiotic therapy leads to the patients' recovery; fatalities are very uncommon. However, the clinical significance of DNI is raised by the potential spread of the infectious process to the mediastinum, leading to descending necrotizing mediastinitis (DNM). Despite modern approaches to treating DNM using prompt CT imaging, aggressive surgical intervention, and targeted antibiotic therapy, the mortality rate remains high at 24–28%. This rate rises to 60–80% in patients presenting with sepsis and septic shock [7–10]. The occurrence of DNM in oropharyngeal diseases complicated by DNI is extremely rare. In clinical practice, doctors typically encounter only isolated cases, and even regional centers specializing in maxillofacial, ENT, and thoracic surgery have limited experience in treating DNM [11–13].

The infection spreads to the neck within the layers of the deep cervical fascia and its further progression to the mediastinum is due to both gravitational forces and the suction effect during thoracic cavity respiratory movements [2, 4, 6, 12]. The microflora causing DNI and DNM is quite diverse. Coccal microflorae are most commonly detected, but aerobes and anaerobes demonstrate extremely high virulence, often precipitating early-onset sepsis and shock [1, 8, 9, 13, 14]. The timely diagnosis of DNM is challenging, as the use of non-steroidal anti-inflammatory drugs and antibiotics in the early stages of oropharyngeal diseases masks the typical clinical signs and the general condition of patients often does not raise concerns among specialists. Only early CT imaging of the neck and chest can reveal signs of purulent/necrotic inflammation such as swelling, accumulation of fluid and gas in the neck spaces and mediastinum, and most importantly, the spread of exudate pathways, which is crucial for determining surgical approaches [6, 12, 15]. Aggressive and timely surgery focusing on controlling the infection source by sanitizing and draining the primary focus, DNI, and DNM is paramount for reducing mortality [2, 6–13, 15].

Recent studies indicate an improving trend in the treatment outcomes for this category of patients. However, further research is needed into the disparities in treatment outcomes between dental and tonsillo-pharyngeal origins of DNM [3, 4, 6, 7, 10, 15].

RESEARCH OBJECTIVE

The aim was to study the characteristics of the course and treatment of patients with DNI and descending mediastinitis resulting from oropharyngeal diseases.

MATERIALS AND METHODS

A retrospective analysis was conducted, examining the clinical course, diagnostics, and treatment strategies of 139 patients with DNI due to oropharyngeal inflammatory diseases. The patients were treated from 2012 to 2023 at the M.V. Sklifosovskyi Poltava Regional Clinical Hospital, which serves as a regional center for specialized care for this category of patients. The cohort was comprised of 108 men (77.7%) and 31 women (22.3%), with a mean age of 46 years (ranging from 22 to 76 years).

The inclusion criteria for the study were signs of DNI, DNI, and mediastinitis complicating the clinical course of inflammatory conditions of the oral cavity and pharynx. Patients with iatrogenic injuries to the throat and esophagus, and other diseases of the neck and thoracic cavity organs and structures, were excluded from the study. The diagnosis of DNM was established based on the typical signs delineated by Estrera et al. [9]. In accordance with the standard protocol, all patients who presented with an inflammatory process of the oropharynx and neck underwent a comprehensive set of clinical laboratory tests and CT imaging of the neck and chest upon hospitalization.

The extent of the infection was initially determined based on the results of the CT imaging of the neck and thorax by visualizing infiltration of tissues, accumulation of fluid and gas, and displacement of the mediastinal structures, pleural cavities, and pericardium. The definitive assessment was based on findings during surgical exploration via cervicotomy and mediastinotomy. The spread of DNM was stratified according to the types proposed by Endo et al. (1999) [2], as modified by Sugio et al. (2021) [15]. Thus, Type I referred to DNM affecting only the upper mediastinum (above the tracheal bifurcation); Type IIA indicated the spread of the process to the retrosternal space; Type IIB denoted comprehensive involvement of the upper and lower mediastinum; and Type IIC described the spread of the process to the posterior mediastinum along the retropharyngeal and prevertebral spaces.

Based on the data regarding the primary source of infectious/purulent inflammation, the patients were divided into two groups. Group I included 25 patients (20 men [80.0%]; average age: 46 years)

with tonsillo-pharyngeal origin of DNI. Group II consisted of 114 patients (90 men [79.0%]; average age: 47 years) with dental origin of DNI.

The study assessed the course, diagnosis, and treatment outcomes based on the parameters of gender, age, etiological factor, underlying pathology, disease duration before hospitalization, initiation of antibiotic therapy, type of DNM based on spread pattern, causative agent, the severity of condition upon admission (severe: 8–15; extremely severe: 16+ according to APACHE-II), signs of sepsis (SIRS + organ dysfunction), surgical intervention type, postoperative complications, duration of treatment in the intensive care unit (ICU), overall hospitalization duration, and mortality.

Statistical analysis was conducted to determine significant differences in the selected parameters between the study groups and to identify risk factors for the onset of DNM. The t-test, chi-square test, and a p-value of < 0.05 were used to determine statistical significance.

RESEARCH RESULTS

Characteristics of group I patients

DNI was the result of spreading peritonsillar (n = 9 [36.0%]) and parapharyngeal infection (n = 16 [64.0%]). DNM developed in 11 (44.0%) of these patients, with only 2 (18.2%) of them having DNM as a result of paratonsillar infection. The disease duration from onset to DNM confirmation and hospitalization ranged from 2 to 15 days (6 days on average), with most of these patients (n = 10 [90.9%]) receiving initial outpatient treatment and being prescribed antibiotics. Most patients (n = 7 [63.6%]) presented with both DNI and DNM upon hospitalization. The remaining 36.4% (n = 4) were diagnosed with DNM after initial transoral surgical intervention of the primary focus and opening of the phlegmon during dynamic monitoring on day 2–8 (on average, day 4) post-surgery, based on repeated CT scans in the absence of clinical improvement.

The presence of DNM exacerbated the patients' condition compared to its absence ($p < 0.05$): extremely severe in 5 (45.5%) of them *vs.* in 1 (7.1%) without mediastinitis; severe in 5 (45.5%) *vs.* 8 (57.1%); and moderate severity in 1 (9.1%) *vs.* 5 (35.7%). Systemic inflammatory response syndrome (SIRS) was observed in 18 (72.0%) patients with DNI: in 9 (81.8%) in the presence of DNM *vs.* in 9 (64.3%) in its absence. Sepsis was observed in 9 (36.0%) patients – in 4 (36.4%) *vs.* 5 (35.7%) – while septic shock was present in 3 (12.0%) patients – 1 (9.1%) *vs.* 2 (14.3%) – but the differences were not statistically significant ($p > 0.05$). Clinically significant comorbidities were observed in 7 (28.0%) patients and diabetes mellitus in 6 (24.0%) (Tab. I.).

According to the initial CT findings and subsequent surgical revision, all patients exhibited DNI (Tab. II.). Thus, for parapharyngeal and paratonsillar origins, the primary routes of infection spreading to the mediastinum were predominantly central perivascular, posterior retropharyngeal, and prevertebral.

All patients underwent cervicotomy along the inner edge of the sternocleidomastoid muscle (left-sided in 8 [32.0%], right-sided in 9 [36.0%], and bilateral in 8 [32.0%]) for neck phlegmon incision, sanitation, and drainage. The nature of the intervention in the mediastinum depended on the spread of DNM. For Type I (3 patients), transcervical drainage of mediastinitis was performed; for Type IIB (4 patients), a combined transcervical and right-sided transthoracic approach was used, with 2 of them undergoing additional drainage of the opposite pleural cavity. Among the 4 Type IIC patients, 2 had a combined approach with bilateral thoracotomy and 1 had transcervical drainage of the pleural cavity. Microbiological examination in patients with DNI but without DNM was initially conducted in 71.4% of the subjects, with the results indicating a predominant mono-infection (n = 11 [78.6%]): *S. aureus*, *S. epidermidis*, or *Enterobacter*. In contrast, 5 (45.5%) of those with DNM presented with mono-infection (*S. epidermidis* [n = 3], *Enterobacter*, and *E. coli*). In 3 (27.3%) patients, no microflora was detected; in 3 (27.3%), a mixed microflora was found (*E. faecalis*, *K. pneumoniae*, *Acinetobacter*, *P. aeruginosa*, and *Candida*).

Tab. I. Severity of condition and sepsis in patients hospitalized with tonsillopharyngeal DNI.

SEVERITY OF CONDITION AND SEPSIS	DNI	DNI WITHOUT DNM	DNI WITH DNM
Extremely severe	24.0% (n = 6)	7.1% (n = 1)	45.5% (n = 5)*
Severe	52.0% (n = 13)	57.1% (n = 8)	45.5% (n = 5)*
Moderate	24.0% (n = 6)	35.7% (n = 5)	9.1% (n = 1)*
SIRS	72.0% (n = 18)	64.3% (n = 9)	81.8% (n = 9)*
Sepsis	36.0% (n = 9)	35.7% (n = 5)	36.4% (n = 4)**
Septic shock	12.0% (n = 3)	14.3% (n = 2)	9.1% (n = 1)**

*difference is statistically significant ($p < 0.05$); **difference is not statistically significant ($p > 0.05$)

Postoperative complications developed in 3 (12.0%) patients, including laryngeal stenosis, recurrent laryngeal nerve paresis, erosive bleeding, and pulmonary embolism. The average treatment for recovered patients lasted 18 days (ranging from 9 to 32 days) in the absence of DNM and 29 days (ranging from 12 to 45 days) in its presence; the average treatment in the ICU was 7 days (ranging from 1 to 14 days) in the absence of DNM and 11 days (ranging from 1 to 33 days) in its presence. Six patients with DNI died, resulting in a mortality rate of 24.0%; five (35.7%) had no DNM and 1 (9.1%) had DNM. All of the deceased patients had clinically significant comorbidities, signs of sepsis, and septic shock upon hospitalization; the majority of the deceased were men (66.7%), at an average age of 58 years; 50.0% of them had diabetes.

Characteristics of group II patients

Odontogenic inflammatory diseases that led to the onset of DNI predominantly included purulent/destructive odontogenic diseases (n = 84 [73.7%]), cellulitis of the mouth floor (n = 11 [9.7%]), and lymph gland inflammation (n = 19 [16.7%]). DNM developed in 8 (7.0%) of these patients, with only 1 (18.2%) having cellulitis of the mouth floor; no lymph gland inflammation was observed. The disease duration from onset to DNM confirmation and hospitalization ranged from 3 to 5 days (with an average of 4 days). Only half of the patients with DNM (n = 4 [50.0%]) received initial outpatient treatment with prescribed antibiotics. In most patients (n = 6 [75.5%]), both DNI and DNM were detected upon hospitalization, while in the rest of the cases (n = 2 [25.5%]), DNM

Tab. II. Verification and spread of DNM in tonsillopharyngeal DNI.

Hospital verification (DNI+DNM)	63.6% (n = 7)
DNM verification in DNI progression (4th night)	36.4% (n = 4)
DNM, I type	27.3% (n = 3)
DNM, II type, among them:	72.7% (n = 8)
DNM, type IIA	not detected
DNM, type IIB	36.4% (n = 4)
DNM, type IIC	36.4% (n = 4)

Tab. III. Severity of condition and sepsis in patients hospitalized with odontogenic DNI.

SEVERITY OF CONDITION AND SEPSIS	DNI	DNI WITHOUT DNM	DNI WITH DNM
Extremely severe	16.7% (n = 19)	14.2% (n = 15)	50.0% (n = 4)*
Severe	50.9% (n = 58)	50.9% (n = 54)	50.0% (n = 4)**
Moderate	26.3% (n = 30)	28.3% (n = 30)	–
Satisfactory	6.1% (n = 7)	6.6% (n = 7)	–
SIRS	68.4% (n = 78)	66.0% (n = 70)	100% (n = 8)*
Sepsis	23.7% (n = 27)	24.5% (n = 26)	62.5% (n = 5)*

*difference is statistically significant (p < 0.05); **difference is not statistically significant (p > 0.05)

Tab. IV. Severity of condition and sepsis in patients hospitalized with odontogenic DNI.

Hospital verification (DNI+DNM)	75.5% (n = 6)
DNM verification in DNI progression (2nd night)	24.5% (n = 2)
DNM, I type	12.5% (n = 1)
DNM, II type, among them:	87.5% (n = 7)
DNM, type IIA	37.5% (n = 3)
DNM, type IIB	50.0% (n = 5)
DNM, type IIC	not detected

was detected after prior transoral surgical treatment of the primary focus and opening the phlegmon during dynamic monitoring on the 2nd day, based on repeated CT imaging in the absence of clinical improvements.

The onset of DNM in these patients significantly exacerbated their condition compared to those without DNM: 50.0% were in an extremely critical state *vs.* 16.7% without mediastinitis ($p < 0.05$). SIRS was observed in 78 (68.4%) patients with DNI: 8 (100%) in the presence of DNM *vs.* 70 (66.0%) in its absence ($p < 0.05$); sepsis was present in 27 (23.7%) patients: 5 (62.5%) with DNM *vs.* 26 (24.5%) without it ($p < 0.05$); and septic shock was observed in 7 (6.1%) patients: 3 (37.5%) with DNM *vs.* 4 (3.8%) without it ($p < 0.05$). Clinically significant comorbidities were observed in 27 (23.7%) of these patients; diabetes was present in 22 (19.3%). No statistically significant differences were found concerning the presence of DNM and comorbidities ($p > 0.05$) (Tab. III.).

CT imaging revealed signs of DNI in all patients, with 8 (7.0%) showing the spread of the process to the mediastinum (Tab. IV.). Thus, with an odontogenic origin, the primary routes for infection spreading to the mediastinum were mainly anterior pretracheal and central perivascular.

All patients underwent DNI sanitation and drainage through cervicotomy along the inner edge of the sternocleidomastoid muscle, with 58 (50.8%) having a unilateral procedure, and 56 (49.1%) having a bilateral one. Additionally, 72 (63.2%) subjects underwent procedures in the submandibular region. In 1 patient with Type I DNM, transcervical mediastinitis drainage was performed. Type IIA (n = 3) and Type IIB patients (n = 4) had combined interventions, with 2 additionally undergoing drainage of the opposite pleural cavity. Microbiological examination was initially conducted in 74.5% of the patients with DNI but without DNM. Mono-infection was observed in 16 (15.1%) (*S. epidermidis*, *S. pyogenes*, *E. coli*, *S. aureus*, and *Enterobacter*); no microflora was detected in 21 (19.8%), and mixed infection (*S. pyogenes*, *E. coli*, *S. aureus*, *Enterobacter*, *E. faecalis*, *K. pneumoniae*, *Acinetobacter*, *S. epidermidis*, and *Candida*) was found in 69 (65.1%). In the case of DNM, mixed microflora predominated (n = 6; 75.0%) ($p < 0.05$), and mono-infection was detected in 2 (25.0%) patients ($p < 0.05$). The composition of pathogen species did not show significant differences.

Postoperative complications developed in 9 (7.9%) of these patients: 7 (6.6%) in the absence of DNM and 2 (25.0%) in its presence ($p < 0.05$). The average treatment duration for recovered patients was 16 days (ranging from 8 to 28 days) in the absence of DNM and 26 days (ranging from 15 to 40 days) in its presence ($p < 0.05$). The average treatment duration in the ICU was 6 days (ranging from 1 to 12 days) in the absence of DNM and 7 days (ranging from 3 to 9 days) in its presence ($p > 0.05$). Fourteen patients with DNI died, resulting in a mortality rate of 12.3%: 10 (9.4%) without DNM and 4 (50.0%) with DNM. All of the deceased patients had clinically significant comorbidities, signs of sepsis, and septic shock upon hospitalization. The majority of them were men (64.3%), at an average age of 64 years; 42.9% of them had diabetes. The criteria of gender, age, and comorbidities did not demonstrate statistically significant differences concerning the presence or absence of DNM.

DISCUSSION

In the vast majority of cases, DNM results from the spread of DNI, which has either odontogenic or tonsillopharyngeal origin. A majority of studies confirm that oropharyngeal infection is the predominant cause of both DNI and DNM [1, 3–8, 10, 13–15]. The high virulence of this infection and its rapid spread to the deep fascial spaces of the neck and mediastinum account for the severe progression of the disease and high mortality rate [5–8, 10, 13]. However, the patient profile of DNI with DNM is heterogeneous and depends on the disease's etiology. For instance, 25 cases with tonsillopharyngeal DNI and 114 cases with odontogenic origin were recorded in a regional center over a decade. DNM was observed in 44.0% and 7.0% of these patients, respectively ($p < 0.05$).

The adverse progression of the disease is linked to delayed diagnosis and treatment initiation. Specific clinical signs are only typical of primary oropharyngeal diseases. Both DNI and DNM present with non-specific symptoms, predominantly severe intoxication signs. The early use of non-steroidal anti-inflammatory and antibacterial drugs can often obscure these symptoms [1, 4, 5, 7, 8, 10, 14]. The outpatient phase of the disease also presents some unique characteristics, although many lack significant statistical differentiation. For instance, tonsillopharyngeal DNI and DNM

patients typically have a longer pre-hospitalization disease duration. However, these patients more frequently ($p < 0.05$) received primary disease treatment and were prescribed antibiotics, which is especially evident in patients with DNM: 90.9% *vs.* 50.0% with odontogenic DNM. Regardless of disease origin, the most effective diagnostic method for DNI and DNM is CT imaging. This method can confirm most cases (63.6–75.5%) during the initial examination or dynamic monitoring after 48 hours [6, 12, 15].

The severity of patients' condition upon hospitalization is determined by the intensity of intoxication and has no statistically significant connection with the disease origin. However, there was a discernible difference in the species composition of the causative agents. For instance, coccus mono-infection was predominant in tonsillopharyngeal DNI and DNM ($p < 0.05$), while a mixed infection was more common in odontogenic origin cases ($p < 0.05$). It should be noted that patients with tonsillopharyngeal DNI and DNM had a higher frequency of sepsis and septic shock diagnoses ($p < 0.05$). In this context, post-hospitalization septic shock was an absolute marker of adverse progression for both DNI and DNM, regardless of their origin. The presence of clinically significant comorbidities, irrespective of the origin of DNI and DNM, significantly increases the likelihood of a fatal outcome [8, 9, 13].

Regardless of the origin of DNI and DNM, the surgical approach was primarily informed by CT imaging data; this was followed by a re-evaluation of the infection boundaries based on intraoperative findings. For instance, infection control within the neck was typically achieved via unilateral or bilateral cervicotomy. The use of bilateral access was more frequently required in the case of odontogenic disease origin ($p < 0.05$). Type I DNM (upper mediastinitis) was more commonly observed in cases of tonsillopharyngeal origin ($p < 0.05$) and was absent in Type IIA, whereas Type IIC DNM was not observed in odontogenic cases. Consequently, the primary routes for DNI progression into the mediastinum for tonsillopharyngeal cases are perivascular and posterior retropharyngeal/prevertebral; in the case of odontogenic origin, it is pretracheal and perivascular. In our view, an important practical aspect of surgical treatment for odontogenic DNI is the predominance ($p < 0.05$) of bilateral involvement of the deep neck layers and central (perivascular) progression into the mediastinum, underscoring the need for re-examination of these regions.

In most cases, the postoperative period in patients with DNI and DNM took a severe course, with the correlation between severity and disease origin being inconclusive. Post-surgical complications were mostly associated with the progression of organ dysfunctions and the extent of the purulent/destructive process, showing no statistically significant difference based on the disease's origin. Similarly, the duration of hospital stays and treatment in the ICU for recovered patients did not differ significantly based on the origin of the disease. Mortality in patients with odontogenic DNI origin was 13.2%, with 9.4% in the absence of DNM and 50.0% in its presence. An adverse course in cases with tonsillopharyngeal DNI origin was observed in 24.0% ($p < 0.05$) of patients, with 35.7% ($p < 0.05$) in the absence of DNM and 9.1% ($p < 0.05$) in its presence. Thus, odontogenic infections primarily accounted for oropharyngeal DNI diseases, marked by reduced mortality and a lower probability of DNM onset. However, the course of DNM of odontogenic origin demonstrated the highest likelihood of unfavorable treatment outcomes. These characteristics of the course of complicated oropharyngeal infection align with findings from other studies [3–5, 7, 8, 10, 13].

CONCLUSION

1. Oropharyngeal infection is the predominant cause of DNI and DNM. Notably, odontogenic DNI is more frequently observed than tonsillopharyngeal DNI (82.0% *vs.* 18.0%). Its progression is characterized by lower mortality (13.2% *vs.* 24.0%) and a reduced risk of DNM onset (7.0% *vs.* 44.0%). However, odontogenic DNM is more often associated with adverse outcomes (50.0% *vs.* 9.1%).
2. The spread of odontogenic DNI into the mediastinum occurs through the pretracheal or perivascular (carotid) pathways, while tonsillopharyngeal DNI spreads via the perivascular (carotid) or retropharyngeal (prevertebral) route. Odontogenic DNI often presents as bilaterally affecting the deep neck spaces and progressing perivascularly into the mediastinum, necessitating appropriate surgical re-examination of these areas.
3. Factors contributing to the adverse progression of DNI and DNM, irrespective of their etiology, include clinically significant comorbidities, the severity of the condition, and the presence of sepsis and septic shock upon hospitalization. Odontogenic DNM is characterized by the highest mortality due to delayed treatment of oral cavity diseases, high virulence of the causative agents, and early onset of sepsis and septic shock.

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