Original Article

A Clinicobiochemical Study of C - Reactive Protein among Patients with Odontogenic Infections in a Nigerian Tertiary Hospital

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Abstract

Background: It appears that studies on the association between CRP levels and odontogenic infections are limited. The aim of this study is to determine the difference in CPR levels between the different types of odontogenic infections.

Methodology: All consecutive patients that were diagnosed and treated for dentoalveolar and fascial space infections of odontogenic origin that met the inclusion criteria were studied. The data collected were age, gender, tobacco use, alcohol intake, and drug abuse. Other collected data were pain, trismus, dysphagia, antibiotics abuse, pre-existing medical condition, pulse rate, blood pressure, respiratory rate, body temperature, white blood cell, type of odontogenic infection, type of treatment, length of hospital stay and C-reactive protein. All analysis were done using IBM SPSS version 21.0 (IBM Corp, New York, USA). P- Value less than 5% was considered statistically significant.

Results: A total of 44 patients with a mean age of 45.3±1.39 years ranging from 10 to 60 years were enrolled in this study. The C-reactive protein was significantly higher among patients with positive history of pain compared to those without pain (P = 0.01). The patients with fascial space infection had C-reactive protein levels higher than those with dentoalveolar infection and the difference in their means was statistically significant (P = 0.02). Likewise, the C-reactive protein was 17.5mg/dl significantly higher in the patients that stayed more than 5days in the hospital compared to those that stayed less than the same days(P = 0.03).

Conclusions: The total mean of C-reactive protein of 75.4±3.53 mg/dl was greater than the critical level while the 9.62 X 10^9/L of WBC counts was lower than that of the reference value. Patients that had pain, fascial space infection and stayed more than five days in the hospital had higher levels of C-reactive protein following odontogenic infections.

Keywords: Odontogenic infection; C-reactive protein; Fascial space infection.

Introduction

Odontogenic infections (OIs) are seen daily in oral and maxillofacial clinics worldwide[1-3]. They range from dentoalveolar infections to fascial space infections[4]. Uncontrolled dentoalveolar infections may spread to involve the head and neck fascial spaces which present as cellulitis or abscess. Presenting clinical features vary considerably from a local swelling to life-threatening complications like respiratory obstruction, necrotizing fasciitis, pericarditis, descending mediastinitis, artery rupture, brain abscess and sepsis[5]. Despite the use of antibiotics and advances in health care services,

Access this article online

Quick Response Code:

Website: www.nigerianmedjournal.org

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How to cite this article: Okoye GO, Saheeb BD, Egbor EP, Ekaniyere B E. A Clinicobiochemical Study of C - reactive protein among Patients with Odontogenic Infections in a Tertiary Hospital, Niger J Med 2022; 62(5):356-363

the course of the infection can be unpredictable and can lead to severe morbidity or even mortality[6]. Various local and systemic predisposing factors such as diabetes mellitus, immunosuppression and previous radiotherapy are known to increase the severity of these infection[7]. Severe odontogenic infection can cause prolonged hospital stay which ultimately lead to unfavorable outcome, and substantial financial cost[8].

It is paramount to assess the odontogenic infections as early as possible. The clinical signs may be delayed or not have enough information to provide an interpretation of the severity of an infection. The first stage of odontogenic infection begins with inoculation of the organism in the tissues which starts with the initial spread of the microbes into the soft tissues[9]. During the cellulitis stage the inflammatory process reaches a peak. The next stage is abscess formation, where there is a process of necrosis with clinical appearance of fluctuation. The final stage of odontogenic infection is the rupture of an abscess that occurs spontaneously or with therapeutic drainage[10]. The dentoalveolar infections are regarded as low grade odontogenic infection while the fascial space infection are graded as moderate and high grade depending on the proximity to vital structure or the airway[11].

Several attempts have been made to utilize CRP levels as an inflammatory prognostic factor in management of odontogenic infection. Its rapid rise and fall make it much more sensitive predictor than erythrocyte sedimentation rate (ESR) and white blood cell (WBC) count[12]. CRP has shown to be able to discriminate bacterial and viral infections and gauge of inflammatory response[13]. CRP is one of the acute phase protein synthesized in the liver in response to tissue damage. CRP was discovered in 1930 in a pneumococcal pneumonia patient[14]. It is synthesized in the liver and was initially thought to be a pathogenic secretion because it was found to be elevated in acute inflammatory conditions and so was described as an acute phase protein.. It has a range of 0.1 – 10 mg/dl in normal healthy individuals[15]. It is involved in the process of innate immune system such as complement activation, antigen clearance and mediation of phagocytosis by activation of neutrophils[16]. Production of CRP is triggered by pro-inflammatory cytokines called interleukins (IL) such as IL-6, IL-1 and tumor necrosis factor (TNF)-alpha secreted by macrophages and adipocytes in response to microbial infections[17].

Though the several studies have reported the correlation between CPR levels with length of hospital stay[18,19], severity of odontogenic infections[20,21], type of treatment[22] and WBC counts[23], it appears that there is no such report between CPR levels and types of odontogenic infections. The purpose of this study is to determine the difference in CPR levels between the different types of odontogenic infection in patients seen in a tertiary hospital.

Materials and Method

Ethical approval (ADM/E22/VOL.V11/1181) was obtained from the Research and Ethics Committee of the hospital and written informed consents were also obtained from the patients before the commencement of the study. The design was a prospective cohort study which was conducted at the Department of Oral and Maxillofacial Surgery, University of Benin Teaching Hospital, Benin-City, from June 2016 to September 2017. All the patients were treated on an in-patient basis and were observed by the same surgeon. All consecutive patients that were diagnosed and treated for dentoalveolar or fascial space infections of odontogenic origin that met the inclusion criteria were studied. Odontogenic infections that did not spread to the fascial spaces were considered as dentoalveolar infections while those that spread to the fascial spaces were considered as fascial space infections[24]. Excluded from the study were patients with oral and maxillofacial infections that were not odontogenic in origin. Also excluded, were patients with history of chronic illnesses e.g chronic renal failure, chronic liver disease, and those with blood dyscrasias. Those with malaria in the course of study were also excluded. Using sample size formula of $n = Z^2 p q / d^2$ and a prevalence of 11.3% from a previous study[25], the sample size was 44 patients.

The data collected were age, gender, tobacco use, alcohol intake, and drug abuse. Other collected data were pain, trismus, dysphagia, antibiotics abuse, pre-existing medical condition, pulse rate, blood pressure, respiratory rate, body temperature, white

blood cell, type of odontogenic infection, type of treatment, length of hospital stay and C-reactive protein. Blood pressure was measured using a mercury sphygmomanometer (Model: Accoson; Made in England, 2006) and a stethoscope (Model: Littmans; Made in England 1995). Temperature readings were taken with a mercury glass thermometer (Ogotex C-scale type; Model- CRW 23 A) placed in the patient's axilla for 5 minutes. Pulse rate was obtained by palpating the radial artery at the wrist of the patient for 60 seconds using a chronologic wrist watch. Respiratory rate was obtained by visual examination of the patient's chest movement (inspiration and expiration) for 60 seconds using a chronologic wrist watch. Pain, trismus, and dysphagia were assessed as present or absent. Routine laboratory investigations were done along with airway assessment. At the onset of treatment, samples were obtained from infection sites and sent for culture and sensitivity. Blood samples of all the patients were taken to evaluate WBC and CRP levels before starting any treatment. The assessment of level of CRP was repeated on the 5th day of treatment. The pain, trismus, dysphagia, pulserate, blood pressure, respiratory rate, body temperature and, white blood cell count were assessed preoperatively. Empirical antibiotic of intravenous amoxicillin with clavulanic acid 1.2 g 12hourly and 500mg metronidazole 8 hourly were given to prevent infection. Appropriate analgesics were also given for pain relief using intramuscular diclofenac sodium 75 mg 12hourly

Infection was said to be present if CRP level exceeds 11mg/dl. In the descriptive analysis, the categorical data was summarized in frequency and percentages while the continuous data was summarized in means and standard deviations. In the inferential statistics, the differences between means were tested for statistical significant with independent t-tests. All analysis were done using IBM SPSS version 21.0 (IBM Corp, New York, USA). P- Value less than 5% was considered statistically significant.

Results

A total of 44 patients with a mean age of 45.3±1.39 years ranging from 10 to 60 years were studied. Table 1 shows the demographic characteristics of the patients. A greater (63.6%) proportion of the patients were in age group lesser than 45 years. Thirty-two (52.3%) of the patients studied were females. There were only 7(15.9%) patients that gave positive history of tobacco use. Less than half (34.1%) of the patients gave positive history of alcohol intake and same (47.7%) with those that abused drugs.

Table 2 shows the clinical characteristics of the patients. Pain was present in majority (72.7%) of the patients. Only 7(15.9%) of the patients had trismus but majority (79.5%) of them had dysphagia. More than half (54.5%) of the subjects reported a positive history of antibiotic abuse. Immunologically compromising conditions were seen in only 4(9.10%) patients. Though the increased pulse rate was seen in more than half (61.4%) of the patients, the elevation of blood pressure was seen in less than half (25.0%) of the patients. Respiratory rate was increased in 13(29.5%) patients. Almost (93.2%) of all the subjects had increased body temperature. Incidentally, less than half (22.7%) of the patients had increased white blood cell counts. Thirty-three (75.0%) patients presented with fascial space infection while the remaining patients had dentoalveolar infections. The most (47.7%) common modality of treatments was incision and drainage with extraction and intravenous (IV) antibiotics while the least (25.0%) given treatment was extraction and IV antibiotics alone. Interestingly, more than half (56.8%) of the patients with fascial space infection stayed more than 5 days in the hospital.

The total means of C-reactive protein and white blood cells counts following odontogenic infection was 75.4±3.53 mg/dl and 9.62 X 10^9/L respectively Table 3 shows the clinicobiochemical characteristics of patients according to the levels of C-reactive proteins in odontogenic infection. The C-reactive protein was significantly higher among patients with positive history of pain compared to those without pain (P = 0.01). The patients with fascial space infection had C-reactive protein levels higher than those with dentoalveolar infection and the difference in their means was statistically significant (P = 0.02). Likewise, the C-reactive protein was 17.5mg/dl significantly higher in the patients that stayed more than 5days in the hospital compared to those that stayed less than same days(P = 0.03).

Table 1: The demographic characteristics of the patients (n = 44)

Variables	Category	Frequency	Percentage
Age (years)	< 45	28	63.6
		16	36.4
gender	Male	32	52.3
	Female	21	47.7
Tobacco use	Yes	7	15.9
	No	37	80.1
Alcohol intake	yes	15	34.1
	No	29	65.9
Drug abuse	Yes	21	47.7
	No	23	52.3

Table 2: The clinical characteristics of the patients (n = 44)

Variables	Category	Frequency (n)	Percentage (%)
Pain	Present	32	72.7
	Absent	12	27.3
Trismus	Present	07	15.9
	Absent	37	84.1
Dysphagia	Present	09	20.5
	Absent	35	79.5
Pretreatment antibiotics usage	Yes	24	54.5
	No	20	45.5
Pre-existing medical condition	Yes	04	9.1
	No	40	90.9
Increase pulse rate	Present	27	61.4
	Absent	17	38.6
Increase blood pressure	Present	11	25.0
	Absent	33	75.0
Increase respiratory rate	Present	13	29.5
	Absent	31	70.0
Increase body temperature	Present	41	93.2
	Absent	03	6.80
Increased white blood cell	Present	10	22.7
	Absent	34	77.3
Type of odontogenic infection	Dentoalveolar infections	11	25.0
	Fascial space infections	33	75.0
Type of treatment	Antibiotic only	00	0.00
	Extraction + Antibiotics	11	25.0
	I & D + Antibiotics	12	27.3
	I & D + Extraction + Antibiotic	21	47.7
Length of hospital stay(days)	0	11	25.0
	1-5	25	56.8
	>5	08	18.2

Table 3: The clinicobiochemical characteristics of patients according to the levels of C-reactive proteins in odontogenic infection (n = 44)

Variables	Category	M± SD	MD	95%C.I	P-Value
Age	<45	76.4±3.39	4.80	-18.7-7.6	0.70
		71.9±3.94			
Gender	Male	81.3±2.96	12.3	-9.25 -33.6	0.26
	Female	69.0±4.04			
Tobacco use	Yes	80.2±3.43			
	No	74.5±3.59	5.70	-23.9 -35.3	0.70
Alcohol intake	Yes	84.8±3.11			
	No	70.4±3.68	14.4	-8.26 -36.7	0.21
Drug abuse	Yes	83.8±3.09			
	No	67.7±3.79	16.1	-5.15 -36.5	0.13
Pain	Present	89.6±2.84			
	Absent	74.5±3.84	15.1	-30.9 -18.7	0.01*
Trismus	Present	81.2±3.22			
	Absent	74.3±3.62	6.90	-22.8 -36.5	0.64
Dysphagia	Present	87.3±2.32			
	Absent	72.3±3.74	15.0	-11.6 -41.5	0.26
Antibiotics abuse	Yes	72.1±3.73			
	No	78.7±3.38	6.60	-28.2 -15.1	0.55
Pre-existing medical condition	Yes	86.3±3.75			
	No	70.8±3.39	15.5	-7.89 -38.7	0.19
Increase pulse rate	Present	72.1±3.79			
	Absent	80.7±3.09	8.60	-13.5-30.8	0.44
Increase blood pressure	Present	62.3±4.28			
	Absent	79.7±3.20	17.4	-7.10-41.9	0.16
Increase respiratory rate	Present	75.7±3.74			
	Absent	83.7±3.14	8.00	-5.70 - 50.1	0.52
Increase body temperature	Present	75.0±3.56			
	Absent	80.7±3.59	5.70	-48.8 - 37.3	0.79
Increased white blood cell	Present	79.3±4.54			
	Absent	74.4±3.39	4.90	-22.0 - 31.8	0.71
Type of odontogenic infection	Dentoalveolar infections	62.6±3.57			
	Fascial space infections	87.1±3.13	24.5	-4.06 - 44.0	0.02*
Type of treatment	I & D + Antibiotics	85.4±2.15			
	I & D + Extraction Antibiotic	79.2±3.42	6.20	-1.78 - 32.7	0.85
	1-5	72.4±1.72			
Length of hospital stay(days)	>5	89.9±3.77	17.5	-11.1 - 44.3	0.03*

*Significant at P < 0.05; M= Mean of C-reactive protein; SD = Standard deviation of the mean; MD = Mean difference.

Discussion

Odontogenic infection is a frequent and global public health burden and its severity range from mild to rapidly fatal type if inappropriately managed[26]. They are seen daily in oral and maxillofacial clinics worldwide[1]. The infections could involve the dentoalveolar region that could develop into dentoalveolar infections which could range from gingival abscess to periapical abscess. These types of odontogenic infection are mild and reversible if the causative factor is promptly removed. However, they could spread to the fascial spaces to cause the life-threatening odontogenic infections called fascial space infections. The role of C-reactive protein as an inflammatory marker in odontogenic infections is well studied globally[12,13].

The acute phase response is a complex set of

systemic and metabolic reactions elicited by infections or other causes of injury[14]. Besides other physiological, metabolic and biochemical changes, the acute phase response is characterized by alterations in the hepatic synthesis and serum levels of some proteins. Thus, while the levels of positive acute phase proteins like C-reactive protein, complement 3, serum amyloid A, alpha-1 and glycoprotein etc. increase due to stimulation of hepatic synthesis , depression of hepatic production of visceral transport proteins(negative acute proteins) like albumin , transferring , thyroxin binding prealbumin (TBPA) and retinol binding proteins(RBP) etc. occurs[15] .Close linkage of the severity and duration of acute infection turns C-reactive protein into a highly sensitive marker for inflammatory processes. Erythrocyte sedimentation rate (ESR) and white blood cell count (WBC) are exhaustively investigated indicators for inflammation but without the sensitivity and the temporal accuracy which is provided by C-reactive protein[16]

In this study, 44 patients were studied and there was slight male predominance over the female patients. Previous reports [5,8] had it that more males are affected with odontogenic infections but other studies reported otherwise[3,6]. A greater proportion of the patients were in the age group less than 45 years. Though contrary findings[2,4] were reported in the literature, similar findings[3,8] were also reported. The most probable reason for predominance of odontogenic infections in this age group could be related to higher proportion of epithelial rest cell in this group of population[11].

The total mean of C-reactive protein was 75.4 mg/dl which indicates the presence of infection since it is higher than the reference value of 11mg/dl. While higher value than 75.4mg/dl was reported in some studies[18,23] others [20,21] reported a lesser value following odontogenic infections. It was observed in this study that there was a significant increase in C-reactive protein levels in all the patients at presentation. Similar increase in C-reactive protein levels were reported in earlier studies[18-23,] Patients with fascial space infections were found to have higher C-reactive protein levels compared to those with localized dentoalveolar infections. This finding agreed with the works of Ylijoki *et al*[23] and Sharma *et al*[21] that linked the severity of the odontogenic infection to the C-reactive protein level. Bagul *et al*[27] also wrote that the increase in C-reactive protein level in response to inflammation could vary from as little as 50% to as much as 10,000 fold if the inflammation was profound. Heim *etal*[28]also reported an increase as high as 50,000 fold of the C-reactive protein in patients with severe odontogenic infection who had comorbid conditions and further stated that an increase of about 1,000 fold was a significant indicator of odontogenic infection.

The C-reactive protein was significantly higher among patients with positive history of pain compared to those without pain. The clinical signs and symptoms of odontogenic infections studied were pain, dysphagia, and trismus but only those in pain had a significant increase in C-reactive protein. This could be related to pain mediators that also stimulate the hepatocytes to release the acute-phase protein as pain is reported as one of the cardinal symptoms of an acute inflammatory process[29]. As no previous study relate pain to levels of C-reactive protein, comparison of this finding could not be possible.

In this study, the patients with fascial space infection had C-reactive protein levels higher than those with dentoalveolar infection and the difference in their means was statistically significant. This finding could be related to the fact that fascial space infection is more severe than dentoalveolar infection, however no previous findings for comparison. Odontogenic infections usually arise from invasion of bacteria into the pulp of the tooth through carious lesions; cracks in the enamel, fractures of the crown and root of the tooth; through the periodontal pocket and open restoration margins[30]. Bacterial products migrate to the tooth apices through the root canal or accessory canal resulting in apical periodontitis.[6] As this progresses unabated, the infection at the tooth apex causes inflammatory cells and osteoclasts to migrate to the site causing inflammatory reaction which results in bone resorption and pus formation (suppuration).[7] Local factors such as orientation of the tooth and its root, thickness of the surrounding bone, the muscles and fascia may influence the progression and direction of spread of the infection. Systemic factors

such as diabetes mellitus, alcoholism, malnutrition, acquired immunodeficiency syndrome (AIDS), cancer chemotherapy and organ transplant may predispose the patient to rapid spread in the tissue planes resulting in cellulitis or where these factors are absent, the infection could remain confined to the bone and surrounding tissues as localized abscess which may drain to the surface via a sinus tract.[8]

The mean length of stay varies worldwide in patients with odontogenic infection. In previous studies[26,,31-33,] a mean range of between 3.69 to 8.27 days was reported as the approximate range worldwide. In this study, the mean length of stay was 3.0±1.77 days with an actual range of 1 to 8 days. This was in contrast to Osunde *et al*[3] who reported a longer mean length of stay of 10.7± 8.6 days. Odontogenic infections are common and they account for substantial proportion of maxillofacial hospital admissions. In these patients prolonged hospital stay is associated with an unfavorable outcome, and also incurs substantial financial costs to the patients and health services. In this study, the C-reactive protein was 17.5mg/dl, which was significantly higher in the patients that stayed more than 5days in the hospital compared to those that stayed less than same days. Despite several works on odontogenic infection in Nigeria in the literature,[1, 3-6] none related the length of hospital stay to C-reactive protein. In this study, it was observed that there is a relationship between C-reactive protein levels, severity of the odontogenic infection and the length of stay. Patients with fascial space infections were observed to have longer length of hospital stay and higher C-reactive protein than those with localized infections at presentation. This was in agreement with the work of Stathopoulos *etal*[18]who reported that the severity of odontogenic infection is a predictor of length of hospital stay and C-reactive protein levels. It was also observed that patients who had fascial space infections also had greater decline in their postoperative C-reactive protein levels than those with localized infections. This was also consistent with Krpata *et al*[20] who concluded in their work that the length of stay was best predicted on the basis of location of the infection.

This study has some limitations. First, though statistically deduced, the sample size is small. Second, there were no or few studies for detailed comparison of the major findings. Third, this was a single centre study and variations in the parameters obtained may be seen in multi centre studies.

In conclusion, the total mean of C-reactive protein of 75.4±3.53 mg/dl obtained was greater than the critical level while the 9.62 X 10^9/L of WBC counts was lower than that of the reference value. Patients that had pain, fascial space infection and stayed more than five days in the hospital had higher levels of C-reactive protein following odontogenic infections. The assessment of C-reactive protein in relation to type of odontogenic infection will help to detect odontogenic infection early for optimum outcome following appropriate treatment planning.

Acknowledgment

We want to thank our colleagues for allowing us to recruit their patients as samples in this study**.**

References

1. Edetanlen B E, Saheeb BD. Comparison of outcomes in conservative versus surgical treatment for Ludwig's angina. *Med Princ Pract* 2018; **27**:362-366.
2. Flynn TR. Severe Odontogenic Infection. *J. Oral Maxillofac Surg* . 2006; **60**:72-77.
3. Osunde DO, Akhiwu BI, Efunkoya AA, Adebola AR, Iyogun CA, Arotiba JT. Management of fascial space infections in a Nigerian teaching Hospital: A 4 year review. *Nig Med J* 2012; **53**:12-15.
4. Akinbami BO, Akadiri O, Gbujie DC. Spread of odontogenic infection in Port-Harcourt, Nigeria. *J Oral Maxillofac surg.* 2000; **68**: 2472-7.
5. Osunde DO, Akhiwu BI, Efunkoya AA, Adebola AR, Iyogun CA, Arotiba JT. Management of fascial space infections in a Nigerian teaching Hospital: A 4 year review. *Nig Med J* 2012; **53**:12-15.
6. Ndukwe KC, Okeke IN, Akinwande JA, Aboderin AO, Lamikanra A. Bacteriology and antimicrobial susceptibility profile of agents of orofacial infections in Nigerians: *Afr J Clin Exp Microbiol* **5**: 272-277.

7. Keswani ES, Venkateshwar G. Keswani ES, et al. J Odontogenic Maxillofacial Space Infections: A 5-Year Retrospective Review in Navi Mumbai. *Maxillofac Oral Surg*. 2019; **18**:345-353.
8. Parara E, Krasadakis C, Toursounidis I, Tsekoura K, Mourouzis C, Rallis G. Parara E, et al.Significant rise in neck infections progressing to descending necrotizing mediastinitis during the COVID-19 pandemic quarantine. *J Craniomaxillofac Surg*. 2021; **49**:1182-1186.
9. Zirk M, Zoeller JE, Peters F, Ringendahl L, Buller J, Kreppel M. Zirk M, et al.Cefazolin versus ampicillin/sulbactam as an empiric antibiosis in severe odontogenic neck infection descending from the lower jaw-retrospective analysis of 350 cases. *Clin Oral Investig*. 2021; **25**:563-570.
10. Abbot PV. Assessing restored teeth with pulp and periapical disease for the presence of cracks, caries and marginal breakdown. *Aust Dent J* 2004; **49**: 33-39.
11. Adewale RA, Ugwumba C, Ogunbajo VO, Odebode AP. Ludwig's Angina: an audit of 14 cases in 2 tertiary hospital in Lagos, Nigeria. *Nig J Oral Maxillofac Surg*. 2015; **2**: 11-18.
12. Lee Y, Lim J, Choi SW, Han S, Park B, Youm JY. Lee Y, et al. Changes of Biomarkers before and after Antibiotic Treatment in Spinal Infection. *Korean J Neurotrauma*. 2019; **15**:143-149.
13. Drazic R, Jurisic M, Markovic A, Colic S, Gacic B, Stojcev-Stajcic L. C-reactive protein as an inflammatory marker in monitoring therapy effectiveness of acute odontogenic infections. *J. Clin. Invest* 2011; **139**: 446-45.
14. Vajpayee N, Graham SS, Bem S. Erythrocyte sedimentation rate. In: McPherson RA, Pincus MR. eds. Henry's Clinical Diagnosis and Management by Laboratory Methods. 22nd ed. *Philadelphia Pa Elsevier/Saunders*; 2011; 519-522
15. Sproston NR, Ashworth JJ. Role of C-reactive protein at sites of inflammation and infection. Front. *Immunol*. 2018; **9**:754
16. Bali R, Sharma P, Ghanghas PO, Gupta N, Tiwari JD. Singh A. To compare the efficacy of C- reactive protein and total white blood cell count as markers for monitoring the course of odontogenic infection. *H Maxillofac Oral Surg.* 2017; **16**: 322-7.
17. Gershov D, Kim S, Brot N, Elkon KB. C-reactive protein binds to apoptotic cells, protect the cells from assembly of the terminal complement components and sustain an anti-inflammatory innate immune response: implications for systemic autoimmunity. *J Exp Med* 2000; **192**: 1353-1363.
18. Stathopoulos P, Igoumenakis D, Shuttleworth J, Smith W, Ameerally P. Predictive factors of hospital stay in patients with odontogenic maxillofacial infections: the role of C-reactive protein. *Br J Oral Maxillofac Surg*. 2017; **55**:367-370.
19. Heim N , Wiedemeyer V, Reich R, Martini M. The role of C-reactive protein and white blood cell count in the prediction of length of stay in hospital and severity of odontogenic abscess *J Craniomaxillofac Surg* 2018; **46**:2220-2226.
20. Krpata DM, Keller DS, Samia H, Lawrence J, Obokhare I, Marderstein E, Brady KM, Delaney CP.Evaluation of inflammatory markers as predictors of hospital stay and unplanned readmission after colorectal surgery. *Pol Przegl Chir*. 2013; **85**:198-203.
21. Sharma A, Giraddi G, Krishnan G, Shahi K. 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; **13**:1–9.
22. Prihandana A N, Yusuf HY, Nurwiadh A, Lismayanti L. Correlation of odontogenic infection severity score with C-reactive protein levels on the patient with odontogenic infection in Dr Hasan Sadikin General Hospital Bandung. *Padjadjaran Journal of Dentistry*. 2021; **33**: 19-25.
23. Ylijoki S, Suuronen R, Jousimies-Somer H, Meurman J, Lindquist C. Differences between patients with or without the need for intensive care due to severe odontogenic infection. *J Oral Maxillofac Surg.* 2001; **59**: 867-872
24. Stefanopoulos PK, Kolokotronis AE. The Clinical significance of anaerobic bacteria in acute odontogenic infections. *Oral Surg Oral Med Oral Pathol Oral RadiolEndod* 2004; **98**: 398-408.

25. Sanchez R, Mirada E, Arias J, Pano JR, Burgueno M. Severe Odontogenic infections: epidemiological, microbiological and therapeutic factors. *Med Oral Pathol Oral* 2011; **16**: 670-676.
26. Peter E, Fong B, Wormuth D, Soni S, Risk factors affecting hospital length of stay in patients with odontogenic maxillofacial infections. *J Oral Maxillofac Surg.* 1996; **54**:1386-1391.
27. Bagul R, Chandan S, Dilip Sane V, Patil S, Yadav D. Comparative evaluation of C-reactive protein and white blood cell count in fascial space infections of odontogenic origin. *J Maxillofac Oral Surg* 2017; **16**:238-42
28. Heim N, Berger M, Wiedemeyer V, Reich R, Martini M. Heim N, et al. A mathematical approach improves the predictability of length of hospitalization due to acute odontogenic infection: A retrospective investigation of 303 patients. *J Craniomaxillofac Surg.* 2019; **47**:334-340.
29. Alotaibi N, Cloutier L, Khaldoun E, Bois E, Chirat M, Salvan D. Alotaibi N, et al.Criteria for admission of odontogenic infections at high risk of deep neck space infection. *Eur Ann Otorhinolaryngol Head Neck Dis.* 2015; **132**:261-4.
30. Lewis MAI, Macfarlane TW, McGowan DA. A microbiology and clinical review of the acute dentoalveolar abscesses. *Br J Oral Maxillofac Surg*. 1990; **28**:359-366.
31. Wang J, Ahani A, Pogrel MA. A five year retrospectivestudyofodontogenic maxillofacial infections in a large urban public hospital. *Int J Oral Maxillofac Surg*. 2005; **34**: 646-649.
32. Thomas SJ, Atkinson C, Hughes C, Revington P, Ness AR. Is there an epidemic of admissions for surgical treatment of dental abscesses in the UK? *Br Med J* 2008; **336**:1219-1220.
33. Brennan MT, Runyon MS, Batts JJ. Odontogenic signs and symptoms as predictors of odontogenic infections: a clinical trial. *J Am Dent Assoc* 2006; **137**: 62-66.