C-REACTIVE PROTEINS AND ESR LEVELS IN PATIENTS WITH AND WITHOUT APICAL PERIODONTITIS. A CROSS-SECTIONAL STUDY

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Abstract

Objectives: *This research aimed to determine the relative levels of serum C-reactive Proteins (CRP)and Erythrocyte Sedimentation Rate (ESR) and compare them in patients with and without apical periodontitis by using Mann-Whitney U test and Spear man correlation coefficient between CRP level, ESR and CAL values.*

Materials & Methods: *This analytical, cross-sectional study was conducted at Khyber College of Dentistry Peshawar with a sample of 150 subjects. Data were analyzed using SPSS version 20. And Significance level was (P<0.05).*

Results: *The mean CRP level in patients with apical periodontitis was higher than healthy control, but this was statistically insignificant (P>0.05). Similarly, the mean ESR level in patients with apical periodontitis was higher than healthy control, but this was statistically insignificant (P>0.05).*

Conclusion: *In subjects free from the systemic disease the CPR and ESR level does not differ among apical periodontitis versus non-periodontitis. This means the apical periodontitis in systemically healthy individuals remains localized and do not produce systemic inflammation.*

Key Words: *Apical periodontitis, inflammatory markers, C-reactive proteins, Erythrocyte Sedimentation Rate.*

Introduction

Periapical periodontitis (also called apical periodontitis, or peri-radicular periodontitis) may be acute or chronic inflammatory lesion around the root apices due to bacterial attack on the pulp of dentition[1]. Periodontitis is a sort of inflammatory pathology of tooth-supporting tissues and is caused by speciic bacteria. Aggressive destruction of alveolar bone and periodontal ligament (PDL) along with the formation of pocket and gingival recession is found in this disease. While gingivitis is the inflammation of gingival tissue but there is no loss of attachment apparatus[2]. Periodontitis having attachment loss greater than one millimeter has the prevalence of 3 to 47%, the prevalence of periodontitis with more than 2mm attachment loss is 9.4 to 24.5% and prevalence of periodontitis with more than 3mm attachment loss is 0 to 10.1%[3]. The response of the host to periodontal disease is through both innate and adaptive immunity. Though periodontitis is a chronic disease, there is an acute phase reaction in this morbidity which shows that it is systemic inflammation[4,5]. Periodontitis is multi factorial inflammatory pathology, and it has effects through mediators like CRP, interleukin-6 and tumour necrosis factor- α (TNF- α). The response of this sort of inflammation is either activated locally due to bacterial infection or systemically through

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toxins released by bacteria during the pathogenesis of periodontitis. The process of thromboembolic events and atherogenesis is modified by antigens of bacteria, endotoxins and inflammatory mediators, e.g. c-reactive protein, interleukin-6 and TNF- α[6].

The CRP is released after injury, periodontitis, hypoxia and trauma. The concentration of CRP shows variation in healthy people over time and increases as the age advances. This age-related increase in CRP may be due to the rising incidence of pathologic conditions at a subclinical level. Association have been reported between high CRP level and smoking, obesity, triglycerides, and diabetes[7]. In periodontal disease, there is an alteration in molecular and cellular components because of inflammatory nature[8].

The half-life of CRP in plasma is about 12 to 18 hours, and it remains constant under most of the circumstances. So the synthesis rate of CRP is the good determinant of the intensity of pathologic phenomena[9]. C-reactive protein quantified the acute phase reaction directly. The normal range of CRP in sound and healthy individuals is from 1mg/L to <10 mg/L[10].

Materials and Methods

Written approval for this research project was obtained from the hospital ethical committee. Participants were strictly selected by predefine inclu - sion criteria. The aim, details and possible risk and advantages were fully explained to each participant. A written informed consent was obtained from all participants.

The total sample size was calculated as 150 and subjects were selected from the Outpatient Department, Khyber College of Dentistry, Peshawar. Subjects were divided into two groups; one group containing patients with apical periodontitis (case) and other group containing healthy individuals (control). These subjects were categorized based on clinical attachment loss (CAL) and features of apical periodontitis into two equal groups of healthy (control) and apical periodontitis (case). The control group consisted of patients with CAL = 0 and none of the feature of apical periodontitis. The apical periodontitis group included patients with, CAL> 2 mm and features of apical periodontitis. The examination was done on four sides (mesial, distal, buccal and lingual) with CPITN (community periodontal index of treatment need) probe. The included subjects were sent to pathology laboratory of Khyber College of Dentistry, Peshawar, to determine the level of C-reactive proteins in the blood.

Results

A total of 150 participants selected among which 75 had apical periodontitis, and 75 were normal. Of the total sample, 89 (59.3%) were males. The mean age was 30.76±8.867 years. The age range was from 17 to 63 years. The mean clinical attachment loss (CAL) was 1.34±1.43 mm. The value of serum CRP was 4.76±5.67 mg/Land of ESR was 20.96±19.36 mm/hr. The details are shown in Table 4.1.

The most common tooth affected by apical periodontitis was mandibular 1st molar (n=27, 36%) followed by mandibular 2nd molar (n=27, 18.7%). The mandibular teeth were more affected than maxillary teeth. (Table 4.2)

In the case group, the most common cause of periapical periodontitis was dental caries. The causes of the periapical periodontitis were dental caries (n=62, 41.3%), filling (n=7, 4.7%), crown (n=3, 2%) and trauma (n=3, 2%). In patients affected by apical periodontitis, 71 (94.6%) cases had tenderness on percussion, 16 (10.7%) had extruded feeling of the affected tooth, 15 (10%) had sinus tract formation in and 64 (42.7%) had radiographic radiolucency.

The mean CRP level in patients with apical periodontitis was higher than healthy control, but this was statistically insignificant (P>0.05). The details of descriptive statistics are given in Table 4.3.

Similarly, the mean ESR level in patients with apical periodontitis was higher (24.2±2.3 mm/hr) than healthy control (17.61±15.36 mm/hr), but this was statistically insignificant (P>0.05). The mean difference of CRP level was 17.61±15.36 mm/hr. The details of statistics are given in Table 4.4.

Discussion

Apical periodontitis is an inflammatory lesion in the periodontal tissues that is caused mostly by bacterial elements derived from the infected root canal system of teeth. In non-treated teeth, apical periodontitis represents a defensive response to primary infection in a necrotic pulp[12].

In this study, we aimed to determine systemic

Table 4.1: Descriptive statistics for age, CAL, serum CRP and ESR

Variable	N	Minimum	Maximum	Mean±SD
Age (years)	150	17.00	63.00	30.70±8.86
Clinical Attachment Loss (CAL) (mm)	150	0.00	4.75	1.34±1.43
serum CRP level (mg/L)	150	2.00	48.00	4.76±5.67
ESR (mm/hr)	150	1.00	95.00	20.90±19.36

Table 4.2: Pattern of Teeth Afected by Apical Periodontitis

Tooth	Frequency	Percent (%)
Maxillary central incisor	02	2.70
Maxillary lateral incisor	01	1.30
Maxillary canine	02	2.70
Maxillary 1st premolar	05	6.70
Maxillary 2nd premolar	05	6.70
Maxillary 1st molar	02	2.70
Maxillary 2nd molar	05	6.70
Mandibular central incisor	02	2.70
Mandibular canine	01	1.30
Mandibular 1st premolar	04	5.30
Mandibular 2nd premolar	05	6.70
Mandibular 1st molar	27	36.0
Mandibular 2nd molar	14	18.70
Total	75	100.0

markers of inflammation (CRP & ESR) in patients with apical periodontitis and compared them to the healthy controls. Most of the previous studies are conducted on the CRP level and ESR level in patients with chronic periodontitis [6,7]. However, research has proven that the underlying connection between chronic periodontitis and systemic condition similar to that of apical periodontitis (AP) and general health. In many ways, microbiological features of chronic periodontitis are similar to endodontic infection[13].

In the present study, the most common tooth affected by apical periodontitis was mandibular 1st and 2nd molars. The mandibular teeth were more affected than maxillary teeth. The most common cause of apical periodontitis is dental caries and pulpal infection. In literature, various reasons for the differing caries susceptibilities have been proposed, such as different tooth surface morphology or different post-eruptive enamel maturation of the surfaces. The caries susceptibility of a tooth surface also varies over time[14]. The mandibular molars are erupting earlier than maxillary molars, so more susceptibility to dental caries. The second reason is the gravitational force on maxillary arch remove food deposit while accumulating in the mandibular arch which leads to dental caries[15].

In our study, the most common cause (n=62, 41.3%) of periapical periodontitis was dental caries. Periapical diseases are due to the direct or indirect contribution of oral bacteria. The causative agent is the degenerating pulp. A periapical lesion occurs within a region of apical periodontitis which cannot form by itself and is inflammatory in origin[16].

In the current study, the most common features of apical periodontitis were tenderness to percussion and periapical radio flucency on the radiograph. Tendearness to percussion and periapical radio fluency on the radiograph is because of endotoxins released by bacteria within the canals which leads to bone loss. Cardoso et al.[17] conducted a study to determine the correlation between the volume of bone loss due to apical periodontitis determined by CBCT analysis and endotoxin levels in the infected root canal. They

Table 4.3: Comparison of CRP level between Cases (n=75) and Controls (n=75)

Cases	Controls	Mean diference (mg/L)	P value*
Mean ±SD (mg/L)	Mean± SD (mg/L)		
5.06±6.10	4.46±5.23	0.6± 0.87	0.479

Mann-Whitney U test, P<0.05 signiicant level

Table 4.4: Comparison of ESR level between Cases (n=75) and Controls (n=75)

Cases	Controls	Mean diference	P value*
Mean ±SD (mm/hr)	Mean± SD (mm/hr)		
24.2±22.30	17.61±15.36	0.659± 6.94	0.213

Mann-Whitney U test, P<0.05 signficant level

reported that maximum cases affected by periodontitis had tenderness to percussion and periapical radio flucency.

In this study, the mean CRP level in patients with apical periodontitis was higher than healthy control, but this was statistically insignificant (P>0.05). The mean difference (0.6± 0.87 mg/l) of CRP level was low. Similar results for ESR were also found. These results show in healthy individuals the apical periodontitis inflammatory effects remains localized and do not spread systemically. In literature, to our knowledge, no study has been conducted on healthy individuals (free from systemic disease) having apical periodontitis to determine CRP and ESR level.

In contrast to our study, a meta-analysis titled "Can apical periodontitis modify systemic levels of inflammatory markers?" showed that though the evidence is limited but in apical periodontitis patients the CRP and other inflammatory level is high in blood showing that systemic immune response not confined to the localized lesion, potentially leading to increased systemic inflammation[18]. But the detailed inspection of that meta-analysis showed that they included studies having a most participant with a systemic illness like rheumatoid arthritis, hypertension etc. So these results may not be comparable to the current study as per se.

However some studies had been conducted to determine CPR and other inflammatory markers in patients with apical periodontitis and systemic illness like hypertension[19]. These authors reported that systemic levels of CRP, IL-6, and fibrinogen levels are affected by the presence of chronic apical periodontitis in hypertensive patients.

Conclusion

TA most common cause of apical periodontitis is dental caries. Tenderness to percussion and periapical radio fluency is common features of periapical periodontitis. In subjects free from the systemic disease the CPR and ESR level does not differ among apical periodontitis versus non-periodontitis. This means the apical periodontitis in systemically healthy individuals remains localized and do not produce systemic inflammation.

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