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# Determination of the Impact of Smoking on the Apical Region of Adequately Root-Filled Teeth in Indian Males with and Without Periodontal Disease: A Prospective Study

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## ABSTRACT

One of the most intriguing elements facing the dentistry scientific community is the potential link between apical periodontitis (AP), periodontal disease (PD), and the poor habit of smoking. The goal of this study was to determine the impact of smoking on the apical region of adequately root-filled teeth in Indian males with and without periodontal disease.

**Materials and methods:** A total of 75 patients were chosen, and their teeth were subdivided into six subgroups (G-a: light smokers without Periodontal involvement, G-b: heavy smokers without Periodontal involvement, G-c: non-smokers without Periodontal involvement, G-d: light smokers with Periodontal involvement, G-e: heavy smokers with Periodontal involvement, G-f: non-smokers with Periodontal involvement, G-f The condition of periodontal tissue in the apical region (AP) of root canal treated teeth was assessed using panoramic and periapical radiography.

**Results:** Only 89 teeth out of 1859 were found to have both coronal and endodontic restorations in place. The following were the AP rates: (G-a: 11.1 percent , G-b: 25 percent , G-c: 0 percent , G-d: 18.8 percent , G-e: 35.7 percent , G-f: 4.8 percent ). In terms of the AP, there was a statistically significant difference between heavy smokers' groups (G-b, G-e). Furthermore, there was a significant difference between heavy smokers with PD (G-e) and light smokers without PD (G-a) and non-smokers without PD (G-c), as well as a significant difference between heavy smokers without PD (G-b) and non-smokers with PD (G-c) (G-f). While there was no significant difference between non-smokers' groups (G-c, G-f) and light smokers' groups (G-a, G-b), there was a significant difference between non-smokers' groups (G-c, G-f) and light smokers' groups (G-a, G-d).

**Conclusion:** Smoking has a significant detrimental impact on the severity and prognosis of AP, which is exacerbated when AP is accompanied by lateral periodontitis.

**Keywords**: periapical index, smoking, apical periodontitis, root canal therapy

## I. INTRODUCTION

The infamous habit of smoking, as well as its damaging effects on human tissues, throws a shadow over numerous treatments and outcomes in dentistry and medicine. Endodontically treated teeth and their periodontal health are one of the treatments we'll look at in this study.[1]Clinical markers of a hygienic periodontium include the lack of cardinal symptoms of inflammation, gingival recession, and attachment loss. Long-term inflammation caused by many forms of subgingival bacteria has a deleterious impact on the periodontium, resulting in diseased periodontal tissue.[2]Nonetheless, the periodontal disease is affected by multiple etiological factors influence Parkinson's disease (PD), including age, gender, smoking, drugs, alcohol intake, and systemic disorders. The patient's behavior change and lifestyle improvement can help to eliminate or control PDs.[3]

The basic goal of endodontic treatment is to heal the damaged periapical tissue and eliminate inflammation. The clinical indications that are intended to indicate pulpal or periapical inflammation are still ambiguous because they are not highly specific to the disease.4 Because cross-

sectional and longitudinal studies[5] show that smoking has negative effects on periodontal bone and is a risk factor for periodontitis, it was assumed that it affects the apical periodontium of endodontically compromised teeth, worsening periapical bone destruction and increasing the size/and/or number of periapical lesions.[6-8] In general, perio-endo lesions have two major sources of infection: periodontal (where the tooth is still vital) or endodontic (where the tooth is usually nonvital). However, vitality (pulp testing) alone is not enough to rule out the causative factor, especially in multi-rooted teeth, so mobility, palpation, percussion, radiographs, and fistula tracking are used. True conjugation between the two sources occurs in a few number of cases, and these are known as true Perio-endo lesions.9 It is difficult to know the origin of the lesion ahead of time, so endodontic therapy should be delivered first and the tooth put under observation before we attempt periodontal therapy, because bacteria that reside in the empty spaces of root canals can transfer to the teeth in most cases.[10,11]

After the root canal has been properly sealed with filling material and sealant, the focus will shift to the periodontal status; if the lesion persists, it is of periodontal origin, and routine periodontal treatment will be administered. PDs, of which chronic periodontitis is the most common, have always been treated by disrupting microbial plaque aggregation on tooth surfaces. Even if there are actual Perio-endo lesions with both endodontic and periodontal causes of infection present at the same time, the treatment order will not change: endodontic first, periodontal second, etc.[12-14]

Apical periodontitis (AP) is an inflammatory condition that occurs at the root's apex and is caused by microbial infection of the pulp area. As a result, it's classified as a bacterial infection.15 Because the AP is complex and linked to a number of risk factors, the source of which can be intra-canal or extra-canal, it should be considered a potential Perio-endo lesion. The quality of endodontic fillings and coronal restoration are two risk factors for developing AP in endodontically treated teeth.16 In Europe, the prevalence of AP is as high as (34– percent) of individuals and (2.8–4.2 percent) of teeth.[17,18] The goal of this study was to see how smoking behaviors affect the apical area status of adequate root-filled teeth in Indian men with and without periodontal disease.

## II. MATERIALS AND METHODS

This study uses a cross-sectional observational methodology. Patients between the ages of 18 and 45 who reported to the dental college in North India in 2019 were enlisted at random. They were clinically and radiographically assessed after their complete medical, dental, and endodontic histories were collected. Ethical Issues The study was conducted in conformity with the principles of the Helsinki Declaration. All of the patients read the study's information and signed a letter of written consent for participation in research and data publication.

Criteria for Inclusion
1. With RCT, you can save at least one tooth.
2. Patients who are men.
3. RCT and coronal restoration teeth were fitted.

Criteria for Exclusion
1. To counteract the effects of hormonal fluctuations in female patients.
2. Patients with systemic diseases, such as cardiovascular disease (CVD), diabetes mellitus (DM), chronic liver disease, blood problems, and osteoporosis.
3. Patients having fewer than eight teeth.
4. The teeth of the third molar.
5. Patients who are mentally retarded.
6. Patients under the age of 18 or over 45 years old, due to age-related deterioration of oral hygiene, where plaque accumulation causes microbe introduction through the periodontium, resulting in PD.[20,21]
7. RCT-unfitted teeth (over or under obturation) (using x-ray).
8. RCT-fitted teeth with a poor coronal repair (using x-ray).

**Configuration of Patient Groups**

The total number of patients having a history of root canal therapy (RCT) was initially 205, but after applying historical and clinical exclusion criteria, we were able to narrow it down to only 155. After a panoramic X-ray was taken and radiographic exclusion criteria were applied, the final included sample was 75 patients, who were divided into three groups (each with 25 patients) based on their smoking habits:

1. Group 1 (G1): 25 light smokers (less than 10 cigarettes per day).[22]
2. Group 2 (G2): 25 heavy smokers (more than 10 cigarettes per day).
3. Group 3 (G3) consists of 25 non-smokers (Control).

The teeth in each group were then separated into two subgroups (those without PD and those with PD) as follows:

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1. Group a (G-a): Light smokers who do not have periodontal disease.
2. Group b (G-b): Heavy smokers who do not have periodontal disease.
3. Control group (G-c): No periodontal involvement.
4. Group d (G-d): Moderate smoker with periodontal disease.
5. Group e (G-e) consists of heavy smokers with periodontal disease.
6. Group f (G-f): Periodontal involvement with control.

RCT Teeth Presence

The first x-ray type taken to patients is panoramic radiography (OPG), which was taken by the "CarestreamR" instrument "model CS9000, USA" after getting a history of RCT from the patient. The OPG was used to display all teeth in a single x-ray, as well as to reduce the amount of time spent taking x-rays with intraoral periapical x-ray (PA x-ray) films in order to accomplish total mouth radiography (we need approximately 14 periapical x-rays at minimum to show all teeth while single OPG can give accepted information about RCT teeth). [23,24] The radiography was seen in a standardized manner, with all films being evaluated in the same amount of indoor illumination/light intensity (darkened room).

Radiographs were displayed on a computer screen with the light regulated using the "CarestreamR" software to achieve the best possible contrast.

**Quality of RCTs**

A panoramic radiograph was used to identify teeth with RCT and primary assessment,25 and the quality of the RCT was later re-assessed with a second x-ray, a PA x-ray, to reach a high degree evaluation since PA x-ray quality is superior to OPG quality.[23,26] Periapical x-ray taken with "CarestreamR RVG5200, USA" sensor and "CarestreamR" software. The parallel technique was applied, in which digital film holders were used to fine-tune the alignment of the film/tooth to eliminate picture elongation or shortening in all patients.

Only degree 1 (adequate root canal filling) was accepted in the study (where gutta-percha ends at 2mm away from the apex on x-ray film), but degrees 2 and 3 were afterwards excluded (degree 2: gutta-percha ends shorter than 2mm away from radiographical apex, degree 3: over-filling as the filling material extends beyond the radiographic apex). [25]

**Assessing the Coronary Fillings**

Clinical examination with a dental mirror, sickle dental probe, and afterwards OPG was performed to rule out the fitting of the coronal filling where the unified restoration contained it (overhang, open margin, recurrent caries, temporary filling or no filling).

A William's periodontal probe (Hu-Friedy, UK) was used to measure indices of (PI, GI, PPD, CAL) for the selected teeth, while the Nabers probe was also utilized to measure furcation involvement (Hu-Friedy, UK).

1. Probing pocket depth (PPD): From the base of the pocket to the free gingival border, the PPD was measured to the nearest mm. A tooth with a PPD of less than 4 mm was classified as having PD. [28]
2. Clinical attachment loss (CAL): From the junction (CEJ) to the base of the pocket, the CAL was calculated. A tooth with a CAL of less than 2 mm was considered to have PD. [28]
3. Gingival recession (GR): The GR was the measurement of the distance between the CEJ and the gingival margin. 29 (Grade I: 3mm, Grade II: 3–4mm, Grade III: >4mm) are the GR grades. Any tooth with one of the aforementioned grades was regarded as having PD.[30]
4. Posterior tooth furcation involvement (FI): The furcation degrees are (Degrees I: horizontal loss of periodontal tissue support less than 3mm, Degree II: horizontal loss of periodontal tissue support exceed- ing 3mm but not entirely passing the total width of furcation area, Degree III: horizontal through and through the destruction of periodontal tissue in the furcation). Any tooth with one of the degrees above is deemed to have PD.[31]
5. Tooth mobility (TM) is divided into three categories: 0.2–1 mm horizontal crown motion, higher than 1 mm horizontal crown motion, and horizontal and vertical crown motion. A tooth with degrees 2 and 3 is referred to as a PD tooth. 32

The severity of the following abnormalities was not taken into account, and once a tooth showed any of the PD signs, it was classified as a PD tooth in a dichotomous 1/0 manner.

**Periapical Tissue Evaluation**

The periapical state of each tooth was evaluated and measured using a PA x-ray and the Periapical Index (PAI). Grades 1–2 indicated healthy teeth, but grades 3–5 indicated the existence of AP, according to PAI. The poorest grade of all roots was taken in multi-rooted teeth. If there was any question, a higher grade was given. Orstavik et al. categorised the PAI index as follows in 1986: 25

(a) Grade 1 (Gr1): Periapical structures that are normal.

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(b) Grade 2 (Gr2): Minor periapical bone alterations or structural changes suggestive but not pathognomonic for AP.
(c) Grade 3 (Gr3): Periapical bone structural changes with some loss of mineral content characteristic of AP
(d) Grade 4 (Gr4): Periodontitis with a well-defined radiolucent region or well-defined radiolucency, periapical bone demineralization.
(e) Grade 5 (Gr5): Periapical bone demineralization, worsening periodontitis, or radiolucency with radiating expansions of bone structural alterations.

**The Examiners' Calibration**

The radiographs and periodontal characteristics were assessed by two highly skilled and trained observers with over 5 years of clinical experience (Inter-examiner agreement for scores of two observers).

**Statistical analysis**

In this study, descriptive statistics in the form of means were collected using the computer software IBM® SPSS® version 21. To compare groups in general, a one-way analysis of variance (ANOVA) test was utilized. In addition to the least significant difference (LSD) calculation that was used to compare the means of the six subgroups. Furthermore, P0.05 was accepted as the level of significance (S), P0.01 as highly significant (HS), and P0.05 as non-significant (NS).

## III. RESULTS

Our study included 75 patients who were separated into three groups of 25 patients each. The total number of teeth evaluated was 1859 (G1: 607, G2: 627, G3: 625), the number of missing teeth was 241 (G1: 93, G2: 73, G3: 75), and the number of RCT teeth was 235. (12.64 percent from all examined teeth) Table 1

**Table 1**
Prevalence of Variables in All Three Main Groups

| **Variable** | **No. (%)** | | | |
|---|---|---|---|---|
| **G1 (Light Smoker)** | **G2 (Heavy Smoker)** | **G3 (Control)** | **Total** | |
| Examined teeth | 607 (32.65%) | 627 (33.73%) | 625 (33.62%) | 1859 (100%) |
| Missing teeth | 93 (38.59%) | 73 (30.29%) | 75 (31.12%) | 241 (100%) |
| RCT teeth | 70 (29.79%) | 91 (38.72%) | 74 (31.49%) | 235 (100%) |
| Fitted RCT teeth | 27 (28.13%) | 35 (36.46%) | 34 (35.42%) | 96 (100%) |
| Under RCT teeth | 43 (32.82%) | 50 (38.17%) | 38 (29.01%) | 131 (100%) |
| Over RCT teeth | 0 (0.00%) | 6 (75.00%) | 2 (25.00%) | 8 (100%) |
| Fitted coronal restoration teeth | 25 (28.09%) | 32 (35.96%) | 32 (35.96%) | 89 (100%) |
| Unfitted coronal restoration teeth | 2 (28.57%) | 3 (42.86%) | 2 (28.57%) | 7 (100%) |
| Fitted restoration without periodontal involvement teeth | 9 (37.50%) | 4 (16.67%) | 11 (45.83%) | 24 (27%) |
| Fitted restoration with periodontal involvement teeth | 16 (24.62%) | 28 (43.08%) | 21 (32.31%) | 65 (73%) |

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| Variable | RCT of all examined teeth | Fitted RCT teeth of all RCT teeth | Fitted RCT, coronal restoration | | Fitted RCT, coronal restoration without periodontal involvement teeth | Fitted RCT, coronal restoration with periodontal involvement teeth |
|---|---|---|---|---|---|---|
| Of all fitted RCT teeth | Of all examined teeth | | | | | |
| No. (%) | 235 (12.64%) | 96 (40.85%) | 89 (92.71%) | 89 (4.79%) | 24 (26.97%) | 65 (73.03%) |

Only 96 teeth had fitting endodontic treatment (G1: 27, G2: 35, G3: 34), 131 teeth had under root canal teeth (G1: 43, G2: 50, G3: 38), and 8 teeth had an over endodontic filling among the RCT teeth (G1: 0, G2: 6, G3: 2) Table 1

There were 89 teeth with fitted coronal restorations (92.71 percent of all RCT teeth, 4.79 percent of all examined teeth) compared to 7 teeth with unfitted coronal restorations (G1: 25, G2: 32, G3: 32). (G1: 2, G2: 3, G3: 2) Table 1

In Table 1, we discovered that 24 teeth (26.97%) of fitted restorations had no periodontal involvement, while 65 teeth (73.03%) had periodontal involvement.

Table 2 shows that the highest rate of periodontal parameters (PPD, CAL, GR, FI, TM) was discovered in G2, the lowest rate of PPD and CAL was found in G1, the lowest rate of GR and TM was found in G3, and the lowest rate of FI was found in both G1 and G3.

Table 2
Percentages and No. of Periodontal Parameters in All Three Main Groups

| Groups | Periodontal Parameters | | | | | |
|---|---|---|---|---|---|---|
| PPD | CAL | GR | FI | TM | Total | |
| G1 Light smoker | 16 (28.1%) | 16 (7.7%) | 1 (24.6%) | 1 (16.7%) | 1 (25%) | 35 (24.1%) |
| G2 Heavy smoker | 21 (36.8%) | 28 (43.1%) | 9 (69.2%) | 4 (66.7%) | 3 (75%) | 65 (44.8%) |
| G3 Control | 20 (35.1%) | 21 (32.3%) | 3 (23.1%) | 1 (16.7%) | 0 (0%) | 45 (31%) |
| Total | 57 (100%) | 65 (100%) | 13 (100%) | 6 (100%) | 4 (100%) | 145 (100%) |

The lowest AP grade, Gr5, was not seen in all groups. In addition, the Grs4 was not found in all groups, with the exception of G-e and G-d, which had rates of (7.1 percent and 6.3 percent, respectively) as shown in Table 3.

In general, the rates of AP were (11.1 percent ) for G-a, (25 percent ) for G-b, (0 percent ) for G-c, (18.8 percent ) for G-d, (35.7 percent ) for G-e, (4.8 percent ) for G-f as shown in Table 3.

Table 3
Percentages and No. of Grades of PAI in All Subgroups (G-a, G-b, G-c, G-d, G-e, G-f)

| Groups | Grades | | | | | |
|---|---|---|---|---|---|---|
| | Healthy | | AP | | | |
| Gr1 | Gr2 | Gr3 | Gr4 | Gr5 | Total | |
| G-a Light smokers without Perio | 6 (66.7%) | 2 (22.2%) | 1 (11.1%) | 0 (0%) | 0 (0%) | 9 (100%) |
| (88.9%) | | (11.1%) | | | | |
| G-b Heavy smokers without Perio | 2 (50%) | 1 (25%) | 1 (25%) | 0 (0%) | 0 (0%) | 4 (100%) |
| (75%) | | (25%) | | | | |
| G-c Control without Perio | 10 (90.9%) | 1 (9.1%) | 0 (0%) | 0 (0%) | 0 (0%) | 11 (100%) |
| (100%) | | (0%) | | | | |
| G-d Light smokers with Perio | 10 | 3 (18.8%) | 2 (12.5%) | 1 (6.3%) | 0 (0%) | 16 (100%) |

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| | | | | | | |
|---|---|---|---|---|---|---|
| | (62.5%) | | | | | |
| (81.2%) | | (18.8%) | | | | |
| G-e Heavy smokers with Perio | 14 (50%) | 4 (14.3%) | 8 (28.6%) | 2 (7.1%) | 0 (0%) | 28 (100%) |
| (64.3%) | | (35.7%) | | | | |
| G-f Control with Perio | 17 (81%) | 3 (14.3%) | 1 (4.8%) | 0 (0%) | 0 (0%) | 21 (100%) |
| (95.2%) | | (4.8%) | | | | |
| Total | 59 (66.3%) | 14 (15.7%) | 13 (14.6%) | 3 (3.4%) | 0 (0%) | 89 (100%) |
| (82%) | | (18%) | | | | |

The Gr3 of AP did not appear in only one group which was the G-c group. Whereas the remaining groups showed different rates. The highest rates appeared in heavy smokers groups (G-e, G-b); (28.6 percent , 25 percent ) respectively, while the light smokers groups (G-d, G-a) showed lessor rates of Gr3 than heavy smokers groups; (12.5 percent , 11.1 percent ) respectively, while the least rate of Gr3 appeared in Gf (4.8 percent ) Table 3.

The G-c showed a healthy apical area, where only contain on Grs1 and 2; (90.9 percent , 9.1 percent ), respectively, Table 3.

The rates of Gr1 appeared in highest rate in non- smokers group (G-c, G-f); (90.9 percent , 81 percent ) and to lessor rate in light smokers groups (G-a, G-d); (66.7 percent , 62.5 percent ) respectively, while the least rate appeared in heavy smokers groups (G-b, G-e); (50 percent ) Table 3.

Moreover, the rates of Gr2 appeared in highest rate in smokers without periodontal involvement groups (G-b, G-a,); (25 percent , 22.2 percent ), and to lesser rate in periodontal involvement groups (G-d, G-e, G-f); (18.8 percent , 14.3 percent , 14.3 percent ) respectively, while the least rate of Gr2 was appeared in G-c (9.1 percent ) Table 3.

In Table 4, there is a (S) difference among groups according to the ANOVA test.

Table 4
ANOVA Test for Means of 6 Subgroups (G-a, G-b, G-c, G-d, G-e, G-f)

| | **Sum of Squares (SS)** | **df** | **Mean Squares (MS)** | **F-Test** | **P-value** | **Sig.** |
|---|---|---|---|---|---|---|
| Between Groups | 15.153 | 5 | 3.031 | 2.644 | 0.026 | S |
| Within Groups | 165.040 | 144 | 1.146 | | | |
| Total | 180.193 | 149 | | | | |

As shown in Table 5, the LSD test showed a (NS) difference between light smokers without PD (G-a) and othergroups (G-b, G-c, G-d, and G-f) except for a sole (S) difference between G-a and non-smoker without PD (G-c), Table 5. There was a (S) difference between heavy smokers without PD (G-b) and non-smoker with PD (G-f). Also, the same result obtained when compared the G-c with G-e as showed in Table 5.

Table 5
LSD of 6 Subgroups (G-a, G-b, G-c, G-d, G-e, G-f)

| **Groups** | | **Mean Difference** | **SE** | **P-value** | **Sig.** |
|---|---|---|---|---|---|
| Light smokers without Perio (G-a) | Heavy smokers without Perio (G-b) | 0.200 | 0.302 | 0.510 | NS |
| | Control without Perio (G-c) | −0.080 | 0.302 | 0.792 | NS |
| | Light smokers with Perio (G-d) | −0.280 | 0.302 | 0.357 | NS |
| | Heavy smokers with Perio (G-e) | −0.760 | 0.302 | 0.013 | S |
| Control with Perio (G-f) | −0.480 | 0.302 | 0.115 | NS | |

| Groups | | Mean Difference | SE | P-value | Sig. |
|---|---|---|---|---|---|
| Heavy smokers without Perio (G-b) | Control without Perio (G-c) | −0.280 | 0.302 | 0.357 | NS |
| | Light smokers with Perio (G-d) | −0.480 | 0.302 | 0.115 | NS |
| | Heavy smokers with Perio (G-e) | −0.960 | 0.302 | 0.002 | HS |
| Control with Perio (G-f) | −0.680 | 0.302 | 0.026 | S | |
| Control without Perio (G-c) | Light smokers with Perio (G-d) | −0.200 | 0.302 | 0.510 | NS |
| | Heavy smokers with Perio (G-e) | −0.680 | 0.302 | 0.026 | S |
| Control with Perio (G-f) | −0.400 | 0.302 | 0.189 | NS | |
| Light smokers with Perio (G-d) | Heavy smokers with Perio (G-e) | −0.480 | 0.302 | 0.115 | NS |
| Control with Perio (G-f) | −0.200 | 0.302 | 0.510 | NS | |
| Heavy smokers with Perio (G-e) | Control with Perio (G-f) | 0.280 | 0.302 | 0.357 | NS |

Whereas comparing between two heavy smokers' groups (G-b, G-e), the result showed (HS) difference Table 5.
While comparing of G-b with groups (G-c and G-d) the result gave (NS) difference, same result of the comparison between G-c with G-d, and too, when comparing the
G-d with groups (G-e, G-f), and also when comparing G-e with G-f as shown in Table 5.

## IV. DISCUSSION

In India, most of the researchers care about the estimation of the effect of smoking on the periodontium, but there is little information about the AP itself. However, there are no data on this topic in the Iraqi population. Our results came in agreement with other authors like (Ali et al 2013, Correia-Sousaa et al 2015, and Peršić Bukmir et al 2016) regarding smoking negative role in AP,33–35 alongside with other authors; knowing that smoking alters the microvas- culature and the function of immune system, the cadmium that is found in cigarettes also consumes the Super Oxide dismutase protective enzyme which is an anti-oxidant- in blood and saliva, by replacing the Manganese,

Although some authors (Bergström et al 2004, Bahammam 2012, Balto et al 2019) reached different results where the development of AP was irrelevant to smoking habit6,36,37 suggesting that the development of AP in RCT teeth is multifactorial.

Coronal restoration is the crucial factor since failure of the proper coronal seal might cause seepage of infection towards deeper points apically even if the endodontic treatment was performed carefully,[26] while other authors like

The endodontic treatment itself is multifactorial too because it passes through instrumentation, irrigation, and lastly obturation, unfortunately, we cannot trace the first two procedures because its operator-dependent and un-documentable, so we depended on the quality of obturation obtained from OPG's and later from PA X-ray to judge the quality of RCT.

Intentionally we only chose the endodontically properly filled teeth to neutralize the remaining bacterial infection in the canals factor and the insult caused by the excess/overfilling materials, this way we can observe the effect of smoking alone in the development of AP.

Assessment of periodontal condition included both radiographical and clinical measures, as AP cannot be seen with the naked eye, the PA x-ray proved to be a potent way for examination.

The periodontal apparatus is an inter-linked system, it even manifests itself radio-graphically as a continuous thin radiolucency surrounding the tooth, so it is not uncommon to witness a communication between the lateral and peri-apical periodontal tissue of smokers, inflammation and/or infection in one region can affect the other and vice-versa, this was apparent in the results as teeth of smokers with lateral

periodontal involvement (clinical attachment loss, active pocket) had AP too but this relationship was not significant in the absence of smoking; thus, there was a synergistic relationship between smoking and lateral periodontal involvement on the persistence of AP.

The location of these accessories and lateral canals are in coronal, middle, furcation and apical third, so if any inflammation to periodontium due to PD like loss of attachment or actively inflamed pocket or GR can lead to advancement and pushing of bacteria through these routes to pulp space, so the inflammation inside pulp was spread to all areas and finally reach to periodontal tissue in the apical region and result in AP.

The heavy smokers with periodontal involvement showed a higher rate of periodontal parameters than other groups due to the effect of smoking on periodontium as discussed in the past paragraph.

Not only that the cause of both AP and PD share the same agent (G – anaerobic bacteria) but the two diseases are involved in elevation cytokine levels as well as inflammatory mediators. Smoking, on the other hand, is not far from participating in developing AP, by affecting immunity and periodontal tissue integrity. It also disturbs the balance between the reactive oxidase species and its buffers; the anti- oxidants. Such a condition of imbalance will affect the lipids in membranes, intracellular proteins, and most importantly damage to DNA, leading inevitably into cellular death.[33]

## V. CONCLUSION

The results showed a negative, harmful effect of smoking with or without periodontal involvement on the apical status of endodontically treated teeth.

The combination of smoking and periodontal effects was higher than the smoking effect alone.

The continuous daily consumption of cigarettes for a long period of more than 5 years increases the possibility of AP especially RCT teeth.

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