Original paper

Comparison of clinical and bacterial profile of odontogenic and non-odontogenic maxillofacial infections

Porównanie profilu klinicznego i bakteriologicznego zębopochodnych i niezębopochodnych zakażeń szczękowo-twarzowych

Bartłomiej Kamiński[1], Konrad Kołomański[2], Maciej Sikora[2,3], Katarzyna J. Błochowiak[4]

[1]Department of Otolaryngology, Maria Skłodowska-Curie District Hospital, Skarżysko-Kamienna, Poland|
[2]Department of Maxillofacial Surgery, Hospital of the Ministry of Interior, Kielce, Poland
[3]Department of Biochemistry and Medical Chemistry, Pomeranian Medical University, Szczecin, Poland
[4]Department of Oral Surgery, Periodontal Diseases and Oral Mucosal Diseases, Poznan University of Medical Sciences, Poznan, Poland

Medical Studies/Studia Medyczne 2024; 40 (2): 143–156
DOI: https://doi.org/10.5114/ms.2024.140977

Key words: peritonsillar abscess, odontogenic infections, abscesses, non-odontogenic infections, deep neck infections.

Słowa kluczowe: ropień okołomigdałkowy, zakażenia zębopochodne, ropnie, zakażenia niezębopochodne, głębokie zakażenia szyi.

Abstract

Introduction: Deep carious lesions and their complications are possible causes of odontogenic infections. Although their location and clinical symptoms may mimic non-odontogenic infections, they are characterised by specific features that are helpful in their diagnosis and treatment. It seems worthwhile to create their clinical and microbiological profile.
Aim of the research: To compare the clinical and microbiological features of odontogenic and non-odontogenic infections.
Material and methods: The study was based on the medical records of 403 patients affected by the diseases.
Results and conclusions: There were statistically significant differences in the white blood cell count, the number of accompanying diseases, dysphagia and the occurrence of neck swelling, and the duration of hospitalisation between odontogenic and non-odontogenic infections. We identified the most common pathogens as well as the clinical parameters specific to these infections. Although bacterial distribution was similar in both groups with a predominance of aerobic cocci, non-odontogenic infections were characterised by a relatively high contribution of Staphylococcus aureus and Klebsiella pneumoniae in comparison to odontogenic infections. We also indicated submandibular and peritonsillar spaces as commonly involved fascial spaces in odontogenic and non-odontogenic infections, respectively. Circulatory diseases and connective tissue diseases were identified as a factor predisposing to odontogenic infections. Comorbidities are the most important risk factor for the development of odontogenic infections and their severe course requiring hospitalisation.

Streszczenie

Wprowadzenie: Głębokie ubytki próchnicowe i ich komplikacje są możliwą przyczyną zakażeń zębopochodnych. Chociaż ich lokalizacja i objawy kliniczne mogą naśladować infekcje niezębopochodne, charakteryzują się one specyficznymi cechami, które są pomocne w ich diagnozowaniu i leczeniu. Wydaje się zasadne stworzenie ich profilu klinicznego i mikrobiologicznego.
Cel pracy: Porównanie cech klinicznych i mikrobiologicznych zakażeń zębopochodnych i niezębopochodnych.
Materiał i metody: Badanie oparto na historii chorób 403 pacjentów z zakażeniami szczękowo-twarzowymi.
Wyniki i wnioski: Nie stwierdzono statystycznie znaczących różnic w liczbie białych krwinek, liczbie chorób towarzyszących, dysfagii i występowaniu obrzęku szyi, czasie hospitalizacji między zakażeniami zębopochodnymi i niezębopochodnymi. Zidentyfikowano najczęstsze patogeny i cechy kliniczne specyficzne dla tych infekcji. Chociaż rozkład bakterii był podobny w obu grupach z przewagą tlenowych ziarniaków, zakażenia niezębopochodne charakteryzowały się relatywnie wysokim udziałem *Staphylococcus aureus* i *Klebsiella pneumoniae* w porównaniu z zakażeniami zębopochodnymi. Wskazano przestrzeń podżuchwową i okołomigdałkową jako najczęściej zajęte odpowiednio w zakażeniach zębopochodnych i niezębopochodnych. Choroby układu krążenia i choroby tkanki łącznej zidentyfikowano jako czynniki predysponujące do zakażeń zębopochodnych. Choroby współwystępujące są najważniejszymi czynnikami ryzyka w rozwoju zakażeń zębopochodnych i ich ciężkiego przebiegu prowadzącego do hospitalizacji.

Introduction

Deep caries and their complications may lead to severe odontogenic infections involving adjacent maxillofacial areas. Their extensive spread or failed treatment can result in life-threatening complications including airway obstruction, phlegmon, sepsis, and septic shock. These severe complications occur more often in medically compromised patients and require hospitalisation and intensified therapy, constituting a serious medical problem and a significant burden on the medical care system [1–6]. Other odontogenic causes of these infections include pericoronitis, severe periodontitis, cyst suppuration, and odontogenic sinusitis. In turn, a similar course of infections results from non-odontogenic causes. Both odontogenic and non-odontogenic infections may involve the same maxillofacial areas and present similar symptoms. Apart from similar symptomatology and localisation, both odontogenic and non-odontogenic infections may be caused by similar bacterial flora saprophytic in the oral cavity, and their course may be closely related to similar local and systemic factors. Finally, they can lead to life-threatening complications similar to odontogenic infections. This similarity in the clinical course may be a source of malpractice. In our opinion, it is reasonable to find the unique features of odontogenic and non-odontogenic infections, which differentiate them and help in the implementation of more effective diagnostic and therapeutic methods. One of the postulated predictive factors for their more aggressive course is multimorbidity of the patients. The second difference between odontogenic and non-odontogenic infections that may determine their pharmacological treatment is the microbiological profile. Therefore, more data are needed to explore the relationship between odontogenic and non-odontogenic maxillofacial infections and between accompanying systemic diseases and bacterial load.

Aim of the research

The aim of this study is to compare comprehensive clinical profiles of both odontogenic and non-odontogenic maxillofacial infections, and to find their unique systemic or local features determining the differences in the way we deal with them.

Material and methods

The study comprised 403 patients diagnosed with maxillofacial infections (female : male ratio 180 : 223, age range: 7–91 years) admitted to the Department of Otolaryngology, Skarzysko-Kamienna Hospital, and in the Department of Maxillofacial Surgery of the Hospital of the Ministry of Interior in Kielce from January 2014 to June 2022. All patients were divided according to the primary cause of infections into 2 groups: odontogenic and non-odontogenic, including 133 patients and 270 patients, respectively. Exclusion criteria included head and neck tumours and superficial skin abscesses. The diagnosis was made on the basis of the patient's history, a clinical examination, and an ultrasonic examination or computed tomography (CT). Moreover, the data related to the occurrence of systemic diseases were taken from all patients. Laboratory tests including the erythrocyte sedimentation rate (ESR), white blood cell count (WBC), and C-reactive protein (CRP) value and microbiological examinations were carried out. All data were introduced into the medical records and then statistically analysed. The reference ranges for standard values in our laboratory were 4×10^3–10×10^3/mm^3 for the WBC count, less than 5 mg/l for the C-reactive protein, and 1–10 mm/h for ESR. All patients were treated surgically by incision and drainage performed under local or general anaesthesia or in combination with antibiotics. Initially implemented empirical antibiotics were modified after the results of microbiological analysis.

The protocol for this study was approved by the Bioethics Committee of Holycross Medical Chamber, Poland (No. 11/2021-VIII). This study was performed in accordance with the ethical standards laid down in the relevant version of the World Medical Association Declaration of Helsinki from 2013. Written informed consent was obtained from each patient or from the patient's legal representative before any study procedure was carried out. In minor patients over 16 years of age, both written informed patient's consent and the consent of their legal representative were obtained. For minor patients under 16 years of age, the consent of their legal representative was obtained.

Statistical analysis

The calculations were carried out with Microsoft Excel 2016 and Statistica software (v.13 TIBCO, Palo Alto, CA, USA). Distributions of continuous variables were evaluated for normality using the Shapiro-Wilk test. The differences between the 2 groups were tested using the Mann-Whitney *U* test. For qualitative variables, the numbers (*n*) and proportions (%) were calculated and collected in cross-tables. Categorical variables are presented in contingency tables, and their associations were tested, depending on the number of cases, with Fisher's exact test or χ^2 Pearson's test. $P \leq 0.05$ was considered statistically significant. The confidence level for our study similarly to other studies from medical sciences was determined at 95%.

Results

Table 1 presents a summarised demographic, clinical, and laboratory profile of patients with maxillofacial infections. A detailed distribution of the patients' ages showed that 6.94% of patients were under

Table 1. Demographic, clinical, and laboratory characteristics of patients with maxillofacial infections

Parameter	Values
Number of individuals, *n*	403
Gender, female/male, *n*	180/223
Age, mean ± SD [years]	41.14 ±17.89
Age, median (range) [years]	37.0 (7.0–91.0)
BMI [kg/m^2] median	24.5
Symptoms, *n* (%):	
Pain	373 (92.5)
Trismus	183 (45.5)
Dysphagia	199 (49.5)
Otalgia	21 (5.22)
Fever	55 (13.68)
Dyspnoea	8 (1.99)
Neck swelling	171 (42.5)
Sialorrhoea	5 (1.24)
Hoarseness	5 (1.24)
Other symptoms	107 (26.6)
Number of symptoms, *n*:	
≤ 2 symptoms	164
3 symptoms	124
≥ 4 symptoms	108
Space involvement, *n* (%):	
Submandibular	116 (28.8)
Parapharyngeal	12 (2.98)
Peritonsillar	177 (44.0)
Buccal	72 (17.9)
Parotid	8 (1.99)
Temporal	5 (1.24)
Infratemporal	4 (0.99)
Submental	9 (2.23)
Orbital	5 (1.24)
Neck	30 (7.46)
Other space involvement	12 (2.98)
Causes, *n* (%):	
Odontogenic	133 (33.0)
Tonsilitis	179 (44.4)
Sialadenitis	11 (2.7)
Sinusitis	3 (0.74)
Posttraumatic	15 (3.72)
Postoperative	11 (2.72)
Iatrogenic	5 (1.24)
Systemic	5 (1.24)
Undetermined	32 (7.94)
Laboratory values, mean:	
WBC [K/µl]	11.43
ESR [mm/h]	56.13
CRP [mg/l]	74.02
Duration of hospitalisation [days] median	5.0
Biopsy, *n* (%)	316 (78.4)
Incision, *n* (%)	335 (83.1)
Local anaesthesia, *n* (%)	325 (80.6)
General anaesthesia, *n* (%)	49 (12.15)
Surgical treatment combined with antibiotics, *n* (%)	401 (99.5)
Use of single antibiotic, *n* (%): cefuroxime, lincomycin, penicillin, gentamicin, amoxicillin + clavulanic acid, meropenem, clindamycin, azithromycin, vancomycin	83 (20.59)
Multi-antibiotic therapy:	
Use of 2 antibiotics, *n* (%):	290 (71.96)
Second-choice antibiotics: metronidazole, cefazolin, cefuroxime, clindamycin, gentamycin	
Use of 3 antibiotics, *n* (%)	28 (6.94)
Third-choice antibiotics: gentamycin, cefuroxime, metronidazole, penicillin, clindamycin, lincomycin,	
Intubation, *n* (%)	50 (12.4)
Tracheostomy, *n* (%)	3 (0.74)
Death, *n* (%)	1 (0.24)

SD – standard deviation, n – number, % – percentage, BMI – body mass index, WBC – white blood cells, ESR – erythrocyte sedimentation rate, CRP – C-reactive protein.

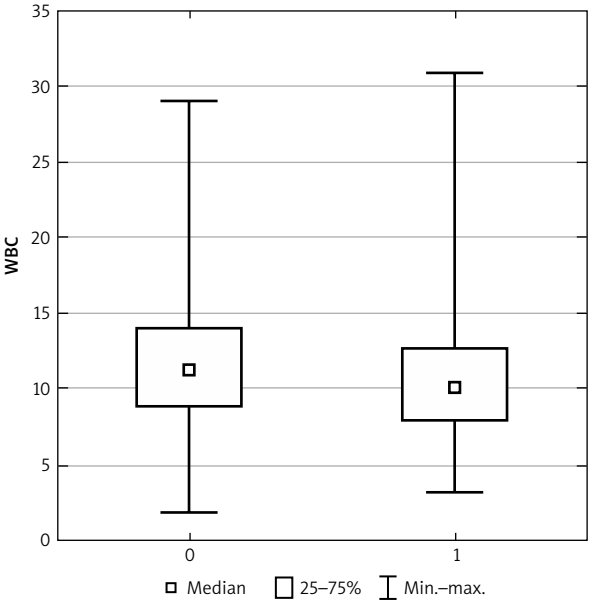

Figure 1. A statistically significant difference between WBC levels in patients with non-odontogenic (0) and patients with odontogenic (1) infections

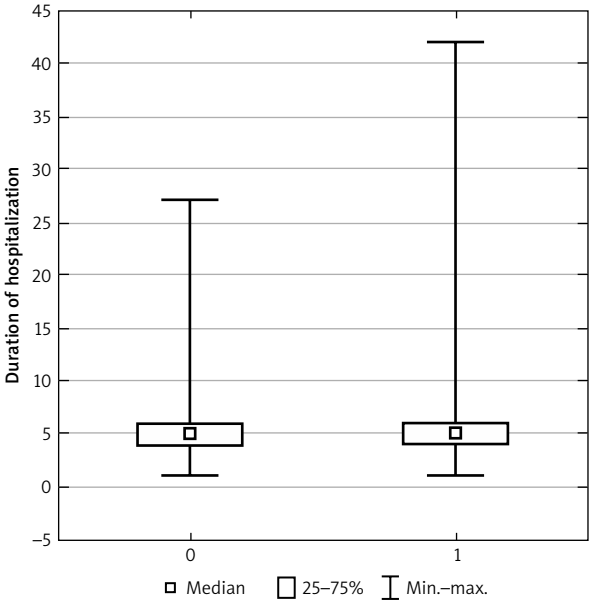

Figure 2. A statistically significant difference in duration of hospitalization between non-odontogenic (0) and odontogenic (1) infections

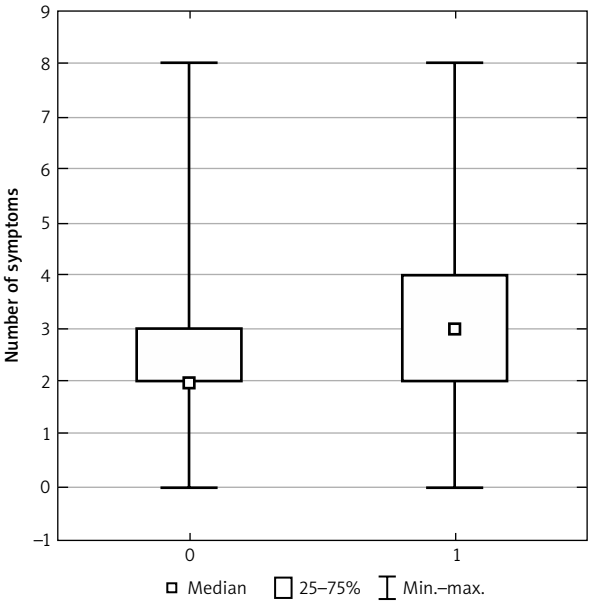

Figure 3. A statistically significant difference in number of symptoms in non-odontogenic (0) and odontogenic (1) infections

20 years of age, 50.12% were 21–40 years old, 24.15% were 41–60 years old, and 17.61% were > 60 years old. Predominant symptoms in maxillofacial infection were pain (92.5%), followed by dysphagia (49.5%), trismus (45.5%), and neck swelling (42.5%). The most frequently involved areas were peritonsillar (46.2%), submandibular (27.96%), and buccal (16.41%).

Comparison of clinical, demographic and microbiological profile between patients with odontogenic and non-odontogenic infections

Both groups were homogeneous in age and gender distribution as well as body mass index (BMI) values. No age or gender differences were noted between the 2 compared study groups. In the analysis of laboratory parameters, differences were detected in the WBC level between patients with odontogenic and non-odontogenic infections. The value of WBC was significantly higher in non-odontogenic infections (0) compared to odontogenic infections (1) ($p = 0.007$) (Figure 1).

There were no differences in CRP and ESR values between odontogenic and non-odontogenic infections. The duration of hospitalisation due to odontogenic infections was longer than in those resulting from non-odontogenic causes ($p = 0.037$) (Figure 2).

The prevalence of pain and dysphagia in patients with non-odontogenic infections was significantly higher than in patients with odontogenic infections ($p = 0.013$ and $p < 0.001$, respectively). In turn, odontogenic infections were characterised by a significantly greater number of accompanying symptoms, as shown in Figure 3, richer symptomatology of the inflammatory process, and more frequent neck swelling compared to non-odontogenic infections.

Submandibular, buccal, submental, and infratemporal areas were statistically more often involved in odontogenic infections, indicating a clear relationship between the location of these infections and their origin. In turn, the peritonsillar area was involved more

frequently in non-odontogenic infections in comparison with odontogenic infections ($p < 0.001$). Either tonsilitis or sialadenitis were identified as a cause of maxillofacial infections significantly frequently in patients with non-odontogenic infections ($p < 0.001$ and $p = 0.018$, respectively). Among the possible causes of non-odontogenic infections, tonsilitis, sialadenitis, sinusitis, a postoperative cause, and a systemic cause were detected. Odontogenic cause of sinusitis was excluded in all patients enrolled on the study on the basis of dental examination and CBCT scans of the maxillary sinuses. All cases of sinusitis were classified as non-odontogenic infections. All teeth in the affected area were vital. Moreover, previous oroantral communication as a potential cause of sinusitis was excluded in all cases presented in the study. In turn, iatrogenic and posttraumatic infections were included in either non-odontogenic or odontogenic causes of infections. Statistically significant differences were noted between odontogenic and non-odontogenic infections in the applied anaesthesia and surgical treatment. In non-odontogenic infections, biopsy, incisions, and local anaesthesia were more often used compared to odontogenic infections ($p < 0.001$ for all). Odontogenic infections predisposed to general anaesthesia and intubation ($p < 0.001$ for all). A detailed comparison of selected clinical and laboratory data between the odontogenic and non-odontogenic patients is presented in Table 2.

Comparing the incidence of comorbidities between patients with odontogenic and non-odontogenic cause of infection, there was a higher likelihood of the odontogenic origin of infections in patients with peripheral vascular diseases, cerebrovascular diseases and connective tissue diseases ($p < 0.001$, $p = 0.001$, and $p = 0.041$, respectively), Table 2. Although a statistically significant difference was observed for peptic ulcer disease and renal failure, the low level of confidence precluded identifying these comorbidities as a factor predisposing to a higher incidence of either odontogenic or non-odontogenic maxillofacial infections.

Patients with odontogenic infections had statistically more comorbidities and statistically more diseases other than diabetes compared to patients in the non-odontogenic group ($p < 0.001$ for all) Figures 4 and 5.

Microbiological profile of patients with odontogenic and non-odontogenic maxillofacial infections

In 59.30% of the patients, a positive microbiological culture was obtained. In the group of odontogenic infections, a positive microbiological culture was obtained in 53.38% of the patients, whereas in the group of non-odontogenic infections, a positive microbiological culture was obtained in 61.1% of the patients. Single bacterial strains were isolated in 60.6% of the odontogenic infections, whereas the multi-bacterial nature of these infections was confirmed in 39.4% of the odontogenic infections. In odontogenic infections, the most frequently isolated strains were *Streptococcus mitis* and *Staphylococcus epidermidis*, whereas in non-odontogenic infections the most common strains were *Staphylococcus aureus* and *Streptococcus mitis*. In both odontogenic and non-odontogenic infections, aerobic cocci predominated. We found a large contribution of *Staphylococcus aureus* and *Staphylococcus epidermidis* as well as *Streptococcus mitis*, *Streptococcus anginosus*, and *Streptococcus haemolyticus*, in non-odontogenic infections in comparison to odontogenic infections. In turn, in odontogenic infections, *Streptococcus sanguinis* and *Streptococcus oralis* were detected more frequently in comparison to non-odontogenic infections. Among gram-negative bacteria *Pseudomonas aerigunosa*, *Escherichia coli*, and *Klebsiella pneumoniae* predominated in non-odontogenic infections. Detailed data related to the microbiology of both odontogenic and non-odontogenic infections are presented in Table 3.

Discussion

Although the aetiology and clinical course of maxillofacial infections is well-known, the identification of factors specific to odontogenic and non-odontogenic infections requires careful study. The different aetiology of these infections is reflected in the participation of different factors modifying the course and severity of these infections and predisposing to them. To meet these needs, in the present study, a thorough analysis of infections of the head and neck area has been conducted and a comparison of odontogenic and non-odontogenic maxillofacial infections has been made. We found several important clinical and microbiological features by comparing odontogenic and non-odontogenic infections that facilitate their differentiation and implementation of the effective policy against them and can help identify groups of patients particularly vulnerable to these infections and their severe course.

The results obtained in our study on demographic data and their distribution de-pending on gender and age are similar to previous studies [1, 7–9]. In accordance with other studies, our research confirmed that both odontogenic and non-odontogenic infections occur most frequently in the 21–40-year-old age group [7]. We confirmed a mild male predilection for this kind of infections. Male predominance and the relatively young age of patients were consistent with previous studies [7, 10]. These results prove that in both odontogenic and non-odontogenic infections there are similar risk factors, and their greatest intensity is observed in young men aged 21–40 years. Moreover, some local factors and comorbidities, including diabetes mellitus, obesity, poor oral hygiene,

Table 2. Comparison of selected demographic, clinical, and laboratory data between patients with odontogenic (1) and non-odontogenic infections (0)

Parameters	Odontogenic Infections (1)	Non-odontogenic infection (0)	*P*-values
Number of individuals, *n*	180	223	
Age [years] median (confidence interval)	37 (18.0–90.0)	36.5 (7.0–91.0)	0.266[a]
Gender, female/male, *n* (%)	62 (46.62)/71 (53.38)	118 (43.7)/152 (56.3)	0.580[b]
BMI [kg/m^2] median (confidence interval)	24.02 (16.06–53.71)	24.16 (14.64–49.51)	0.776[a]
WBC [K/µl] median (confidence interval)	10.10 (3.23–31.0)	11.24 (1.9–29.0)	0.007[a]
ESR [mm/h] median (confidence interval)	50 (6.0–160.0)	46.0 (2.0–160.0)	0.608[a]
CRP [mg/l] median (confidence interval)	49.25 (1.2–425.4)	57.0 (1.56–421.0)	0.156[a]
Duration of hospitalisation [days] median (confidence interval)	5 (1–42)	5 (1–27)	0.037[a]
Symptoms:			
Number of symptoms, median (QRL)	3 (2)	2 (1)	< 0.001[a]
Pain *n*, yes/no	117 (87.97)/16 (12.03)	256 (94.81)/14 (5.19)	0.013[b]
Trismus, *n* (%) yes/no	69 (51.88)/64 (48.12)	114 (42.22)/156 (57.78)	0.067[b]
Dysphagia, *n* (%) yes/no	44 (33.08)/89 (66.92)	155 (57.41)/115 (42.59)	< 0.001[b]
Otalgia *n* (%) yes/no	7 (5.26)/126 (94.74)	14 (5.19)/256 (94.81)	0.973[b]
Fever *n* (%) yes/no	20 (15.04)/113 (84.96)	35 (12.96)/235 (87.04)	0.568[b]
Neck swelling *n* (%) yes/no	103 (77.44)/30 (22.56)	68 (25.28)/201(74.72)	< 0.001[b]
Dyspnoea *n* (%) yes/no	1 (0.75)/132 (99.25)	7 (2.60)/262 (97.4)	0.279[c]
Hoarseness *n* (%) yes/no	1 (0.75)/132 (99.25)	4 (1.48)/266 (98.52)	1.000[c]
Sialorrhea *n* (%) yes/no	3 (2.26)/130 (97.74)	2 (0.74)/268 (99.26)	0.337[c]
Other symptoms *n* (%) yes/no	69 (51.88)/64 (48.12)	38 (14.07)/232 (85.93)	< 0.001[b]
Space involvement:			
Submandibular *n* (%) yes/no	74 (55.64)/59 (44.36)	42 (15.56)/228 (84.44)	< 0.001[b]
Parapharyngeal *n* (%) yes/no	4 (3.01)/129 (96.99)	8 (2.96)/262 (97.04)	1.000[c]
Peritonsillar *n* (%) yes/no	1 (0.75)/132 (99.25)	176 (65.19)/94 (34.81)	< 0.001[b]
Buccal *n* (%) yes/no	42 (31.58)/91 (68.42)	30 (11.11)/240 (88.89)	< 0.001[b]
Parotid *n* (%) yes/no	2 (1.50)/131 (98.50)	6 (2.22)/264 (97.78)	1.000[c]
Temporal *n* (%) yes/no	2 (1.50)/131 (98.50)	3 (1.11)/267 (98.89)	0.666[c]
Infratemporal *n* (%) yes/no	4 (3.01)/129 (96.99)	0 (0.00)/270 (100)	0.011c
Submental *n* (%) yes/no	7 (5.26)/126 (94.74)	2 (0.74)/268 (99.26)	0.007[c]
Orbital *n* (%) yes/no	2 (1.50)/131 (98.50)	3 (1.11)/267 (98.89)	0.666[c]
Lacrimal sac *n* (%) yes/no	0 (0)/133 (100)	1 (0.37)/268 (99.63)	1.000[c]
Cervical *n* (%) yes/no	12 (9.02)/121 (90.98)	18 (6.67)/252 (93.33)	0.422[c]
Causes:			
Tonsillitis *n* (%) yes/no	0 (0)/133 (100)	179 (66.30)/91 (33.70)	< 0.001[b]
Sialadenitis *n* (%) yes/no	0 (0)/133 (100)	11 (4.07)/259 (95.93)	0.018[c]
Sinusitis *n* (%) yes/no	0 (0)/133 (100)	3 (1.11)/267 (98.89)	0.553[c]
Posttraumatic *n* (%) yes/no	1 (0.75)/132 (99.25)	14 (5.19)/256 (94.81)	0.025[c]

Table 2. Cont.

Parameters	Odontogenic Infections (1)	Non-odontogenic infection (0)	*P*-values
Postoperative *n* (%) yes/no	0 (0)/133 (100)	11 (4.07)/259 (95.93)	0.018[c]
Iatrogenic *n* (%) yes/no	3 (2.26)/130 (97.74)	2 (0.74)/268 (99.26)	0.337[c]
Systemic *n* (%) yes/no	0 (0)/133 (100)	5 (1.85)/265 (98.15)	0.175[c]
Unidentified *n* (%) yes/no	0 (0)/133 (100)	32 (11.85)/238 (88.15)	< 0.001[b]
Treatment and complications:			
No anaesthesia *n* (%) yes/no	5 (3.76)/128 (96.24)	6 (2.22)/264 (97.78)	0.516[c]
Local anaesthesia *n* (%) yes/no	88 (66.17)/45 (33.83)	237 (87.78)/33 (12.22)	< 0.001[b]
General anaesthesia n (%) yes/no	34 (25.56)/99 (74.44)	15 (5.56)/255 (94.44)	< 0.001[b]
Antibiotics *n* (%) yes/no	132 (99.25)/1 (0.75)	269 (99.63)/1 (0.37)	0.551[c]
Biopsy *n* (%) yes/no	78 (58.65)/55 (41.35)	238 (88.15)/32 (11.85)	< 0.001[b]
Incision *n* (%) yes/no	94 (70.68)/39 (29.32)	241 (89.26)/29 (10.74)	< 0.001[b]
Intubation *n* (%) yes/no	30 (22.56)/103 (77.44)	20 (7.41)/250 (92.59)	< 0.001[b]
Tracheostomy *n* (%) yes/no	0 (0)/133 (100)	3 (1.11)/267 (98.89)	0.553[c]
Death *n* (%) yes/no	0 (0)/133 (100)	1 (0.37)/269 (99.63)	1.000[c]
Accompanying diseases:			
Number of accompanying diseases, median (QRL)	0 (1)	0 (0)	< 0.001[a]
Number of accompanying diseases excluding diabetes, median (QRL)	0 (1)	0 (0)	< 0.001[a]
Myocardial infarction *n* (%) yes/no	1 (0.75)/132 (99.25)	3 (1.11)/267 (98.89)	1.000[c]
Circulatory failure *n* (%) yes/no	16 (12.12)/116 (87.88)	20 (7.41)/250 (92.59)	0.137[c]
Peripheral vascular diseases *n* (%) yes/no	31 (23.31)/102 (76.69)	14 (5.20)/255 (94.80)	< 0.001[b]
Cerebrovascular diseases *n* (%) yes/no	10 (7.52)/123 (92.48)	3 (1.12)/265 (98.88)	0.001[c]
Dementia *n* (%) yes/no	3 (2.26)/130 (97.74)	3 (1.11)/267 (98.89)	0.401[c]
Chronic obstructive pulmonary disease *n* (%) yes/no	3 (2.26)/130 (97.74)	5 (1.85)/265 (98.15)	0.722[c]
Connective tissue diseases *n* (%) yes/no	5 (3.76)/128 (96.24)	2 (0.74)/268 (99.26)	0.041[c]
Peptic ulcer disease *n* (%) yes/no	3 (2.26)/130 (97.74)	0 (0)/269 (100)	0.035[c]
Stroke *n* (%) yes/no	3 (2.26)/130 (97.74)	1 (0.37)/269 (99.63)	0.107[c]
Diabetes without complications *n* (%) yes/no	12 (9.02)/121 (90.98)	18 (6.67)/252 (93.33)	0.396[b]
Diabetes with complications *n* (%) yes/no	3 (2.26)/130 (97.74)	5 (1.85)/265 (98.15)	0.722[c]
Renal failure *n* (%) yes/no	4 (3.01)/129 (96.99)	1 (0.37)/269 (99.63)	0.042[c]
Liver failure *n* (%) yes/no	4 (3.03)/128 (96.97)	2 (0.75)/266 (99.25)	0.095[c]
Paresis *n* (%) yes/no	1 (0.75)/132 (99.25)	0 (0)/270 (100)	0.330[c]
Solid tumours *n* (%) yes/no	3 (2.26)/130 (97.74)	3 (1.11)/267 (98.89)	0.401[c]
Disseminated tumours *n* (%) yes/no	1 (0.75)/132 (99.25)	0 (0)/269 (100)	0.330[c]

n – number, % – percentage, BMI – body mass index, WBC – white blood cells, ESR – erythrocyte sedimentation rate, CRP – C-reactive protein, [a]Mann-Whitney U test, [b]χ^2 Pearson's test, [c]Fisher's exact test, ORL – interquartile ranges.

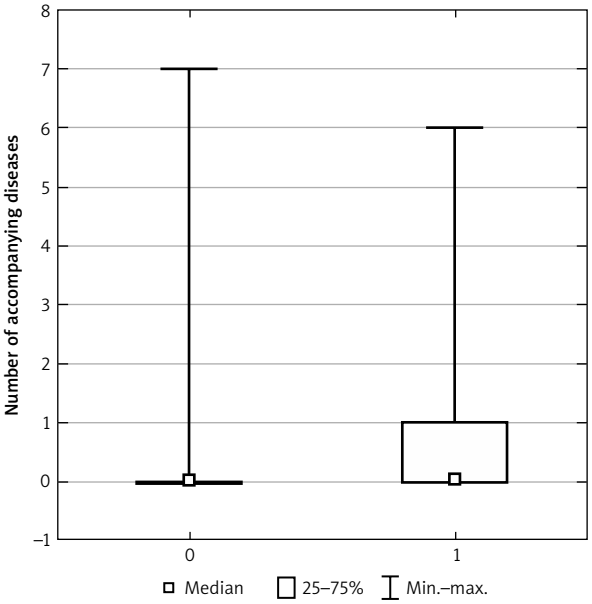

Figure 4. A statistically significant difference in the number of accompanying diseases in non-odontogenic (0) and odontogenic (1) infections

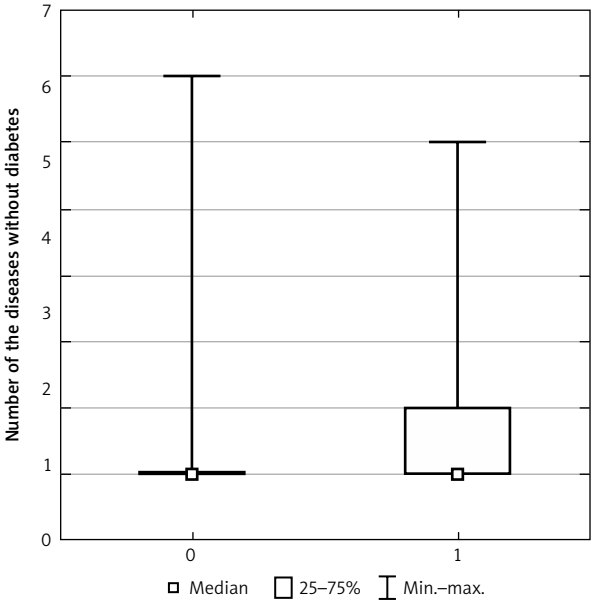

Figure 5. A statistically significant difference in the number of accompanying diseases excluding diabetes in non-odontogenic (0) and odontogenic (1) infections

and long-term nicotine or alcohol abuse, have so far been identified as potential predisposing factors for septic progression of odontogenic infections [1]. In our opinion, for both types of infection, smoking and drinking alcohol could be predisposing factors for their spread. They cause damage to the mucous membrane of the mouth and throat, which is the first immune barrier against the spread of microorganisms. Disruption to the physiological immune balance can lead to the development of both odontogenic and non-odontogenic maxillofacial infections. Another predisposing factor for maxillofacial infections is poor oral hygiene. It has traditionally been attributed as an important risk factor for odontogenic infections, but it can also significantly impair local immune defences and promote the spread of potentially benign pharyngeal infections. It is in the 21–40 age group that the consequences of neglecting oral hygiene, caries, and periodontitis may be most clearly marked, which, combined with limited access to the dentist, may result in a higher risk of maxillofacial infections. In this age group, avoiding visits in the dentist's and neglect of oral hygiene may result in the first symptoms of periodontitis and caries, leading to severe odontogenic maxillofacial infections. Both severe caries and periodontitis are the main causes of early tooth loss. Finally, in this age group, pathologies related to the eruption of the lower third molars are most common, which are postulated as a frequent cause of severe odontogenic infections [2, 11]. This could significantly increase the incidence of odontogenic infections in this age group.

Although odontogenic and non-odontogenic head and neck infections have similar clinical signs, such as neck swelling, trismus, pain, and dysphagia, some symptoms can help distinguish between odontogenic and non-odontogenic origin of these infections. Moreover, some of these symptoms including dysphonia, fever, dyspnoea, anterior floor oedema, limitation of tongue protraction, oropharyngeal oedema may indicate a more severe course [2, 12]. It is worth noting that trismus traditionally attributed to odontogenic infections is not a symptom differentiating odontogenic from non-odontogenic infections. In turn, such a specific symptom for non-odontogenic infections may be dysphagia, and for odontogenic infections swelling of the neck. These findings are consistent with the previous studies that indicated pain and swelling as predominant symptoms of odontogenic infections [13]. The differences observed in clinical symptoms between odontogenic and non-odontogenic symptoms also reflect preferences in occupying specific fascial spaces. Statistically, more frequent involvement of the submandibular space in odontogenic infections causes more frequent swelling of the neck. On the other hand, the involvement of the peritonsillar and parapharyngeal space in non-odontogenic infections is not manifested by increased swelling of the neck. Furthermore, the relatively rare occurrence of fever in our study is a consequence of the small participation of children in our study group. Previous studies have confirmed that fever is more common in children as a symptom of maxillofacial infections [9]. Due to their similar

Table 3. Comparison of microbiological profiles between odontogenic and non-odontogenic infections

Detected pathogens	Odontogenic infections	Non-odontogenic infections
Microbiology, *n*:		
Gram-positive bacteria:		
Staphylococcus:		
aureus	5	34
epidermidis	16	21
capitis	2	2
hominis	1	2
warneri	1	3
auricularis	0	1
xylosus	0	1
pasteuri	0	1
Unidentified	0	3
Streptococcus:		
mitis	20	26
anginosus	8	18
haemolyticus	1	11
sanguinis	8	3
mutaris	0	9
oralis	7	0
viridans	2	5
Identified as gr C	0	2
salivarius	2	1
parasanguinis	3	0
constellatus	1	2
pluranimalium	1	2
intermedius	2	0
pseudoporcinus	0	2
agalactiae	1	1
ovis	0	1
gordonii	0	1
liquefaciens	0	1
vestibularis	1	0
Unidentified	1	6
Identified as Beta Haemolytic *streptococcus*	1	0
Rothia mucilaginosa	1	0
Enterococcus:		
faecalis	2	3
casseliflavus	1	0

Table 3. Cont.

Detected pathogens	Odontogenic infections	Non-odontogenic infections
Leuconostoc mesenteroides	2	0
Eggerthella lenta	1	1
Gram-negative bacteria:		
Pseudomonas aerigunosa	2	9
Pseudomonas fluorescens	1	0
Acinetobacter baumannii	0	2
Escherichia coli	2	5
Klebsiella pneumoniae	1	5
Moraxella catarrhalis	0	1
Citrobacter:		
freundii	2	1
braakii	1	1
Enterobacter cloacae	4	2
Serratia marcescens	1	1
Anaerobic bacteria:		
Leuconostoc mesenteroides	2	0
Eggerthella lenta	1	1
Escherichia coli	2	5
Morganella morganii	1	0
Peptoniphilus harei	0	1
Fusobacterium nucleatum	1	0
Fungi: *Candida albicans*	8	5

location, both odontogenic and non-odontogenic infections cause several similar clinical symptoms. However, odontogenic infections are characterised by a richer symptomatology. A significantly greater number of clinical symptoms in odontogenic infections compared to non-odontogenic infections has been noted. In addition, these infections more often caused the occurrence of non-specific symptoms and thus far have not been directly associated with the ongoing infection.

In our study, pharyngotonsillitis and odontogenic infections were identified as the most common cause of maxillofacial infections. This result is consistent with some previous studies [8, 9, 14, 15]. The most common space involvement presented in our study reflects the primary cause of infection, and this was closely related to the symptoms reported by patients, such as trismus, dysphagia, and neck swelling. Parapharyngeal and peritonsillar abscess as the most common space involvement in non-odontogenic infections was also reported in previous studies [14]. Most of the odontogenic infections indicate the submandibular space as being the most common location of these infections, followed by buccal, submental, and infratemporal spaces [10]. Our study confirmed that these spaces may point to the potential odontogenic origin of diagnostically difficult infections.

Both odontogenic and non-odontogenic infections caused a similar duration of hospitalisation assessed for 5 days. This finding is close to the results obtained in previous studies [10, 16, 17]. However, in our study, odontogenic infections resulted in a longer duration of hospitalisation than non-odontogenic infections. It seems that this may be related to the delay in the implementation of effective surgical treatment and causative treatment of odontogenic infections, which are often performed on ambulatory basis. According to Sànchez *et al*., the mean hospital stay due to odontogenic infections was 4.24 days, but it may be prolonged due to the inefficiency of the primary surgical treatment. Furthermore, a lack of effective drainage is the most common reason for readmission to hospital in odontogenic infections. The same authors revealed a strong correlation between the duration of hospitali-

sation and diabetes. However, it was not dependent on the previously used antibiotic therapy [16].

Another factor postulated as potentially modifying the course of both odontogenic and non-odontogenic infections are accompanying systemic diseases. In our previous study, we indicated that diabetes, age, and multimorbidity are important factors that worsen the prognosis and cause a more severe course of maxillofacial infections [18]. Some previous reports have postulated that diabetes is the main factor modifying the course of these infections. Diabetic patients were more likely to experience complications, and they required aggressive surgical and pharmacological treatment, as well as a longer hospital stay [19–21]. Other bacterial strains have also been shown to be present in infections in diabetic patients compared to non-diabetic patients [22]. However, it seems that the impact of accompanying diseases is more significant for odontogenic infections than non-odontogenic infections. In our study, we showed that patients with odontogenic infections had a statistically higher number of comorbidities than patients with non-odontogenic infections. Diabetes was not a predisposing factor determining and differentiating between odontogenic and non-odontogenic diabetes itself. In our study, the distribution of comorbidities in maxillofacial infections was largely consistent with previous reports. Traditionally, an increased incidence of these infections was noted in patients with peripheral and cerebral circulation disorders, diabetes, obesity, uraemia or chronic renal insufficiency, acute myeloid leukaemia undergoing chemo or radiotherapy, bleeding dyscrasias, malnutrition, during treatment with glucocorticoids, and during immunosuppression [1, 9, 23, 24]. Moreover, dental caries are a potent trigger factor of hypertension, indicating a close relationship between circulatory disorders and dental caries and complications such as odontogenic infections [25]. This modifying effect of comorbidities was manifested in a greater number of occupied fascial spaces, a greater number of complications, and a longer hospital stay. A potential mechanism responsible for the increased risk of odontogenic infections among affected patients is a local vascular disorder; hence, there is a large proportion of patients with circulatory disorders among patients with odontogenic infections. Limited blood supply to the mandible caused by a different anatomical and histological structure also explains the more frequent involvement of the mandibular fascial spaces in the case of odontogenic infections. In this pathomechanism, local blood supply failure is the major cause of the infection spread. These disorders are important for the development of odontogenic infections. It is consistent with a previous study by Seppänen *et al*., who traced recent tendencies in the occurrence of odontogenic infections. They compared 2 cohorts of patients with odontogenic infections from 1994 and from 2004, respectively. The proportion of patients with cardiovascular diseases and hypertension significantly increased within one decade. Moreover, patients in 2004 presented a more severe course of infections with higher WBC and CRP values, and a longer hospital stay. Among patients with odontogenic infection, 85% of healthy patients developed local complications, whereas 75% of medically compromised patients developed systemic infection complications with a need for longer hospital stays and a higher risk of death [26]. Similar findings were obtained by Gams *et al*., who revealed that an American Society of Anesthesiologists score of 3 was associated with a longer hospital stay, due to odontogenic infections [17]. If we assume that most odontogenic infections in healthy people take the form of a vestibular abscess, then the more frequent occurrence of a severe course of odontogenic infections in compromised patients proves their significant impact on the development of these infections and can be treated as a risk factor. Moreover, some studies indicated psychiatric disorders as relevant predictors of the complex evolution of odontogenic infections [2]. Their contribution to severe odontogenic infections increases systematically with time [26]. In our opinion, connective tissue diseases could be another risk factor for odontogenic infection development. They disturb the physiological function of the immunological system. Moreover, steroids and immunosuppressants often used in their treatment may predispose to odontogenic infections. The more frequent general anaesthesia and intubation in the course of odontogenic infections compared to non-odontogenic infections may result from a greater number of comorbidities, which already induces their more severe course at the time of admission to the hospital.

In our study, patients with both odontogenic and non-odontogenic infections presented elevated ESR, CRP, and WBC levels. This is consistent with findings from previous studies [27–30]. In our previous study, we confirmed that CRP was the most sensitive laboratory parameter of maxillofacial infections, as well as not being dependent on the number and nature of accompanying diseases [18]. Therefore, we did not find any differences in CRP levels between odontogenic and non-odontogenic infections that were characterised by various accompanying diseases. In turn, WBC value negatively correlated with accompanying diseases. These previous observations may explain the statistically significant differences in WBC levels between odontogenic infections and non-odontogenic infections detected in the present study. These findings result from the higher morbidity of patients with odontogenic infections. However, previous multivariate analysis indicated that CRP levels could be combined with other predictive factors such as penicillin allergy, psychiatric disorders, and immunodepression [2]. A higher level of CRP is associated with a more severe course of maxillofacial infections. According

to Pham Dang *et al.*, patients with a CRP level higher than 200 mg/l have a risk factor of multiple surgeries due to odontogenic infections, which is assessed at 27% [2]. In turn, a CRP level lower than 50 mg/l and with immunodepression condition may predispose to a more severe course of odontogenic infections [2]. These mutual relationships require further research.

The results obtained in our study on microbiological data in odontogenic infections are consistent with previous studies [1, 3, 31]. *Streptococcus* species are still the commonest pathogen in orofacial infections of odontogenic origin. There are no relevant differences in the distribution of the main detected pathogens in our study, *Streptococcus mitis* and *Staphylococcus epidermidis*, between odontogenic and non-odontogenic infections. Most isolated pathogens in both odontogenic and non-odontogenic infections were classified as *Streptococcus viridans*. This broad group of aerobic cocci including *Streptococcus mitis*, *Streptococcus oralis*, and *Streptococcus sanguinis* predominated in both groups. These bacterial species are specific to oral cavity, and they could be causative pathogens for both types of infections. The insignificant changes in the distribution of the mentioned bacteria may result from comorbidities, oral hygiene, and lifestyle and not be the result of different causes of infection [8]. A noteworthy difference in the bacterial profile of non-odontogenic infections compared to odontogenic infections is the high proportion of group A streptococci and the *Streptococcus milleri* group, especially *S. anginosus*, which is assessed as being the most commonly isolated aerobes in peritonsillar abscess [32–34]. Another significant difference in the distribution of bacteria between odontogenic and non-odontogenic infections is the relatively high proportion of *Staphylococcus aureus* in non-odontogenic infections compared to odontogenic ones. Some reports noted a high distribution of *Staphylococcus aureus* in peritonsillar abscess [35]. This may reflect a different primary cause of non-odontogenic infections and their primary location in the nasopharynx. On the other hand, a relatively high contribution of *Staphylococcus aureus* also in odontogenic infections was revealed in a study conducted by Jagadish Chandra *et al.*, who isolated *Staphylococcus aureus* in odontogenic infections by 16S rRNA gene sequencing [36]. Another difference of note is the high proportion of *Klebsiella pneumoniae* in non-odontogenic infections. These reports are consistent with the study by Tsai *et al.*, who indicated *Klebsiella pneumoniae* as the second (after streptococci) bacteria isolated in peritonsillar abscesses [8]. Similar results were obtained by Yang *et al.*, who confirmed that *Streptococcus Viridans*, *Staphylococcus aureus*, and *Klebsiella pneumoniae* are the most common causative bacteria in deep neck infections [9]. The complex aerobic and anaerobic nature of odontogenic infections was confirmed in our study, and their bacteriological results reflect the regularity that metabolites produced by earlier bacterial strains are a suitable substrate for the development of subsequent pathogens [37]. This causes the composition of individual bacterial strains to evolve. Slight shifts in the composition of pathogens within both groups may result from the time of swab collection, as well as from the changing nature of the infection from cellulitis to full-blown abscess.

Conclusions

The results obtained here allow us to classify comorbidities as the most important risk factor for the development and unfavourable progression of odontogenic infections. A similar relationship does not apply to the development of non-odontogenic infections. Patients with circulatory disorders are particularly at risk of severe course of odontogenic infections. Not only diabetes, which is traditionally attributed to odontogenic infections, and advanced age of patients, but also multimorbidity and circulatory disorders should be included as a significant risk group for the severe course of odontogenic infections requiring hospitalisation. This should be reflected in the implementation of close monitoring of these infections in the risk group and the use of more aggressive treatment methods in the initial stage of infections.

Funding

No external funding.

Ethical approval

Bioethics Committee of Holycross Medical Chamber, Poland (No 11/2021-VIII).

Conflict of interest

The authors declare no conflict of interest.

References

1. Weise H, Naros A, Weise C, Reinert S, Hoefert S. Severe odontogenic infections with septic progress – a constant and increasing challenge: a retrospective analysis. BMC Oral Health. 2019 Aug; 19(1): 173.
2. Pham Dang N, Delbet-Dupas C, Mulliez A, Devoize L, Dallel R, Barthélémy I. Five predictors affecting the prognosis of patients with severe odontogenic infections. Int J Environ Res Public Health. 2020 Dec; 17(23): 8917.
3. Jevon P, Abdelrahman A, Pigadas N. Management of odontogenic infections and sepsis: an update. Br Dent J. 2020 Sep; 229(6): 363-370.
4. Haghighat S, Rezazadeh F. Prevalence of non-odontogenic infectious lesions of oral mucosa in a group of Iranian patients during 11 years: a cross sectional study. Iran J Microbiol. 2019 Oct; 11(5): 357-362.
5. Bagul R, Chandan S, Sane VD, Patil S, Yadav D. Comparative evaluation of C-reactive protein and WBC count in

fascial space infections of odontogenic origin. Comparative evaluation of C-reactive protein and WBC count in fascial space infections of odontogenic origin. J Maxillofac Oral Surg. 2017 Jun; 16(2): 238-242.
6. Błochowiak KJ, Kamiński B, Sokalski J. Deep neck infections of non-odontogenic origin: clinical manifestation and treatment. Med Stud. 2018 Mar; 34(1): 98-102.
7. Zawiślak E, Nowak R. Odontogenic head and neck region infections requiring hospitalization: an 18-month retrospective analysis. Biomed Res Int. 2021; 2021: 7086763.
8. Tsai YW, Liu YH, Su HH. Bacteriology of peritonsillar abscess: the changing trend and predisposing factors. Braz J Otorhinolaryngol. 2018 Sep-Oct; 84(5): 532-539.
9. Yang W, Hu L, Wang Z, Nie G, Li X, Lin D, Luo J, Qin H, Wu J, Wen W, Lei W. Deep neck infection: a review of 130 cases in Southern China. Medicine. 2015 Jul; 94(27): e994.
10. Katoumas K, Anterriotis D, Fyrgiola M, Lianou V, Triantafylou D, Dimopoulos I. Epidemiological analysis of management of severe odontogenic infections before referral to the emergency department. J Craniomaxillofac Surg. 2019 Aug; 47(8): 1292-1299.
11. Trybek G, Chruściel-Nogalska M, Machnio M, Smektała T, Malinowski J, Tutak M, Sporniak-Tutak K. Surgical extraction of impacted teeth in elderly patients. A retrospective analysis of perioperative complications – the experience of a single institution. Gerodontology. 2016 Sep; 33(3): 410-415.
12. Neal TW, Schlieve T. Complications of severe odontogenic infections: a review. Biology (Basel). 2022 Dec; 11(12): 1784.
13. Uluibau IC, Jaunay T, Goss AN. Severe odontogenic infections. Aust Dent J. 2005 Dec; 50 (4 Supp 2): S74-81.
14. Kauffmann P, Cordesmeyer R, Tröltzsch M, Sömmer C, Laskawi R. Deep neck infections: a single-center analysis of 63 cases. Med Oral Patol Oral Cir Bucal. 2017 Sep; 22(5): e536-e541.
15. Martínez Pascual P, Pinacho Martinez P, Friedlander E, Martin Oviedo C, Scola Yurrita B. Peritonsillar and deep neck infections: a review of 330 cases. Braz J Otorhinolaryngol. 2018 May-Jun; 84(3): 305-310.
16. Sánchez R, Mirada E, Arias J, Paño JR, Burgueño M. Severe odontogenic infections: epidemiological, microbiological therapeutic factors. Med Oral Patol Oral Cir Bucal. 2011 Aug; 16(5): e670-e676.
17. Gams K, Shewale J, Demian N, Khalil K, Banki F. Characteristics, length of stay, and hospital bills associated with severe odontogenic infections in Houston. J Am Dent Assoc. 2017 Apr; 148(4): 221-229.
18. Kamiński B, Błochowiak K, Kołomański K, Sikora M, Karwan S, Chlubek D. Oral and maxillofacial infections--a bacterial and clinical cross-section. J Clin Med. 2022 May; 11(10): 2731.
19. Rao DD, Desai A, Kulkarni RD, Gopalkrishnan K, Rao CB. Comparison of maxillofacial space infection in diabetic and nondiabetic patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2010 Oct; 110(4): e7-e12.
20. Zheng L, Yang C, Zhang W, Cai X, Kim E, Jiang B, Wang B, Pu Y, Wang J, Zhang Z, Zhou J, Guan X. Is there association between severe multispace infections of the oral maxillofacial region and diabetes mellitus? J Oral Maxillofac Surg 2012 Jul; 70(7): 1565-1572.
21. Rahimi-Nedjat RK, Sagheb K, Sagheb K, Hormes M, Walter C, Al-Nawas B. The role of diabetes mellitus on the formation of severe odontogenic abscesses – a retrospective study. Clin Oral Investig. 2021 Nov; 25(11): 6279-6285.
22. Celakovsky P, Kalfert D, Smatanova K, Tucek L, Cermakova E, Mejzlik J, Kotulek M, Vrbacky A, Matousek P, Stanikova L, Hoskova T. Bacteriology of deep neck infections: analysis of 634 patients. Aust Dent J. 2015 Jun; 60(2): 212-215.
23. Meurman JH, Hämäläinen P. Oral health and morbidity – implications of oral infections on the elderly. Gerodontology. 2006 Mar; 23(1): 3-16.
24. Bakathir AA, Moos KF, Ayoub AF, Bagg J. Factors contributing to the spread of odontogenic infections. Sultan Qaboos Univ Med J. 2009 Dec; 9(3): 296-304.
25. Ostalska-Nowicka D, Paszyńska E, Dmitrzak-Węglarz M, Neyman-Bartkowiak A, Rabiega A, Zachwieja J, Nowicki M. Dental caries-related primary hypertension in children and adolescents: cross-sectional study. Oral Dis. 2021 Oct; 27(7): 1822-1833.
26. Seppänen L, Rautemaa R, Lindqvist C, Lauhio A. Changing clinical features of odontogenic maxillofacial infections. Clin. Oral Investig. 2010 Aug; 14(4): 459-465.
27. Sharma A, Giraddi G, Krishnan G, Shahi AK. Efficacy of serum prealbumin and CRP levels as monitoring tools for patients with fascial space infections of odontogenic origin: a clinicobiochemical study. J Maxillofac Oral Surg. 2014 Mar; 13(1): 1-9.
28. Sharma A, Gokkulakrishnan S, Shahi AK, Kumar V. Efficacy of serum CRP levels as monitoring tools for patients with fascial space infections of odontogenic origin: a clinicobiochemical study. Natl J Maxillofac Surg. 2012 Jul--Dec; 3(2): 148-151.
29. Kusumoto J, Iwata E, Huang W, Takata N, Tachibana A, Akashi M. Hematologic and inflammatory parameters for determining severity of odontogenic infections at admission: a retrospective study. BMC Infect Dis. 2022 Dec; 22(1): 931.
30. Pricop M, Ancusa O, Talpos S, Urechescu H, Bumbu BA. The predictive value of systemic immune-inflammation index and symptom severity score for sepsis and systemic inflammatory response syndrome in odontogenic infections. J Pers Med. 2022 Dec; 12(1): 2026.
31. Fating NS, Saikrishna D, Vijay Kumar GS, Shetty SK, Raghavendra Rao M. Detection of bacterial flora in orofacial space infections and their antibiotic sensitivity profile. J Maxillofac Oral Surg. 2014 Dec; 13(4): 525-532.
32. Galioto NJ. Peritonsillar abscess. Am Fam Physician. 2017 Apr; 95(8): 501-506.
33. Johnston J, Stretton M, Mahadevan M, Douglas RG. Peritonsillar abscess: a retrospective case series of 1773 patients. Clin Otolaryngol. 2018; 43: 940–944.
34. Klug T, Greve T, Hentze M. Complications of peritonsillar abscess. Ann Clin Microbiol Antimicrob. 2020 Jul; 19(1): 32.
35. Mazur E, Czerwińska E, Korona-Głowniak I, Grochowalska A, Kozioł-Montewka M. Epidemiology, clinical history and microbiology of peritonsillar abscess. Eur J Clin Microbiol Infect Dis. 2015 Mar; 34(3): 549-554.
36. Jagadish Chandra H, Sripathi Rao BH, Muhammed Manzoor AP, Arun AB. Characterization and antibiotic sensitivity profile of bacteria in orofacial abscesses of odontogenic origin. J Maxillofac Oral Surg. 2017 Dec; 16(4): 445-452.

37. Fating NS, Saikrishna D, Vijay Kumar GS, Shetty SK, Raghavendra Rao M. Detection of bacterial flora in orofacial space infections and their antibiotic sensitivity profile. J Maxillofac Oral Surg. 2014 Dec; 13(4): 525-532.

Address for correspondence:

Katarzyna Justyna Błochowiak
Department of Oral Surgery,
Periodontal Diseases and
Oral Mucosal Diseases
Poznan University of Medical Sciences
Poznan, Poland
Phone: +48 608 836 850
E-mail: kasia@naszdentysta.com.pl

Received: 25.10.2023
Accepted: 15.05.2024
Online publication: 30.06.2024