

The association of the causative tooth extraction with inflammatory and hematological parameters in an odontogenic infection: A prospective observational cohort study

Shaheen Memon, Muhammad Shahzad, Sundas Kunbher, Ishrat Begum, Ram Pershad Chooni Lal

Liaquat University Hospital, Jamshoro Pakistan

ABSTRACT

Objective: This study aimed to assess the association of extraction of the causative tooth with inflammatory and hematological parameters and short-term clinical outcomes in patients with odontogenic infections.

Material and Methods: This prospective observational cohort study was carried out for six months (April 2025 to September 2025) in the Department of Oral and Maxillofacial Surgery, Liaquat University Hospital, Hyderabad. Using non-probability consecutive sampling, a total of 90 adult clinically and radiographically confirmed odontogenic infection patients were included. Based on treatment approach, patients were assigned into Group-I (n = 45) and Group-II (n = 45). Appropriate statistical tests were carried out for data analysis where $p < 0.05$ was considered significant.

Results: The average age of participants was 36.8 ± 10.4 years and the demographic and inflammatory parameters at baseline were similar between groups. The extraction group had a reduction of 8.2 mg/L of CRP from 16.8 ± 4.2 mg/L to 8.6 ± 3.1 mg/L, while the non-extraction group had a reduction of 5.2 mg/L of CRP from 16.4 ± 4.5 mg/L to 11.2 ± 3.5 mg/L. The extraction group also demonstrated a shorter hospital stay compared with the non-extraction group (3.9 ± 1.2 vs 5.0 ± 1.5 days; $p < 0.001$).

Conclusion: During the acute phase of odontogenic infection management, the removal of the causative tooth was found to be associated with greater reductions in inflammatory markers and improved short-term clinical outcomes.

Keywords: C-reactive protein, Dental extraction, Inflammation, Odontogenic infection, Tooth extraction

This article can be cited as: Memon S, Shahzad M, Kunbher S, Begum I, Lal RPC. The association of the causative tooth extraction with inflammatory and hematological parameters in an odontogenic infection: A prospective observational cohort study. Pak J Pathol. 2026; 37(3): 181-188. DOI: <https://doi.org/10.55629/pakjpathol.v37i3.1046>

INTRODUCTION

Odontogenic infections occur when microorganisms enter periapical and fascial spaces as a result of dental caries, periodontal disease, pericoronitis, or pulpal necrosis. While many cases may remain localized, untreated delayed cases have the potential for extension into the submandibular, sublingual, masticator, parapharyngeal and retropharyngeal spaces and will increase the risk of airway compromise, sepsis, mediastinitis, extended hospital stay and mortality. The biological parameters are thus able to confirm the clinical evaluation and will be used to assess the systemic inflammatory activity as well as the disease severity, the treatment response and its prognosis. Studies have demonstrated the utility of presepsin in the diagnosis of odontogenic infections and that presepsin could be used to

augment standard inflammatory markers to detect any bacterial infection [1]. Odontogenic infections can be a major clinical problem and can escalate from a local dental infection to a major fascial space infection, which needs to be managed in hospital. Moreover, biological markers have been shown to be useful tools for predicting the length of hospital stay and monitoring recovery in patients with odontogenic cellulitis [2].

High levels of C-reactive protein, total leukocytes count, blood glucose and body temperature are commonly measured parameters that are useful in evaluating the severity of odontogenic infections, where higher values indicate increased clinical severity [3]. Biological markers could also be predictive of hospital stay for patients with odontogenic cellulitis, regarding their possible use to monitor recovery and their resource needs [4]. Modern treatment focuses on early diagnosis, antibiotic therapy as needed, surgical removal of purulent collections and elimination of the underlying dental cause [5]. With large head and neck abscesses, prompt drainage (either by image guidance or surgery) can decrease the amount of

Correspondence: Dr. Shaheen Memon, FCPS Resident, Department of Oral and Maxillofacial Surgery, Liaquat University Hospital, Jamshoro Pakistan

Email: shaheenmemon33@gmail.com

Receiving Date: 26 Apr 2026

Revision Date: 08 Sep 2026

Acceptance Date: 27 Sep 2026



Copyright © 2026. Shaheen Memon, et al. This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License, which permits unrestricted use, distribution & reproduction in any medium provided that original work is cited properly.

infectious material; however, definitive source control is necessary to prevent recurrence or persistence [6]. The extraction of the odontogenic infections causative tooth is an essential part of dental source control of odontogenic infections and may affect systemic inflammatory responses. Changes in circulating myeloperoxidase levels following treatment of infected teeth suggest that elimination of the dental source may be associated with alterations in measurable inflammatory activity [7]. Other inflammatory indices, including the neutrophil-to-lymphocyte ratio and C-reactive protein levels, have also been reported as useful markers for assessing the severity and clinical response of odontogenic infections [8]. Moreover, Facial cellulitis of odontogenic origin, including cases occurring in children, requires timely dental intervention to prevent disease progression and hospitalization [9]. Furthermore, host-microbiome interactions in apical periodontitis provide biological plausibility that removal of an infected tooth may be associated with changes in local and systemic inflammatory responses [10].

There are, however, only a few clinical data about the early evolution of biological parameters after the treatment of the causative tooth in odontogenic infections. Thus, this prospective observational cohort study was designed to test the association between extraction of the causative tooth and inflammatory and hematological parameters in patients suffering from odontogenic infections, such as C reactive protein (CRP), neutrophil to lymphocyte ratio (NLR), total leukocyte count (TLC) and erythrocyte sedimentation rate (ESR).

MATERIAL AND METHODS

The study was a prospective observational cohort study that was conducted for six months (April 2025 – September 2025) in the Department of Oral and Maxillofacial Surgery, Liaquat University Hospital, Hyderabad. The study protocol was approved by Research Ethics Committee (REC) Liaquat University of Medical & Health Sciences (LUMHS), Jamshoro (Approval No. LUMHS/REC/-778 dated 26-03-2025). All subjects gave written informed consent for inclusion before they participated in the study, which was carried out in accordance with the ethical principles of the Declaration of Helsinki.

All adult patients (18-60 years) with a clinically and radiographically confirmed odontogenic infection who needed hospital admission or surgery were evaluated for eligibility. Diagnosis of odontogenic infection was

made if the clinical presentation was of dental-origin facial swelling, pain, erythema, fever, trismus and purulent discharge. Radiological evaluation was undertaken either by dental radiograph or any other suitable modality to determine the causative tooth, presence of odontogenic abscess and primary fascial space involvement. Patients in whom the pathognomonic tooth, incision and drainage, or both were performed were included for those with infections involving primary fascial spaces (buccal space, submandibular space, sublingual space). Patients who did not consent, those who had uncontrolled diabetes mellitus, immunocompromised illnesses, pregnancy, those on immunosuppressive therapy, those who had a non-odontogenic infection, those with a secondary fascial space involvement, and those who required extraction of a tooth unrelated to the current infection were excluded.

The sample size was determined to assess difference in inflammatory parameters of the patients with extraction of the causative tooth and with tooth retention within the biological system. Sample size calculation was performed using G*Power software version 3.1.9.7 based on a two-independent-sample t-test approach. The minimum sample size required per group was 39, based on a two-sample t-test design with an 80% sample power, a 5% significance level, an expected difference of 0.8 mg/L in the C-reactive protein (CRP) levels between the groups and assumed standard deviations of 1.2 mg/L and 1.3 mg/L for each group, respectively. To account for the possibility of incomplete data or loss to follow-up, the sample size was increased to 90 subjects (45 per group) to accommodate the 15% loss of subjects. Non-probability consecutive sampling technique was used to recruit the participants.

At the time of admission, detailed medical and dental history was obtained and a comprehensive clinical examination was performed. Demographic data, clinical signs and symptoms, axillary temperature, maximum mouth opening, fascial spaces involved, and dental findings were documented on a predetermined proforma. The prognosis of the causative tooth was made based on radiography and clinical examination. The participants were divided into two groups based on the clinical treatment planning decision on the management of the tooth causing the issue. For extraction group, the causative tooth was removed at the hospital visit as deemed non-restorable. Tooth was considered non-restorable if there was extensive carious destruction, grade III mobility, vertical root fracture, or insufficient tooth

structure to effectively restore. In the non-extraction group, the tooth responsible for the infection was deemed to be restorable and it was managed for the acute infection, but definitive dental management was planned post infection.

All the patients were treated in a uniform manner following institutional protocol. Empirical antimicrobial treatment was started based on institutional protocol for odontogenic infections in all patients. The initial antimicrobial treatment involved intravenous administration of ceftriaxone (1–2 g once a day) plus metronidazole (500 mg every 8 hours). Intravenous clindamycin was used as an alternative when clinically indicated for those who had a known allergy to β -lactams. Based on microbiological culture sensitivity, if available, antibiotic therapy was adjusted based on clinical response. When indicated, surgical management was undertaken, which involved incision and drainage of involved fascial spaces. For teeth that were deemed non-restorable, it was removed during the same hospital stay as the extraction group. Surgical management, including incision, drainage and debridement of involved fascial spaces was undertaken when clinically indicated. Pus samples were drawn aseptically prior to changing antibiotic therapy and sent for aerobic and anaerobic bacterial culture, and antimicrobial sensitivity testing. Patients in the extraction group had the tooth causing the infection removed at the same hospitalization. Paracetamol was given intravenously for pain relief, and patients were cared for until swelling, mouth opening, pain, purulent discharge and discharge criteria were fulfilled.

Venous blood samples were drawn from all the enrolled patients at two predetermined time points, upon admission before definitive dental intervention and three days after the start of dental intervention. Second blood sample was taken at the 3rd day after extraction of the causative tooth in the extraction group and at the 3rd day after conservative treatment of the causative tooth in the non-extraction group. In both groups, the same sampling times were used for comparison of the changes in the biological parameters with time. Biological parameters that were measured included total leukocytes count (TLC), neutrophil-to-lymphocyte ratio (NLR), C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR). The main exposure variable was the treatment of the tooth causing the toothache (extraction versus non-extraction). The main outcomes were the changes in the inflammatory markers such as CRP and NLR from the time of

admission to day 3 after treatment. Standardised laboratory procedures were used for laboratory investigations. An automated hematology analyzer (Sysmex XN-1000, Sysmex Corporation, Kobe, Japan) was used for complete blood count parameters analysis. The C-reactive protein (CRP) concentration was determined by an immunoturbidimetric method on an automated clinical chemistry analyzer (Cobas c 311, Roche Diagnostics, Mannheim, Germany). The analytical range of CRP is 0.3 to 350 mg/l and the erythrocyte sedimentation rate (ESR) was measured by the Westergren method. Neutrophil-to-lymphocyte ratio (NLR) was derived from absolute counts of neutrophils and lymphocytes from the complete blood count that were analysed.

All the data were entered and analyzed with IBM SPSS Statistics for Windows Version 27.0 (IBM Corp., Armonk, NY, USA). The following continuous variables were checked for normal distribution with the use of the Shapiro–Wilk test: age, maximum mouth opening, WBC count, CRP, NLR, ESR, duration of hospital stay. Variables that were normally distributed were reported as mean $\pm$ standard deviation (SD) and those that were not normally distributed were reported as median and interquartile range (IQR). Categorical variables such as gender, fever status, fascial space involvement, bacterial culture result, treatment group and resolution in the local infection signs were presented as frequency and percentage (n%). To compare the normally distributed continuous variables between extraction and non-extraction groups, independent sample t-test was used, and Mann–Whitney U test was used for non-normally distributed continuous variables. Chi-square test or Fisher's exact test was used for comparison of categorical variables between groups. Intragroup comparisons of inflammatory parameters (WBC, CRP, NLR, and ESR) were made from admission to day 3 after initiation of treatment using paired sample t-test. Two-sided P values <0.05 were considered statistically significant.

RESULTS

Of the total 90 cases of odontogenic infections, clinically and radiographically confirmed, were enrolled during the six months study period. Groups were matched between two treatment groups – Group I (causative tooth extraction, n=45) and Group II (non-extraction/conservative management, n=45). Inflammatory biomarkers and clinical outcomes were assessed at baseline and follow-up (Day 3) in all

patients. The main outcomes evaluated were the changes in inflammatory markers such as white blood cell count (WBC), C-reactive protein (CRP), neutrophil-to-lymphocyte ratio (NLR), and erythrocyte sedimentation rate (ESR). Secondary outcomes were length of hospital stay, results of bacterial culture, and clearance of signs of local infection.

The average age of the study group was 36.8 ± 10.4 years. There were no statistically significant differences between both groups for baseline demographic characteristics. The mean age of the extraction group was 37.2 ± 10.1 years, and the mean age of the non-extraction group was 36.4 ± 10.7 years. The male participants made up 62.2% (28/45) of the extraction group and 57.8% (26/45) of the non-extraction group. Of all patients 64.4% in the extraction group and 60.0% in the non-extraction group presented with fever at admission. The most frequently involved fascial space was the buccal space (33.3% vs 31.1%) followed by submandibular space. For both groups, the least common presentation was sublingual involvement. There was no significant difference in the clinical parameters at baseline, indicating a similar degree of disease severity prior to intervention (Table-I).

There were no significant differences in the baseline inflammatory parameters between the two groups. As shown in Table-II. The mean WBC count was $12,840 \pm 2,310$ cells/ μ L in the extraction group and $12,610 \pm 2,450$ cells/ μ L in the non-extraction group ($p=0.65$). Similarly, CRP levels (16.8 ± 4.2 vs 16.4 ± 4.5 mg/L; $p=0.67$), NLR (7.9 ± 2.1 vs 7.7 ± 2.3 ; $p=0.68$), and ESR values (41.2 ± 8.6 vs 40.7 ± 9.1 mm/hr; $p=0.79$) showed no significant difference at admission.

Independent sample t-test was used to perform these comparisons.

Both treatment groups showed statistically significant reductions in inflammatory and hematological parameters from admission to day 3 after initiation of treatment. In the extraction group, mean WBC count decreased from $12,840 \pm 2,310$ cells/ μ L at admission to $9,640 \pm 1,980$ cells/ μ L on day 3 ($p<0.001$). Similarly, CRP levels decreased from 16.8 ± 4.2 mg/L to 8.6 ± 3.1 mg/L ($p<0.001$), NLR from 7.9 ± 2.1 to 3.9 ± 1.4 ($p<0.001$), and ESR from 41.2 ± 8.6 mm/hr to 24.8 ± 6.3 mm/hr ($p<0.001$). In the non-extraction group, significant reductions were also observed in WBC count ($12,610 \pm 2,450$ to $10,820 \pm 2,050$ cells/ μ L; $p<0.001$), CRP (16.4 ± 4.5 to 11.2 ± 3.5 mg/L; $p<0.001$), NLR (7.7 ± 2.3 to 4.8 ± 1.6 ; $p<0.001$), and ESR (40.7 ± 9.1 to 29.6 ± 7.4 mm/hr; $p<0.001$) Table-III.

The inflammatory markers were significantly lower in the extraction group compared to the non-extraction group. The reduction in WBC count (3200 ± 980 vs 1790 ± 850 cells/ μ L), CRP (8.2 ± 2.6 vs 5.2 ± 2.3 mg/L), NLR (4.0 ± 1.3 vs 2.9 ± 1.2), and ESR (16.4 ± 5.1 vs 11.1 ± 4.7 mm/hr) was statistically significant (all $p<0.001$). Additionally, the extraction group had a significantly shorter hospital stay (3.9 ± 1.2 days vs 5.0 ± 1.5 days; $p<0.001$). No significant differences were found between the groups in positive bacterial culture ($p=0.58$), mixed aerobic and anaerobic growth ($p=0.67$) or resolution of the signs of local infection at discharge ($p=0.12$). Independent sample t-test was used to analyze continuous variables and chi-square/Fisher's exact test for categorical variables (Table-IV).

Table-I: Baseline demographic and clinical characteristics of study participants (n=90).

Variable	Extraction group (n=45)	Non-extraction group (n=45)
Age (years), mean $\pm$ SD	37.2 ± 10.1	36.4 ± 10.7
Male, n (%)	28 (62.2)	26 (57.8)
Female, n (%)	17 (37.8)	19 (42.2)
Fever at admission, n (%)	29 (64.4)	27 (60.0)
Maximum mouth opening (mm), mean $\pm$ SD	24.6 ± 5.8	24.2 ± 6.1
Hospital admission due to swelling/pain, n (%)	45 (100)	45 (100)
Involved fascial space n (%)		
Buccal space n (%)	15 (33.3)	14 (31.1)
Submandibular space n (%)	12 (26.7)	13 (28.9)
Sublingual space n (%)	8 (17.8)	9 (20.0)
Other spaces	10 (22.2)	9 (20.0)

Table-II: Comparison of baseline biological parameters at admission (n=90).

Biological parameter	Extraction group (n=45)	Non-extraction group (n=45)	p-value
WBC (cells/ μ L)	$12,840 \pm 2,310$	$12,610 \pm 2,450$	0.65
CRP (mg/L)	16.8 ± 4.2	16.4 ± 4.5	0.67
NLR	7.9 ± 2.1	7.7 ± 2.3	0.68
ESR (mm/hr.)	41.2 ± 8.6	40.7 ± 9.1	0.79

Table-III: Changes in inflammatory and hematological parameters from admission to day 3 after initiation of treatment (n=90)

Biological parameter	Extraction group (n=45) Admission	Extraction group (n=45) Day 3	p-value	Non-extraction group (n=45) Admission	Non-extraction group (n=45) Day 3	p-value
WBC (cells/ μ L)	12,840 $\pm$ 2,310	9,640 $\pm$ 1,980	<0.001*	12,610 $\pm$ 2,450	10,820 $\pm$ 2,050	<0.001*
CRP (mg/L)	16.8 $\pm$ 4.2	8.6 $\pm$ 3.1	<0.001*	16.4 $\pm$ 4.5	11.2 $\pm$ 3.5	<0.001*
NLR	7.9 $\pm$ 2.1	3.9 $\pm$ 1.4	<0.001*	7.7 $\pm$ 2.3	4.8 $\pm$ 1.6	<0.001*
ESR (mm/hr)	41.2 $\pm$ 8.6	24.8 $\pm$ 6.3	<0.001*	40.7 $\pm$ 9.1	29.6 $\pm$ 7.4	<0.001*

Values are presented as mean $\pm$ standard deviation.

p-values were calculated using paired sample t-test for comparison between admission and day 3 after initiation of treatment within each group.

*Statistically significant (p<0.05).

Table-IV: Comparison of inflammatory response and clinical outcomes between study groups (n=90).

Outcome Variables	Extraction group (n=45)	Non-extraction group (n=45)	p-value
Reduction in WBC count (cells/ μ L)	3200 $\pm$ 980	1790 $\pm$ 850	<0.001*
Reduction in CRP (mg/L)	8.2 $\pm$ 2.6	5.2 $\pm$ 2.3	<0.001*
Reduction in NLR	4.0 $\pm$ 1.3	2.9 $\pm$ 1.2	<0.001*
Reduction in ESR (mm/hr)	16.4 $\pm$ 5.1	11.1 $\pm$ 4.7	<0.001*
Hospital stay (days), mean $\pm$ SD	3.9 $\pm$ 1.2	5.0 $\pm$ 1.5	<0.001*
Positive bacterial culture, n (%)	38 (84.4)	36 (80.0)	0.58
Mixed aerobic and anaerobic growth, n (%)	21 (46.7)	19 (42.2)	0.67
Resolution of local infection signs at discharge, n (%)	42 (93.3)	37 (82.2)	0.12

*Statistically significant.

DISCUSSION

This prospective observational cohort study aimed at assessing the association of extraction of the causative tooth on biological inflammatory parameters in patients with odontogenic infections. The results showed that tooth extraction during the acute phase of infection resulted in a larger decrease in inflammatory markers, such as C-reactive protein (CRP), the neutrophil-to-lymphocyte ratio (NLR), white blood cell count (WBC) and erythrocyte sedimentation rate (ESR) compared to conservative tooth retention and that there was a shorter hospital stay for the tooth extraction group. Inflammatory biomarkers have been emerging as valuable objective markers for evaluating the severity of the disease, treatment response and prognosis in odontogenic infections. Malhotra *et al* [11]. showed that inflammatory biomarkers can be useful monitoring tools in a fascial space infection of odontogenic origin, which plays an important role in assessing the course of the infection and the efficacy of treatment. In line with these findings, the inflammatory parameters in our study were significantly reduced after intervention: the largest reduction was seen in patients having the responsible tooth extracted. Extraction of the primary focus of infection might lead to more rapid resolution of systemic inflammatory activation, as evidenced by the marked decrease in both CRP and NLR following extraction [11].

Extension of the infection and subsequent host inflammatory responses play a significant role in the severity of odontogenic infections [12-13]. In our

cohort, patients that were taken to the dentist for extraction had a shorter hospital stay and had improved inflammatory markers when compared to those not taken to dentist for extraction. Similarly, Bond *et al.* reported that patients with deep odontogenic infections had an increased risk of sepsis and had to be recognized and treated appropriately in a timely fashion [14]. There are several clinical parameters, however, that can affect hospital stay – these include baseline infection severity, need for drainage procedures, tooth restorability, the extent of any surgical procedures, and the effectiveness of antibiotic treatment. So, the observed difference in hospital stay must be considered to be a part of the association and not due to extraction alone.

These findings are in line with the results of other studies on inflammatory markers in odontogenic infections that have shown a reduction in CRP after extraction. Chausheva *et al* [15] showed that inflammatory biomarkers such as procalcitonin, CRP and hematological parameters are significantly different in odontogenic abscess depending on bacterial etiology. In the present study, CRP showed a significant reduction from baseline to Day 3 after initiation of treatment in the extraction group compared to the non-extraction group, indicating its ability as an early clinical response indicator. Likewise, NLR has been shown to be a practical and cost-effective inflammatory marker in oral and maxillofacial infections.

Microbiological features of odontogenic infections also play a role in the treatment of such infections. In

the current study culture positivity rates were similar across groups although oftentimes mixed bacterial growth occurred, which confirms the polymicrobial nature of odontogenic infections. Ahde *et al.* [16], emphasized that odontogenic abscesses that require repeated health care visits are characterized by complex clinical features and may need a comprehensive management strategy in addition to initial antimicrobial therapy. Source control, drainage and specific antimicrobial therapy continue to be elements of successful therapy.

In the recent years, with new advances in access to health care and alteration in the microbial pattern, the need for optimized management strategies has been even more highlighted. Nijakowski *et al.* [17] noted changes in clinical features and clinical risk factors for odontogenic abscesses during the pandemic, indicating that perhaps longer time intervals for presentation may increase disease complexity. While in the present study, early surgical intervention with extraction of the infected tooth has been associated with better biological outcome, in cases where the tooth is deemed as non-restorable, early definitive treatment should be performed.

The bacterial spectrum of odontogenic infections has been found to be increasingly complicated with the improvements in microbiological identification techniques. Thol *et al.* [18] indicated that next generation sequencing offered better characterization of odontogenic abscess-associated microbial communities with diverse bacterial population and resistance patterns that were not identified by traditional culture methods. A more recent study by Warwas *et al.* [19] indicated that better pathogen identification by state-of-the-art molecular methods might impact therapeutic choice and clinical outcome. Molecular microbiological analysis was not conducted in the present study, but future research should include molecular microbiology to gain a better understanding of treatment response in terms of the pathogen involved.

The main strength of this study lay in the prospective cohort design with uniform clinical management and serial measurement of inflammatory biomarkers prior and following intervention. The presence of several biological parameters, such as CRP and NLR, enabled to assess the treatment response widely. Also, assessment of hospital stays and clinical recovery parameters yielded clinically relevant outcome measures. In selected odontogenic infections, future multicenter randomized controlled trials are suggested to substantiate the superiority of

early extraction of causative teeth in certain cases. Extended follow-up times should be used to assess recurrence rates, quality of life outcomes and long-term dental outcomes. Use of advanced microbiological techniques such as molecular sequencing may enhance pathogen identification and personalized antimicrobial strategies. Further, the creation of clinical algorithms based on inflammatory markers like CRP and NLR could help clinicians track the progress and response to treatment.

LIMITATIONS

Several limitations should be considered. First, this was a non-randomized observational cohort study and the allocation of treatment was not random, but was determined by clinical assessment of the restorability of the teeth. So, the patients chosen for extraction might have had more extensive tooth damage or more acute local infection, potentially introducing confounding by indication. Even though baseline inflammatory parameters were similar between groups, there may be residual selection bias. Second, the study was performed in one tertiary care hospital, and the findings might not apply to other health care institutions. Third, follow-up was restricted to the early postoperative period and long-term outcomes, such as recurrence, delayed complications, and functional dental outcomes were not evaluated. Lastly, Microbiological culture results were recorded when available; however, detailed organism identification and antimicrobial susceptibility patterns were not analyzed as primary outcomes in this study.

CONCLUSION

A larger decrease in inflammatory markers and a longer hospital stay was seen in teeth that were retained during acute odontogenic infection than in those that were extracted. Patients undergoing extraction of the causative tooth showed greater reductions in inflammatory markers and shorter hospital stays. Biological markers such as CRP and NLR can support clinical judgement to give objective information about the extent of inflammatory activity in the body, severity of disease and the response to treatment.

CONFLICT OF INTEREST

None

GRANT SUPPORT & FINANCIAL DISCLOSURE

Declared none

Disclosure

None

AUTHORS CONTRIBUTION

Shaheen Memon: Study conception and design, data acquisition, manuscript drafting, final approval, responsible for every aspect of the research

Muhammad Shahzad: Study conception and design, supervision, critical revision for important intellectual content, final approval, responsible for every aspect of the research

Sundas Kunbher: Data acquisition, data management, manuscript drafting, final approval, responsible for every aspect of the research

Ishrat Begum: Analysis and interpretation of data, critical review of the manuscript, final approval, responsible for every aspect of the research

Ram Pershad Chooni Lal: Literature review, analysis and interpretation of data, critical review of the manuscript, final approval, responsible for every aspect of the research

REFERENCES

1. Kang ES, Lee JH. Diagnostic value of presepsin in odontogenic infection: A retrospective study. *Maxillofac Plast Reconstr Surg*. 2022; 44(1): 18. DOI: <https://doi.org/10.1186/s40902-022-00353-7>
2. 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; 22(1): 931. DOI: <https://doi.org/10.1186/s12879-022-07934-x>
3. Hammad Y, Namineni S, Flores-Mir C, Aminoshariae A, Kulild JC. Admission C-reactive protein, white blood cell count, glucose, and body temperature in severe odontogenic infections: A retrospective study using severity scores. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2022; 133(6): 639-42. DOI: <https://doi.org/10.1016/j.oooo.2021.11.002>
4. Bègue L, Schlund M, Raoul G, Ferri J, Lauwers L, Nicot R. Biological factors predicting the length of hospital stay in odontogenic cellulitis. *J Stomatol Oral Maxillofac Surg*. 2022;123(3):303-308. DOI: <https://doi.org/10.1016/j.jormas.2021.07.007>
5. Vetró É, Vlocskó M, Piffkó J, Janovszky Á. Current approaches to the diagnosis and management of odontogenic abscesses. *Orv Hetil*. 2022; 163(37): 1455-63. DOI: <https://doi.org/10.1556/650.2022.32587>
6. Limardo A, Blanco L, Menéndez J, Ortega A. Ultrasound-guided drainage versus surgical drainage of deep neck space abscesses: a randomized controlled trial. *Acta Otorrinolaringol Esp (Engl Ed)*. 2022; 73(1): 4-10. DOI: <https://doi.org/10.1016/j.otoeng.2020.08.008>
7. Miller CS, Kryscio RJ, Doneen AL, Bale BF. Myeloperoxidase: a circulating marker of inflammation and tooth infection. *Am J Med*. 2023; 136(4): 390-3. DOI: <https://doi.org/10.1016/j.amjmed.2022.12.008>
8. Rosca O, Bumbu BA, Ancusa O, Talpos S, Urechescu H, Ursoniu S, *et al*. The role of C-reactive protein and neutrophil-to-lymphocyte ratio in predicting the severity of odontogenic infections in adult patients. *Medicina (Kaunas)*. 2023; 59(1): 20. DOI: <https://doi.org/10.3390/medicina59010020>
9. Słotwińska-Pawlaczyk A, Orzechowska-Wylęgała B, Latusek K, Roszkowska AM. Analysis of the clinical status and treatment of facial cellulitis of odontogenic origin in pediatric patients. *Int J Environ Res Public Health*. 2023; 20(6): 4874. DOI: <https://doi.org/10.3390/ijerph20064874>
10. Georgiou AC, van der Waal SV, Buijs MJ, Crielaard W, Zaura E, Brandt BW. Host-microbiome interactions in apical periodontitis: the endodontic microbiome in relation to circulatory immunologic markers. *Int Endod J*. 2023; 56(6): 748-64. DOI: <https://doi.org/10.1111/iej.13912>
11. Malhotra M, Kaur T, Kukreja S, Bhullar RS, Dhawan A, Sandhu A. To evaluate the efficacy of biomarkers as monitoring tools in patients with fascial space infections of odontogenic origin: a clinical study. *J Maxillofac Oral Surg*. 2024; 23(1): 159-66. DOI: <https://doi.org/10.1007/s12663-022-01722-5>
12. Méndez-Millán JA, Martín-González J, Jiménez-Sánchez MC, Segura-Egea JJ. Antibiotic over-prescription by dentists in the treatment of apical periodontitis: a systematic review and meta-analysis. *Antibiotics (Basel)*. 2024;13(4): 289. DOI: <https://doi.org/10.3390/antibiotics13040289>
13. Hellgren C, Hellgren J, Öhrnell Malekzadeh B. Deep neck space infections of odontogenic origin are costly and preventable. *Acta Odontol Scand*. 2024; 83: 469-74. DOI: <https://doi.org/10.2340/aos.v83.41382>
14. Bond J, Soubra YS, Aziz U, Read-Fuller AM, Reddy LV, Kesterke MJ, *et al*. Are deep odontogenic infections associated with an increased risk for sepsis? *J Oral Maxillofac Surg*. 2024; 82(7): 852-61. DOI: <https://doi.org/10.1016/j.joms.2024.03.028>
15. Chausheva GM, Yankov YG, Nenova DD. Impact of bacterial etiology on procalcitonin, C-reactive protein and hematological parameters in odontogenic versus non-odontogenic head and neck abscesses. *Cureus*. 2024; 16(9): e69352. DOI: <https://doi.org/10.7759/cureus.69352>
16. Ahde HR, Vilén ST, Uittamo J, Mäkitie A, Snäll J, Toivari M. Oropharyngeal abscesses with

- repeated health care visits: characteristics in patients with odontogenic and peritonsillar abscesses. *Head Face Med.* 2025; 21(1): 5.
DOI: <https://doi.org/10.1186/s13005-025-00480-2>.
17. Nijakowski K, Ksel S, Marushko O, Nowak A, Jankowski J, Kwiatkowski J, *et al.* Impact of the COVID-19 pandemic on odontogenic abscess clinical patterns and predictive factors: a retrospective cross-sectional study. *J Clin Med.* 2025; 14(19): 6953.
DOI: <https://doi.org/10.3390/jcm14196953>
18. Thol F, Warwas FB, Spuck N, Kramer FJ, Heim N. Microbial spectrum and resistance of odontogenic abscesses: microbiological analysis using next-generation sequencing. *Clin Oral Investig.* 2025; 29(1): 8.
DOI: <https://doi.org/10.1007/s00784-024-06097-0>
19. Warwas FB, Thol F, Sieber MA, Spuck N, Kramer FJ, Heim N. Next-generation sequencing improves pathogen identification in odontogenic abscesses, could this affect clinical outcomes? *Clin Oral Investig.* 2025; 29(9): 430.
DOI: <https://doi.org/10.1007/s00784-025-06504-0>.