



Journal of Contemporary Dental Sciences

Volume : 08, No : 02

July, 2020

Contents

Editorial

- * Asad-Uz-Zaman

Original Articles

- * **P-glycoprotein do not contribute to cisplatin sensitivity in oral squamous carcinoma cells**
Z Ahmed, AAY Khan, Y Deyama, Y Yoshimura, K Suzuki
- * **Effect of modified ridge lap and sanitary Pontic on periodontal health of abutment teeth of the three-unit posterior fixed partial denture**
S Akter, MM Rahman, MU Salma, K Nahar, MZ Habibullah, SK Nath, AE Noor, MTH Chowdhury
- * **Does irradiation affect the mechanical properties of dentine?**
AFMA Chowdhury, A Alam, MM Rahman, U Habiba, A Zaman, M Ahmed

Case Report

- * **Nonsurgical endodontic retreatment of an incisor with a large periapical lesion: management of an endodontic failure case**
N Jahan, AMG Muktedir, A Tabassum, S Shila, AAY Khan, AFMA Chowdhury
- * **Management of Amelogenesis Imperfacta- A case Report**
A Siddika, AA Mahmud, M Hossain



ISSN 2305-9664

Journal of Contemporary Dental Sciences (JCDS)

Recognized by Bangladesh Medical and Dental Council (BM&DC)

Vol. 8, No. 2, July 2020

**An Official Publication of Sapporo Dental College
Uttara, Dhaka**

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E-mail : sdch@bol-online.com, Web : www.sdch.edu.bd

Graphics Idea : Maruf Hussain, SDCH

Printed at

IDEA Printers, Katabon, Dhaka. 02-9662116

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2. Rashid M. Food and Nutrition. In Rashid KM, Rahman M, Haider S eds. Textbook of Community Medicine and Public Health. 4th edn. RHM Publishers 2004; 126-140
3. Sayeed MA, Hussain MZ, Banu A, Rumi MAK, Azad AK. Prevalence of diabetes in a suburban population of Bangladesh Diab Res Clin Prac 1997; 34: 149-155
4. Jarett RJ. Insulin and hypertension (Letter). Lancet 1987; 2: 748- 749
5. Banerji MA, Faridi N, Atulri R, Chiken RI, Lebovitz HE. Body composition, visceral fat, leptin and insulin resistance in Asian Indian men. J Clin Endocrinol Metab 1999; 84: 137-144 (Abstract)
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Editorial



Periodontal health and root caries prevention are the most important aspect of the health of abutment teeth in case of fixed partial denture. This issue includes one article by Akter et al. which concludes that sanitary pontic provide better results than the modified ridge lap pontic in terms of periodontal condition and health of abutment teeth in case of posterior three unit fixed partial denture.

Another two issues are related to common therapeutics like radiotherapy and chemotherapy, which are frequently used in case of any malignancy.

Chowdhury et al identified that Y-radiation therapy which is commonly used for head-and-neck cancer treatment significantly reduce dentine's hardness and elastic modules. Another study by Ahmed et al. identified that P-glycoprotein do not contribute to chemotherapy agent cisplatin sensitivity in cisplatin sensitive and resistant variant of oral squamous cell carcinoma cells.

The rest of this issue's article aim to showcase two interesting clinical cases and their management outcome. Among them one case report by Jahan et al. outlines that use of adequate biomechanical preparation with pure calcium hydroxide intracanal medicament can bring complete resolution of a large periapical lesion within 14 months. In another case report Siddika et al. reported that Multidisciplinary approach is essential for successful treatment of Amelogenesis imperfecta.

In conclusion, this issue covered some interesting article which prioritize in one hand the importance of conservative and appropriate treatment strategy for better outcome in our patient, on the other hand side effect of advance cancer treatment like radiation over dental hard tissues.

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Editorial

Page No.

* Asad-Uz-Zaman

v

Original Articles

- * **P-glycoprotein do not contribute to cisplatin sensitivity in oral squamous carcinoma cells**
Z Ahmed, AAY Khan, Y Deyama, Y Yoshimura, K Suzuki 1-8
- * **Effect of modified ridge lap and sanitary Pontic on periodontal health of abutment teeth of the three-unit posterior fixed partial denture**
S Akter, MM Rahman, MU Salma, K Nahar, MZ Habibullah, SK Nath, AE Noor, MTH Chowdhury 9-16
- * **Does irradiation affect the mechanical properties of dentine?**
AFMA Chowdhury, A Alam, MM Rahman, U Habiba, A Zaman, M Ahmed 21-21

Case Report

- * **Nonsurgical endodontic retreatment of an incisor with a large periapical lesion: management of an endodontic failure case**
N Jahan, AMG Mukhtadir, A Tabassum, S Shila, AAY Khan, AFMA Chowdhury 22-28
- * **Management of Amelogenesis Imperfecta- A case Report**
A Siddika, AA Mahmud, M Hossain 29-33

P-glycoprotein do not contribute to cisplatin sensitivity in oral squamous carcinoma cells

Z Ahmed ^{1,2}, AAY Khan ³, Y Deyama ¹, Y Yoshimura ¹, K Suzuki ¹

Abstract

Background: Reduced drug accumulation is a common alteration found in cisplatin-resistance cell lines. P-glycoprotein, a membrane-associated glycoprotein which affiliated with the ABC superfamily, strongly linked to the MDR to play a role in drug efflux to reduce the drugs therapeutic effect. **Purpose:** Therefore, this study was carried out to investigate the effect of P-glycoprotein in cisplatin accumulation/efflux in sensitive and resistant variant of oral squamous carcinoma cells. **Method:** The most cisplatin sensitive HSC-3 cells and most resistant BHY cells were used in this study. Assessment of cisplatin sensitivity was determined by measuring the ATP level of the cells. Intracellular cisplatin accumulation and cisplatin efflux study was conducted. Assessment of P-glycoprotein was done by western blotting. **Result:** The most cisplatin sensitive HSC-3 cells exhibited an increase intracellular cisplatin accumulation in comparison with most resistant BHY cells. But there were no such differences between the two in the cisplatin efflux level. Moreover, immunoblot analysis failed to detect P-glycoprotein in both the cells. **Conclusion:** P-glycoprotein do not contribute to cisplatin sensitivity in cisplatin sensitive and resistant variant of oral squamous carcinoma cells.

Key Words: P-glycoprotein; Cisplatin; Oral squamous cell carcinoma

(J Cont Dent Sci 2020;8(2): 1-8)

Introduction:

Cancer is one of the leading cause of death globally that is the second only to cardiovascular diseases such as ischemic heart disease and stroke¹. It is likely that cancer will become the primary cause of death worldwide as treatments for cardiovascular diseases continue to improve with modern medicine. The development and implementation of cancer chemotherapy, have significantly improved the quality of life and survival rates for many individuals with cancer. Although systemic chemotherapy can successfully eliminate early disease and help manage disease, improving patient prognosis and overall survival, incomplete elimination of all cancerous tissue can

occur alongside unwanted side effects due to off-target damage to healthy tissue.^{2,3}

Cisplatin [cis-diamminedichloroplatinum(II)], the frequently used and one of the most important chemotherapeutic agents in the treatment of different malignancy and solid tumors; especially testicular, ovarian, head, neck and lung cancers.⁴ An important tumor-killing mechanism of cisplatin is the binding of platinum compounds to DNA, forming Pt-DNA adducts,⁵ but the tumors often develop resistance to cisplatin reducing its efficacy.⁶ However, these resistance mechanisms are not fully known, but a common finding is that resistance tumor cells have lower accumulation of platinum than do sensitive parental cells.⁷ This could be caused by both decrease uptake and increased efflux of platinum, another possible explanation for resistance is binding of platinum to intracellular proteins.⁸⁻¹² One way to explore possible mechanisms is to genetically manipulate tumor cell lines to overexpress or underexpress proteins that could be involved in platinum resistance.

Once tumor cells are resistant to a single antitumor agent, the phenomenon of multidrug-resistance (MDR) confers upon cells the ability against many structurally unrelated antitumor agents.¹³⁻¹⁵

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Article Accepted: 01-05-2020

Article Received: 06-02-2020 ;

Hence, the ability of cancer cells to acquire MDR is a major challenge to successful chemotherapy in a wide variety of advanced malignancies. One known cause of MDR is the over-expression of the ATP-binding cassette (ABC) transporters on the membranes of cancer cells. ABC transporters mediate an energy-dependent efflux can significantly decrease the probability of successful treatment.¹⁶ Phospho-glycoprotein (P-glycoprotein) encoded by MDR1, a membrane-associated glycoprotein which affiliated with the ABC superfamily, strongly linked to the MDR to play a role in drug efflux to reduce the drugs therapeutic effect.¹⁷⁻¹⁹ Moreover, P-glycoprotein the ABC superfamily transporter is the main drug efflux transporters associated with chemotherapy failure in cancer.²⁰⁻²³

We have already established HSC-3 cells and BHY cells as cisplatin sensitive and cisplatin resistant respectively and reported that reduced intracellular cisplatin accumulation is the major cause behind this resistance. Based on the unique ability of P-glycoprotein as drug transporter and to determine the potential of P-glycoprotein as a biological marker for assessment of clinical outcome of chemotherapy, we were interested to initiate this study on the evaluation of P-glycoprotein expression in the cisplatin sensitive HSC-3 cells and cisplatin resistant BHY cells.

Materials and methods

Drugs, reagents and antibodies

Cisplatin, Tween 20 (Sigma, St Louis, MO); Trypsin-EDTA (GibcoBRL β , Life Technologies, Canada); Dulbecco's modified eagle's medium (D-MEM) (ICN Biomedicals Inc., Aurora, Ohio); Minimum essential medium eagle (MEM) (ICN Biomedicals Inc., Costa Mesa, CA); Fetal bovine serum (FBS) (HyClone, Perbio, Canada); Kanamycin sulfate (Meiji, Japan); antibodies against Na⁺, β +-ATPase (anti-Na⁺, β +-ATPase

β -1; Upstate Biotechnology, Lake Placid, NY); antibodies against Na⁺, β +-ATPase (anti-Na⁺, β +-ATPase β -1; Santa Cruz Biotechnology); antibodies against P-glycoprotein (P-Glyco CHECK C219TM ; Centocor, Inc., Malvern, PA); antibodies against ATP7A (ATPase alpha polypeptide Cu⁺⁺ transporting (ATP7A) polyclonal antibody; Orbigen Inc); Antibodies against ATP7B (ATPase alpha polypeptide Cu⁺⁺ transporting (ATP7B) polyclonal antibody; Orbigen Inc); Horseradish peroxidase (HRP)-conjugated rabbit anti-mouse IgG1, IgG2a, IgG (H+L), goat anti-rabbit IgG (H+L) (ZYMED, S. San Francisco, CA); All other drugs and chemicals were obtained from Wako Pure (Osaka, Japan).

Cells and culture conditions

Two different types of human oral squamous carcinoma cells were used in this study and were provided by the Japanese Cancer Research Resources Bank (JCRB, Tokyo, Japan). The cells were cisplatin-sensitive HSC-3 cells (squamous cell carcinoma of tongue, low metastatic type) and cisplatin-resistant BHY cells (epithelial like squamous cell carcinoma of mouth, non-metastatic but highly invasive to muscle and bone).

Cells were grown in a humidified incubator gassed with 5% CO₂ - 95% air at 37°C. The media used for both the cells were D-MEM supplemented with 1 mM L-glutamine, 4.5 g/l glucose, 3.7 g/l NaHCO₃, 66.5 mg/l kanamycin sulfate and 10% FBS. The cells were maintained in 100 x 20 mm tissue culture dish (Falcon β , Becton Dickinson Labware, Franklin Lakes, NJ) and media were renewed every second day. Subcultures were obtained by trypsin-EDTA treatment. Experiments were performed when the dishes became at least 80% confluent.

Assessment of cellular sensitivity to CDDP concentration

The cells were sub-cultured in 96 well plates (Falcon®, Becton Dickinson Labware, Franklin Lakes, NJ). After the cells reached confluence (at least 80%), the cells were treated with various concentrations of freshly prepared CDDP up to 1 mM dissolved in respected medium and incubated for 24 hours. Then sensitivity assay was performed by determination of the ATP (adenosine triphosphate) level of the cells, by luminometer using ViaLight™ Plus (Cambrex Bio Science Rockland, Inc., ME) according to the procedure recommended by the manufacturer. ATP values were expressed as percent (%) of controls. The average of three ATP values was expressed as a single ATP value and the values are the means \pm SD of the three independent experiments.

Time course assay of cellular sensitivity with higher dose 1 mM of CDDP

The cells were sub-cultured in 96 well plates. After the cells reached confluence (at least 80%), the cells were treated with 1 mM concentrations of freshly prepared CDDP dissolved in respected medium and incubated for various time frames up to 24 hours and the ATP level of the cells was measured following the same procedure as described above.

Microsome preparation:

The microsome of HSC-3 and BHY cells were prepared according to Jorgensen's method²⁴ with some modifications²⁵. Briefly, after the cells were reached into confluence they were allowed to grow for two more days. Then the media was removed and the cells were washed twice with 5 ml of 250 mM sucrose solution containing 1 mM tris-EDTA (pH 7.4). Then the cells were collected, by scraping the dishes into the same solution with a rubber

scraper. The cells were then centrifuged by 3,000 rpm for 5 minutes at 4°C. The supernatant was removed and the cells were suspended again on 250 mM sucrose solution containing 1 mM tris-EDTA (pH 7.4). Then the cells were ultrasonicated twice for 20 seconds and centrifuged at 7,000 rpm for 20 minutes at 4°C. The supernatant was transferred into fresh tubes and centrifuged at 18,000 rpm for 40 minutes at 4°C for separation of the microsomal fraction as a pellet. The pellet was homogenized with a Teflon glass homogenizer in 250 mM sucrose containing 1 mM tris-EDTA.

Measurement of protein concentration:

The protein concentration was estimated by using Bio-Rad Protein Assay (Bio-Rad Laboratories, CA) according to the manufacture's protocol with bovine serum albumin as a standard.

Western blot analysis:

The microsomes of HSC-3 cell and BHY cell were prepared and protein content was determined as describe earlier. Equal amounts of protein were boiled for 5 minutes and fractionated by 10% polyacrylamide gel electrophoresis. The electrophoretically separated proteins were then transferred into polyvinylidene difluoride membranes (Millipore corporation, MA) by electroblotting at 25 volt and 108 mA for 90 minutes using a Transbolt SD apparatus (Bio-Rad). The transferred membranes were blocked with blocking reagents (Immuno Block, DS Pharma Biomedical, Osaka, Japan) for 1 hour at room temperature. Membranes were then washed once for 15 minutes then thrice for 5 minutes each with TBST [50 mmol/l tris (pH 7.5), 150 mmol/l NaCl, and 1% tween 20] buffer at pH 7.5 at room temperature. The membranes were incubated overnight at 4°C with anti-P-glycoprotein antibodies.

The antibodies were diluted in the ratio of 1:1000. Followed by washing in TBST (as described earlier) and further incubated with HRP-conjugated rabbit anti-mouse IgG (H+L) for 1 hour at room temperature. After washing with TBST in the same way subsequent detection was carried out by Western Lightning Chemiluminescence Reagent Plus (Perkin Elmer, Boston, MA) according to manufacturer's instruction. Medical x-ray films were used to visualize the chemiluminescence. Kaleidoscope Prestained Standards (Bio-Rad) was used for molecular mass determination. The antibodies were diluted in immuno reaction enhancer solution (Can Get Signal™, Toyobo, Japan); solution 1 used for primary antibodies and solution 2 used for secondary antibodies. MES-SA/DX5 cell lysate (Santa Cruz Biotechnology) was used as a positive control for P-glycoprotein.

Results

Assessment of cellular sensitivity to CDDP concentration

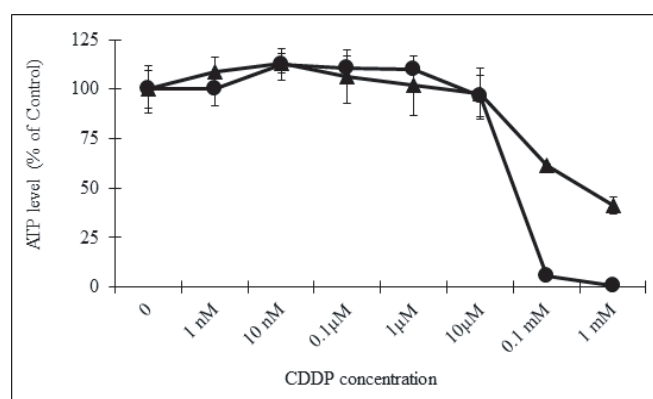


Fig. 1 Viability of HSC-3 & BHY cells 24 hours after cisplatin application concentration dependent

Cells were treated with different concentration of cisplatin for 24 hours and sensitivity assay was performed by determination of the ATP levels of the cells using luminometer. Cell used in this study are HSC-3 (μ) and BHY (μ). In

concentration dependent cisplatin sensitivity assay HSC-3 cells (μ) appeared as most cisplatin sensitive and BHY cells (μ) appeared as most resistant to cisplatin. Data were expressed as percent of control. The average of three ATP values was expressed as a single ATP value and the values are the mean \pm SD of the three independent experiments.

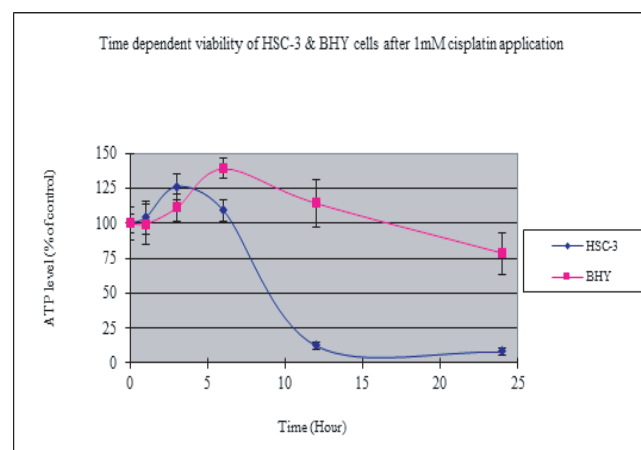


Fig. 2 Time dependent viability of HSC-3 cells & BHY cells after 1 mM cisplatin application

Cells were treated with 1 mM of cisplatin for different time frame and sensitivity assay was performed by determination of the ATP level of the cells using luminometer. HSC-3 cells (μ) and BHY cells (μ) were used. In time dependent cisplatin sensitivity assay HSC-3 cells (μ) appeared as cisplatin sensitive and BHY cells (μ) appeared resistant to cisplatin. Data were expressed as percent of control. The average of three ATP values was expressed as a single ATP value and the values are the mean \pm SD of the three independent experiments.

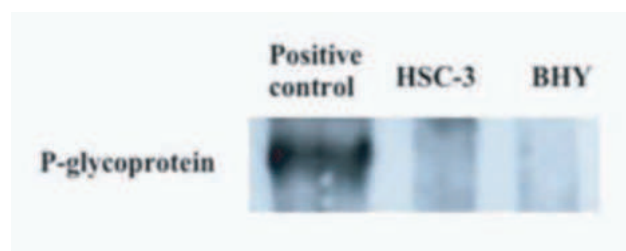


Fig. 3 Equal amounts of protein from HSC-3 and BHY cell microsome were subjected to

SDS-PAGE and analyzed by western blotting with antibodies specific for P-glycoprotein. Positive control (left), HSC-3 cell (middle) and BHY cell (right).

Figure 1. shows the effect of varying concentrations of cisplatin on cellular ATP content (% of control) of HSC-3 and BHY cells. Cells were treated with 0 to 1 mM final concentration of cisplatin and cellular ATP level was measured after 24 hours. Almost no reduction in cellular ATP content was observed with 0 to 10⁻⁶ M cisplatin. When the dose of cisplatin was increased from 100⁻⁶ M to 1 mM, the ATP level of all the cells started to decrease. But the amount and pattern of decreased ATP level varies among the cells. At the dose of 1 mM, HSC-3 cell appeared as most sensitive to cisplatin with about 99% decreased ATP level, while BHY cell appeared as most resistance to cisplatin with 59% decreased ATP level. These data demonstrate that ATP levels are starting to decrease by addition of 0.1 mM cisplatin, suggesting that cells treated with lower dose have no inhibitory effect while higher dose causes the inhibition.

Time course experiments with higher dose 1 mM of CDDP. In separate experiments all cells were harvested to determine the time-related changes after administration of 1 mM cisplatin. No inhibitory effect was observed in ATP levels within first 6 hours of exposure, instead some proliferative effect was observed. However, at 12 hours ATP levels were started to decrease and at 24 hours ATP levels were markedly reduced (Figure 2). This data also demonstrated that after time course experiments with higher dose (1 mM) of cisplatin, HSC-3 cell appeared as most sensitive and BHY cell appeared as most resistant against cisplatin.

Western blot

Expression of P-glycoprotein was assessed in the microsomes of HSC-3 and BHY cells (Figure 3). The immunoblot analysis failed to detect the expression of P-glycoprotein in both the cells.

Discussion

There have been many studies on the mechanism of tumor cells resistance to cisplatin and the decreased intracellular accumulation of cisplatin has been reported in a large number of them. However, the molecular mechanisms that underlie cisplatin resistance are not well understood yet. In the present study, at first we examined the cellular sensitivity to cisplatin in HSC-3 cells and BHY cells. After determining the cellular sensitivity, we assessed the functions of drug uptake and efflux in cisplatin sensitivity using an integrated and quantitative approach. In HSC-3 cells and BHY cells, we measured and compared intracellular cisplatin accumulation, efflux, and expression of P-glycoprotein. Concentration and time dependent profiles of intracellular platinum represent the result of uptake and efflux processes that take place simultaneously. This study showed that, in figure 1 following 24 hour exposure to different concentrations of cisplatin, the intracellular accumulation of platinum was significantly lower in the cisplatin-resistant BHY cells than in the cisplatin-sensitive HSC-3 cells. On the other hand, efflux studies did not reveal such obvious differences between them. Therefore, we concluded that alteration in cisplatin uptake primarily accounted for the differences in the intracellular platinum concentrations between cisplatin-sensitive and -resistant cells. Other investigators also reported decreased uptake and lower intracellular platinum concentrations in cisplatin-resistant cells.²⁶⁻²⁸ Most of the authors found a decreased uptake without changes in efflux,²⁹⁻³¹ but the opposite

situation, i.e., no difference in uptake but increased efflux, was also reported.³²

For understanding the mechanisms leading to alter intracellular platinum concentrations, it is crucial to reveal the functions of transport proteins. Few authors have been reviewed extensively the mechanism of P-glycoprotein mediated drug resistance.^{33,34} In various cancer types, such as acute myeloid leukemia, various childhood tumors and loco-regionally advanced breast cancer, overexpression of MDR1/P-glycoprotein has been found^{35, 36}. A direct relationship has thus been established between MDR1/P-glycoprotein expression and chemo-sensitivity in hematologic malignancies³⁵ and solid tumors,^{36,37} implicating P-glycoprotein mediated multidrug resistance as an important component of clinical drug resistance. Several author also reported down regulation of MDR1/P-glycoprotein in cisplatin sensitive cells,³⁸ moreover, many cisplatin sensitive cells down regulated MDR1/P-glycoprotein,³⁹ furthermore, several in vitro studies showed that MDR1/P-glycoprotein is involved in some drug resistance, but not in cisplatin. Here in this study immunoblot analysis failed to detect the expression of P-glycoprotein in both the cells.

Na⁺,K⁺-ATPase plays an important role in active transport through the cell membrane and in supporting physical ion balances.^{40,41} In addition, its enzyme activity is reported to be essential for the intracellular accumulation of cisplatin except for the passive transport. Kishimoto et al.⁴² reported the role of Na⁺,K⁺-ATPase alpha-1 subunit in intracellular accumulation of cisplatin. Andrews et al.⁴³ pointed out the significance of a cisplatin transport system being dependent on the function of Na⁺,K⁺-ATPase. Furthermore, many studies have confirmed the importance of a membrane potential maintained by Na⁺,K⁺-ATPase in cisplatin accumulation.^{30, 44, 45}

We previously reported⁴⁶ that cisplatin-sensitive HSC-3 cells possessed higher specific activity of Na⁺,K⁺-ATPase than cisplatin-resistant BHY cells and that the expression levels of both alpha and beta-subunits were also higher in cisplatin-sensitive HSC-3 cells than in the -resistant BHY cells. Moreover, pretreatment with ouabain markedly inhibited intracellular cisplatin accumulation. This inhibition of cisplatin accumulation by ouabain is an intriguing observation. The inhibition of cisplatin accumulation by ouabain indicates either that cisplatin accumulation is dependent upon the electro-chemical gradient across plasma membranes, or that cisplatin is transported by Na⁺,K⁺-ATPase which ouabain inhibits. In conclusion, the results obtained in our cell system are consistent with the interpretation that impairment of intracellular drug accumulation is a common event in the development of cellular cisplatin resistance and is regulated by its Na⁺,K⁺-ATPase activity. Moreover, drug efflux transporter P-glycoprotein had a marginal or no role in intercellular cisplatin accumulation.

References

1. Mathers C, Stevens G, Hogan D, Mahanani WR, Ho J. (2017) Global and regional causes of death: patterns and trends, 2000-15. In: Jamison DT, Gelband H, Horton S, Jha P, Laxminarayan R et al (eds) Disease control priorities: improving health and reducing poverty. Washington (DC)
2. Schirrmacher V (2019) From chemotherapy to biological therapy: a review of novel concepts to reduce the side effects of systemic cancer treatment (Review). *Int J Oncol* 2009; 54(2): 407-419
3. Schaue D, McBride WH (2015) Opportunities and challenges of radiotherapy for treating cancer. *Nat Rev Clin Oncol* 2015; 12(9): 527-540
4. Hanigan MH and Devarajan P. Cisplatin nephrotoxicity: molecular mechanisms. *Cancer Therapy* 2003; 1: 47 - 61

5. Wang D and Lippard SJ. Cellular processing of platinum anticancer drugs. *Nat Rev Drug Discov* 2005; Apr 4(4): 307 - 320
6. Siddik ZH. Cisplatin: Mode of cytotoxic action and molecular basis of resistance. *Oncogene* 2003; 22: 7265 - 7279
7. Andrews PA, Howell SB. Cellular pharmacology of cisplatin: Perspective on mechanism of acquired resistance. *Cancer Cell* 1990; 2: 35 - 43
8. Komatsu M, Sumizawa T, Mutoh M, Chen ZS, Chen K et al. Copper-transporting P-type adenosine triphosphatase (ATP7B) is associated with cisplatin resistance. *Cancer Res* 2000; 60: 1312 - 1316
9. Lin XJ, Okuda T, Holzer A, Howell SB. The copper transporter CTR1 regulates cisplatin uptake in *Saccharomyces cerevisiae*. *Mol Pharmacol* 2002; 62: 1154 -1159
10. Ishida S, Lee J, Thiele DJ, Herskowitz I. Uptake of the anticancer drug cisplatin mediated by the copper transporter Ctr1 in yeast and mammals. *Proc Natl Acad Sci USA* 2002; 99: 14298 - 14302
11. Katano K, Safaei R, Samimi G, Holzer A, Rochdi M, Howell SB. The copper export pump ATP7B modulates the cellular pharmacology of carboplatin in ovarian carcinoma cells. *Mol Pharmacol* 2003; 64: 466 - 473
12. Safaei R and Howell SB. Copper transporters regulate the cellular pharmacology and sensitivity to Pt drugs. *Crit Rev Oncol Hematol* 2005; 53: 13 - 23
13. Moscow JA, Morrow CS, Cowan KH. Multidrug resistance. *Cancer Chemother Biol Response Modif*. 1992; 13: 91-114.
14. Pastan I, Gottesman MM. Multidrug Resistance. *Journal of the National Cancer Institute*. 1991; 42: 277-86.
15. Gottesman MM, Fojo T, Bates SE. Multidrug resistance in cancer: role of ATP-dependent transporters. *Nature Reviews Cancer* 2002; 2: 48-58
16. Zhang JT. Use of arrays to investigate the contribution of ATP-binding cassette transporters to drug resistance in cancer chemotherapy and prediction of chemosensitivity. *Cell Research* 2007; 17: 311-23
17. Chen Z, Shi T, Zhang L, Zhu P, Deng M, Huang C, Hu T, Jiang L, Li J. Mammalian drug efflux transporters of the ATP binding cassette (ABC) family in multidrug resistance: A review of the past decade. *Cancer Letters* 2016; 370: 153-64.
18. Pluchino KM, Hall MD, Goldsborough AS, Callaghan R, Gottesman MM. Collateral sensitivity as a strategy against cancer multidrug resistance. *Drug Resist Update* 2012; 15: 98-105.
19. Alakhova DY, Kabanov AV. Pluronic and MDR reversal: an update. *Mol Pharm* 2014; 11: 2566-78.
20. K-nig J, M-ller F, Fromm MF. Transporters and drug-drug interactions: important determinants of drug disposition and effects. *Pharmacol Rev* 2013; 65: 944-66.
21. Levatić J, Ćurak J, Kralj M, Šmuc T, Osmak M, Supek F. Accurate models for P-gp drug recognition induced from a cancer cell line cytotoxicity screen. *J Med Chem* 2013; 56: 5691-708.
22. Shuai ZL, Zhang W, Yin X, Xing S, Xie Q, Cao Z, Zhao B. Binding cassette (ABC) transporters conferring multi-drug resistance. *Anticancer Agents Med Chem* 2015; 15: 423-32.
23. Juliano RL, Ling V. A surface glycoprotein modulating drug permeability in Chinese hamster ovary cell mutants *Biochim. Biochim Biophys Acta*. 1976; 455: 152-62.
24. Jorgensen PL. Purification of Na⁺, K⁺-ATPase: enzyme sources, preparative problems, and preparation from mammalian kidney. *Methods Enzymol* 1998; 156: 29-43.
25. Vasallo PM, Post RL. Calcium ion as a probe of the monovalent cation center of sodium, potassium ATPase. *J Biol Chem* 1986; 261: 16957-16962
18. Ahmed Z, Deyama Y, Yoshimura Y, Suzuki K. Cisplatin inhibits Na⁺,K⁺-ATPase activity depending on its concentration, preincubation time and temperature. *Hokkaido J Dent Sci* (in press)
26. Mellish KJ, Kelland LR, Harrap KR. In vitro platinum drug chemosensitivity of human cervical squamous cell carcinoma cell lines with intrinsic and acquired resistance to cisplatin. *Br J Cancer* 1993; 68: 240-250
27. Johnson SW, Perez RP, Godwin AK, Yeung AT, Handel LM, Ozols RF et al. Role of platinum-DNA adduct formation and removal in cisplatin resistance in human ovarian cancer cell lines. *Biochem Pharmacol* 1994; 47: 689-697
28. Zhengdong L, Duhong B. Experimental study on the mechanism of cisplatin resistance and its reversion in human ovarian cancer. *Chin Med J* 1995; 109: 353-355
29. Andrews PA, Velury S, Mann SC, Howell SB. Cis-diamminedichloroplatinum(II) accumulation in sensitive and resistant human ovarian carcinoma cells. *Cancer Res* 1988; 48: 68-73
30. Loh SY, Mistry P, Kelland LR, Abel G, Harrap KR. Reduced drug accumulation is a major mechanism of acquired resistance to cisplatin in a human ovarian cell line: circumvention studies using novel platinum(II) and (IV) ammine/amine complexes. *Br J Cancer* 1992; 66: 1109-1115

31. Zisowsky J, Koegel S, Leyers S, Devarakonda K, Kassack MU, Osmak M, Jaehde U. Relevance of drug uptake and efflux for cisplatin sensitivity of tumor cells. *Biochem Pharmacol* 2007; 73: 298-307
32. Paeker RJ, Eastman A, Bostick-Bruton F, Reed E. Acquired cisplatin resistance in human ovarian cancer cells is associated with enhanced repair of cisplatin-DNA lesions and reduced drug accumulation. *J Clin Invest* 1991; 87: 772-777
33. Gottesman MM and Pastan I. Biochemistry of multidrug resistance mediated by the multidrug transporter. *Ann Rev Biochem* 1993; 62: 385 - 427
34. Biedler JL. Drug resistance: genotype versus phenotype. Thirty-second G.H.A. Clowes Memorial Award Lecture. *Cancer Res* 1994; 54: 666-678
35. Marie JP. P-glycoprotein in adult hematologic malignancies. *Hematol Oncol Clin North Amer* 1995; 9: 243 - 250
36. Leighton JC and Goldstein LJ. P-glycoprotein in adult solid tumors: expression and prognostic significance. *Hematol Oncol Clin North Amer* 1995; 9: 251 - 274
37. Verrelle P, Meissonner F, Fonck Y, Feillel V et al. Clinical relevance of immunohistochemical detection of multidrug resistance P-glycoprotein in breast carcinoma. *J Nat Cancer Inst* 1991; 83: 111-116
38. Liu XY, Liu SP, Jiang J, Zhang X, Zhang T. Inhibition of the JNK signaling pathway increases sensitivity of hepatocellular carcinoma cells to cisplatin by down-regulating expression of P-glycoprotein. *European Review for Medical and Pharmacological Sciences* 2016; 20: 1098 - 1108
39. Cheng Q, Liao M, Hu H, Li H, Wu L. Asiatic Acid (AA) Sensitizes Multidrug Resistant Human Lung Adenocarcinoma A549/DDP Cells to Cisplatin (DDP) via Downregulation of P-Glycoprotein (MDR1) and Its Targets. *Cell Physiol Biochem* 2018; 47: 279-292
40. Lingrel JB, Kuntzweiler T. Na⁺,K⁺ -ATPase. *J Biol Chem* 1994; 269: 19659-19662
41. Rose AM, Valdes R Jr. Understanding the sodium pump and its relevance to disease. *Clin Chem* 1994; 40: 1674-1685
42. Kishimoto S, Kawazoe Y, Ikeno M, Saitoh M, Nakano Y, Nishi Y et al. Role of Na⁺,K⁺ -ATPase 1 subunit in the intracellular accumulation of cisplatin. *Cancer Chemother Pharmacol* 2006; 57: 84-90
43. Andrews PA, Mann SC, Huynh HH, Albright KD. Role of the Na⁺,K⁺-ATPase in the accumulation of cis-diammine-dichloroplatinum(II) in human ovarian carcinoma cells. *Cancer Res* 1991; 51: 3677-3681
44. Bando T, Fujimura M, Kasahara K, Matsuda T. Significance of Na⁺,K⁺ -ATPase on intracellular accumulation of cis-diamminedichloroplatinum(II) in human non-small-cell but not in small-cell lung cancer cell lines. *Anticancer Res* 1998; 18: 1085-1089
45. Lizuka N, Miyamoto K, Tangoku A, Hayashi H, Hazama S, Yoshino S et al. Downregulation of intracellular nm23-H1 prevents cisplatin-induced DNA damage in oesophageal cancer cells: possible association with Na⁺,K⁺-ATPase. *Br J Cancer* 2000; 83: 1209-1215
46. Ahmed Z, Deyama Y, Yoshimura Y, Suzuki K. Cisplatin sensitivity of oral squamous carcinoma cells is regulated by Na⁺,K⁺-ATPase activity rather than copper-transporting P-type ATPase, ATP7A and ATP7B. *Cancer Chemother Pharmacol* 2009; 63(4): 643-650

Effect of modified ridge lap and sanitary Pontic on periodontal health of abutment teeth of the three-unit posterior fixed partial denture

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Abstract

Background: This study was designed to find out and compare gingival condition, periodontal condition, periodontal pocket depth and root caries of the abutment teeth in response to modified ridge lap and sanitary Pontic. **Method:** It was a prospective comparative clinical study. This study was carried out in department of Prosthodontics, Faculty of Dentistry, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh from March 2017 to August 2019. A total number of 30 patients, aged between 25 and 70 years requiring replacement of a missing posterior tooth either upper or lower arch taken from those patients were reported. The study subjects selected by sequential sampling and grouped as Group A: 15 patients with modified ridge lap Pontic and Group B: 15 patients with sanitary Pontic. **Result:** According to the age distribution, in-group A, mostly 53.3% patients were in the age of 20-40 years and only 13.3% were more than 60 years whereas in-group B mostly 46.7% were in the age of 41-60 years and only 20 % were more than 60 years. Male patients were predominant in-group B whereas female patients were more in-group A. The distribution of patients having partial edentulous arch majority 66.7% and 73.3% were in lower arch in both group whereas only 33.3% and 26.7% were in upper arch in both group. The baseline periodontal health of both groups of patients were normal. However, the grade of gingival indices, periodontal indices was increased more in modified ridge lap Pontic than sanitary Pontic at 6th week, 12th week and 6th month follow up period ($P < 0.05$). Moreover, at 12th week and 6th month follow up period the grade of periodontal pocket depth indices increased more in modified ridge lap Pontic than sanitary Pontic. ($P < 0.05$). However, gradually root caries indices slightly increased in both groups of patients from the baseline up to 6th month. There was no significant difference between two groups at 6th week, 12th week and 6th month follow up period ($P > 0.05$). **Conclusion:** Based on the obtained clinical findings of the study it can be concluded that sanitary Pontic provide better periodontal health and less adverse effect on abutment teeth compared to modified ridge lap Pontic of posterior three unit fixed partial denture. Therefore, data should collected carefully to get significant results that will enlighten the whole treatment scenario in the field of dentistry.

Keyword: Fixed partial denture (FPD), Pontic, Sanitary Pontic, Modified ridge lap Pontic, Gingival index, Periodontal Index, Periodontal Pocket depth, Root Caries.

(J Cont Dent Sci 2020;8(2): 9-16)

Introduction:

The fixed partial denture (FPD) is a common treatment available for the replacement of missing tooth, which is largely dependent upon the health

and stability of the surrounding periodontal structures. Pontic defined as an artificial tooth on a fixed dental prosthesis that replaces a missing natural tooth, restores its function and usually fills the space previously occupied by the clinical crown¹. The function of a Pontic is to withstand masticatory load, to permit effective oral hygiene, preserve underlying residual mucosa and adjacent abutment tooth. Thus provides esthetics as well as restore function².

According to mucosal contact, Pontic can be divided into ridge lap, modified ridge lap, conical and ovoid. Pontic that have no mucosal contact are sanitary and modified sanitary Pontic. The various effects of Pontic design on periodontal tissue observed as periodontium surround and support the teeth. The ridge lap or saddle Pontic has a concave fitting surface that overlaps the residual ridge buccolingually. The modified ridge lap Pontic overlaps the residual ridge on the facial side only and the gingival surface of the Pontic is convex both buccolingually and mesiodistally. The sanitary Pontic does not come in direct contact with ridge.

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The modified sanitary pontic having gingival surface concave mesiodistally and convex buccolingually¹.

Pontic design with reference to form and contour is considered during preparation of FPD. Minimal ridge contact has been produced by the ridge lap Pontic and its modifications and finally definite separation between tissue and Pontic have been found by sanitary Pontic design. The sanitary Pontic is used in the absence of esthetic requirements chiefly in the posterior regions. The conventional design has its undersurface convex in all directions separated from gingiva by at least 1 mm³.

The undersurface of sanitary Pontic shaped slightly convex or flat buccolingually allows easy cleaning from either buccal or lingual aspect. The junction between the undersurface and the buccal and lingual surfaces of the Pontic rounded so as not to irritate the tongue and cheeks⁴.

Faulty Pontic design causes food impaction results in gingival inflammation and other form of periodontal disease, caries of the abutment teeth leading to subsequent failure of the prosthesis⁵.

Good oral hygiene may be able to prevent the development of inflammatory changes in the mucosa in contact with fixed bridge Pontic irrespective of the Pontic material used⁹. The abutment teeth are more prone to periodontal inflammation than the non-abutment teeth in fixed partial dentures. Prevalence of periodontal diseases have been found to be 94.9% increase in the plaque index, 97.4% increase in the gingival index, 100% increase in the probing pocket depth of the abutment teeth in fixed partial dentures⁶. The effect of FPD on periodontal health of abutment have been showed to be (93.7%) increase in the plaque index, (96.8%) increase in the gingival index and 100% increase in probing depth⁷. Therefore, it is logical that there is influence on periodontal health of abutment teeth in fixed partial denture and Pontic design has great role on periodontal health.

In addition to properly designing the undersurface of Pontic, it is imperative to open embrasure spaces adjacent to abutments to allow room for interproximal tissue and for oral hygiene⁸. The

modified ridge lap Pontic has better tissue response than ridge lap Pontic in respects of the condition of the tissue beneath the Pontic, color of the gingiva and condition of the abutment tooth⁹. Pontic design for maxillary and mandibular anterior teeth and maxillary premolars and first molars is modified ridge lap Pontic (47%) because it combines the features of saddle Pontic and also some hygienic.

In case of mandibular molars sanitary Pontic design (19%) should use for because it allows easy cleaning¹⁰.

The sanitary Pontic design in mandibular posteriors, 85% patients have reported to be no complaint from the prosthesis. Only 10% patients have been complaints of tongue irritation due to having 3-4 mm space between ridge and inferior border of Pontic¹¹. The design of sanitary Pontic permits easy cleaning of impacted food particles which allows easier plaque control that subsequently reduces caries and periodontal diseases of abutment tooth. The modified ridge lap Pontic does not make any contact with gingival tissue lingually, while facially it is in contact with the ridge. Therefore, food particles cannot be easily cleaned and less access for oral hygiene^{1, 12, 13}.

The surface smoothness of Pontic has considered more important in maintenance of tissue health than the material itself. The preservation of mucosa and periodontal tissue beneath and surrounding the Pontic are primary requirements regarding the success of fixed partial denture. Therefore, the Pontic must be constructed of highly glazed with minimal adaptation to the underlying tissue and having saliva contact rather than tissue contact^{14, 15, 16}.

Prevalence of root decay (18%), de-cementation of prosthetic restoration (7%) or periapical radiolucency (11%) have been found in the abutment¹⁷. Caries have also been observed to be one of the main reasons for prosthesis failure. Carious lesions recorded in 3.3% at the 5th year, 10% at the 10th year and 12% at the 15th year examination¹⁸. Therefore, Pontic morphology acts as a local risk factor in root decay and periodontal diseases.

The objective of this study was to evaluate the effect of three-unit posterior fixed partial denture with modified ridge lap and sanitary Pontic on periodontal health of abutment teeth.

Material and Methods

It was a prospective comparative clinical study. This study was carried out in department of Prosthodontics, Faculty of Dentistry, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh from March 2017 to August 2019. Patients who were attending in the department of Prosthodontics, BSMMU for replacement of a missing posterior tooth by fixed partial denture and voluntarily included in this study that was carried out in the department of Prosthodontics, BSMMU, Dhaka. The participants were divided into two groups. The sample size was 11 in each group according to sample size calculation. This sample size was increased up to 15 in each group to make allowance for missing answers and incomplete responses. Total sample size was ³⁰.

Purposive sampling technique was performed based on preselected inclusive and exclusive criteria. The study subjects selected by sequential sampling and grouped as follows:

Group A: Consisted of 15 patients with modified ridge lap Pontic and they treated with three-unit posterior fixed partial denture.

Group B: Consisted of 15 patients with sanitary Pontic and they treated with three-unit posterior fixed partial denture.

Clinical Procedure:

Preparation of vital abutment teeth for the fixed partial dentures were completed. Gingival retraction cord used to expose the margin and impression taken by poly vinyl siloxane material. Temporary crown cemented with eugenol free ZnO cement.

Laboratory Procedure:

Cast poured with type IV die stone. After the preparation of wax pattern, casting and finishing of the three unit fixed partial denture, proper evaluation of pontic design done and the metal framework tried in to the patient's mouth. Porcelain bonded with metal substructure and tried in patient's mouth. After all corrections, the three unit fixed partial denture was cemented with type 1 glass ionomer luting cement.

Instructions: Oral hygiene instructions given to the patients, including the use of dental floss and interproximal brushes for maintenance of the prosthesis. Each patient advised for avoiding hard foods and for recall visits at 6th week, at 12th week and at 6th month as mentioned above.

Examination for evaluation: The abutment was examined by visual examination and bleeding on probing regarding assessment of gingival condition. Average periodontal condition, periodontal pocket depth and root caries of the abutments of Fixed partial denture (FPD) was found out by mean of summation of grading of two abutments by a standard periodontal probe (calibrated 0-10 mm) and OPG x-ray.

Ethical clearance and Consent: Ethical clearance for the study taken from the Institutional Review Board of BSMMU (Ref: BSMMU/2018/13265; Date of approval: 22 September 2018) prior to the commencement of the study. Informed written consent obtained from the participants who voluntarily consented to participate in the study.

Data Analysis: Data were processed and analyzed with SPSS (Statistical Package for Social Science), version 22. Continuous data expressed as mean and standard deviation. Chi-square test used for

data analysis. The results presented in tables and figures. Results of significance was express as p-value and p-value < 0.05 considered as highest level of significance.

Results

In-group A, mostly 53.3% patients were in the age of 20-40 years whereas in group B, mostly 46.7% were in the age of 41-60 years. In group A, remaining 33.3% were in the age of 41-60 years and 13.3% were more than 60 years. In group B, remaining 33.3% were in the age of 20-40 years and 20% were more than 60 years.

Table-1: Comparison of periodontal health in between two groups by using gingival index (N=30)

Gingival index	Group A (Modified ridge lap Pontic) (n=15) No. (%)	Group B (Sanitary Pontic) (n=15) No. (%)	p- value
At baseline			
Grade 1	15(100.0%)	15(100.0%)	-
Grade 2	0(0.0%)	0(0.0%)	
Grade 3	0(0.0%)	0(0.0%)	
Grade 4	0(0.0%)	0(0.0%)	
Mean±SD	1.00±0.0	1.00±0.0	
At 6 th week			
Grade 1	3(20.0%)	9(60.0%)	0.036 ^s
Grade 2	9(60.0%)	6(40.0%)	
Grade 3	3(20.0%)	0(0.0%)	
Grade 4	0(0.0%)	0(0.0%)	
Mean±SD	2.00±0.65	1.40±0.51	
At 12 th week			
Grade 1	2(13.3%)	6(40.0%)	0.025 ^s
Grade 2	3(20.0%)	7(46.7%)	
Grade 3	7(46.7%)	2(13.3%)	
Grade 4	3(20.0%)	0(0.0%)	
Mean ± SD	2.73±0.96	1.73±0.60	
At 6 th month			
Grade 1	2(13.3%)	6(40.0%)	0.009 ^s
Grade 2	2(13.3%)	7(46.7%)	
Grade 3	7(46.7%)	2(13.3%)	
Grade 4	4(26.7%)	0(0.0%)	
Mean±SD	2.87±0.99	1.73±0.60	

Table-1 shows gingival indices of both groups of patients at baseline, at 6th week, 12th week and 6th month follow up period. Baseline gingival indices were similar in both groups of patients. However, the grade of gingival indices was increased more in

modified ridge lap Pontic than sanitary Pontic at 6th week, 12th week and 6th month follow up period (P<0.05).

Male patients were predominant in group B whereas female patients were more in group A. In-group A, male was 40% and female were 60%, and in group B, male was 53.3% and female were 46.7%.

Mostly patients distributed in lower dental arch in both groups of patients. 66.7% and 73.3% patients had partial edentulousness in lower arch in group A and group B respectively.

The baseline periodontal health of both groups of patients were normal in terms of gingival index, periodontal index, periodontal pocket depth and root caries.

Table-2: Comparison of periodontal health in between two groups by using periodontal index (N=30)

Periodontal index	Group A (Modified ridge lap Pontic) (n=15) No. (%)	Group B (Sanitary Pontic) (n=15) No. (%)	p-value
At baseline			
Grade 0	15(100.0%)	15(100.0%)	-
Grade 1	0(0.0%)	0(0.0%)	
Grade 2	0(0.0%)	0(0.0%)	
Grade 4	0(0.0%)	0(0.0%)	
Mean±SD	0.00±0	0.00±0	
At 6 weeks			
Grade 0	2(13.3%)	7(46.7%)	0.039 ^s
Grade 1	5(33.3%)	6(40.0%)	
Grade 2	8(53.3%)	2(13.3%)	
Grade 4	0(0.0%)	0(0.0%)	
Mean±SD	1.40±0.74	0.67±0.72	
At 12 weeks			
Grade 0	2(13.3%)	7(46.7%)	0.041 ^s
Grade 1	4(26.7%)	6(40.0%)	
Grade 2	7(46.7%)	2(13.3%)	
Grade 4	2(13.3%)	0(0.0%)	
Mean±SD	1.73±1.16	0.67±0.72	
At 6 months			
Grade 0	2(13.3%)	6(40.0%)	0.040 ^s
Grade 1	4(26.7%)	7(46.7%)	
Grade 2	6(40.0%)	2(13.3%)	
Grade 4	3(20.0%)	0(0.0%)	
Mean±SD	1.87±1.30	0.73±0.70	

Table-2 shows the periodontal indices of both groups of patients at baseline, at 6th week, 12th week and 6th month follow up period. Baseline periodontal indices were similar in both groups of patients. However, the grade of periodontal indices was increased more in modified ridge lap Pontic than sanitary Pontic at 6th week, 12th week and 6th month follow up period ($P<0.05$).

Table-3: Comparison of periodontal health in between two groups by using periodontal pocket depth index (N=30)

Periodontal pocket depth	Group A (Modified ridge lap Pontic) (n=15) No. (%)	Group B (Sanitary Pontic) (n=15) No. (%)	p-value
At baseline			
Grade 1	15(100.0%)	15(100.0%)	-
Grade 2	0(0.0%)	0(0.0%)	
Grade 3	0(0.0%)	0(0.0%)	
Grade 4	0(0.0%)	0(0.0%)	
Mean±SD	1.00±0.0	1.00±0.0	
At 6 th week			
Grade 1	5(33.3%)	8(53.3%)	0.519 ^{ns}
Grade 2	8(53.3%)	6(40.0%)	
Grade 3	2(13.3%)	1(6.7%)	
Grade 4	0(0.0%)	0(0.0%)	
Mean±SD	1.80±0.68	1.53±0.64	
At 12 th week			
Grade 1	2(13.3%)	8(53.3%)	0.037 ^s
Grade 2	8(53.3%)	6(40.0%)	
Grade 3	5(33.3%)	1(6.7%)	
Grade 4	0(0.0%)	0(0.0%)	
Mean±SD	2.20±0.68	1.53±0.64	
At 6 th month			
Grade 1	2(13.3%)	7(46.7%)	0.040 ^s
Grade 2	6(40.0%)	7(46.7%)	
Grade 3	5(33.3%)	1(6.7%)	
Grade 4	2(13.3%)	0(0.0%)	
Mean±SD	2.47±0.92	1.60±0.63	

Table-3 shows the periodontal pocket depth indices of both groups of patients at baseline, at 6th week, 12th week and 6th month follow up period. Baseline periodontal pocket depth indices were similar in both groups of patients. However, gradually periodontal pocket depth indices increased in both groups of patients from the baseline up to 6th month. There was no significant difference at 6th week between two groups

($P>0.05$). However, at 12th week and 6th month follow up period the grade of periodontal pocket depth indices increased more in modified ridge lap Pontic than sanitary Pontic. ($P<0.05$).

Table-4: Comparison of periodontal health in between two groups by using root caries index (N=30)

Root caries	Group A (Modified ridge lap Pontic) (n=15) No. (%)	Group B (Sanitary Pontic) (n=15) No. (%)	p-value
At baseline			
Grade 1	15(100.0%)	15(100.0%)	-
Grade 2	0(0.0%)	0(0.0%)	
Grade 3	0(0.0%)	0(0.0%)	
Mean±SD	1.00±0.0	1.00±0.0	
At 6 th week			
Grade 1	11(73.3%)	13(86.7%)	0.361 ^{ns}
Grade 2	4(26.7%)	2(13.3%)	
Grade 3	0(0.0%)	0(0.0%)	
Mean±SD	1.27±0.46	1.13±0.35	
At 12 th week			
Grade 1	10(66.7%)	12(80.0%)	0.409 ^{ns}
Grade 2	5(33.3%)	3(20.0%)	
Grade 3	0(0.0%)	0(0.0%)	
Mean±SD	1.33±0.49	1.20±0.41	
At 6 th month			
Grade 1	9(60.0%)	11(73.3%)	0.713 ^{ns}
Grade 2	4(26.7%)	3(20.0%)	
Grade 3	2(13.3%)	1(6.7%)	
Mean±SD	1.53±0.74	1.33±0.62	

Table-4 shows the root caries indices of both groups of patients at 6th week, 12th week and 6th month follow up period. Baseline root caries indices were similar in both groups of patients. However, gradually root caries indices slightly increased in both groups of patients from the baseline up to 6th month. There was no significant difference between two groups at 6th week, 12th week and 6th month follow up period ($P>0.05$).

Discussion

In this study in case of modified ridge lap Pontic, 60% patients presented with mild gingivitis at 6th week, 46.7% patients presented with moderate gingivitis at 12th week and at 6th month follow up. In case of sanitary Pontic, 60% patients presented with no gingivitis at 6th week, 46.7% patients presented with mild gingivitis at 12th week and at 6th month follow up.

In case of modified ridge lap Pontic 26.7% patients developed severe gingivitis at 6th month follow up when no patients found with severe gingivitis in case of sanitary Pontic. The result of the present study differs with the study conducted by Khan MMA (2015). In their study in case of ridge lap Pontic severe gingivitis was observed in 35% patients at 6th week, 25% patients at 12th week and 40% patients at 6th month follow up visit. The result of the study differ with the result of the present study may be due to cementing procedure of the FPD. Khan MMA (2015) used temporary cement material for cementation of the FPD, whereas type 1 glass ionomer luting agents was used for cementing the FPD in this study ⁹.

Tolboe et al. (1987) also reported mild to moderate gingivitis in case of ridge lap Pontic after 4 weeks while healthy gingiva was found in case of sanitary Pontic. The amount of accumulated bacterial deposits on the undersurface of the Pontic and the inflammation in the ridge lap area were estimated using microbiological test and histological examination by Tolboe et al. (1987) ¹⁹.

Hirshberg SM (1972) found that, 46 % patients presented with mild gingivitis, 18% with moderate gingivitis and 4% with severe gingivitis in case of ridge lap Pontic after 12 months follow up period. The results of the present study are an accordance with the well-known relationship between gingival inflammation and Pontic design. Although in this study to evaluate gingival condition in response to modified ridge lap Pontic and sanitary Pontic microbiological and histological examination not done. Nevertheless, the inflammatory changes of gingiva observed. As, more food debris were accumulated in modified ridge lap Pontic, gingivitis might have correlation with this food impaction as well. Therefore, the grade of gingival indices increased more in modified ridge lap Pontic than sanitary Pontic at 6th week, 12th week and 6th month follow up period in the present study²⁰.

Regarding periodontal index, 13.3% and 40% patients had shown to be normal periodontal tissue

at 6th month observation in case of modified ridge lap Pontic and sanitary Pontic respectively. Talabani (2016) reported that 88.6% patients had normal periodontal tissue in fixed partial denture after one year. Nevertheless, he did not mention type of Pontic design incorporated in his study ²¹.

In this study in case of modified ridge lap Pontic, 13.3% patients presented normal periodontal tissue and 53.3% patients developed moderate gingivitis at 6th week follow up. While in case of sanitary Pontic 46.7%, patients presented normal periodontal tissue and 13.3% patients developed moderate gingivitis at 6th week follow up. There is a general acceptance of high correlation between gingivitis and periodontal diseases. In case of modified ridge lap Pontic, 13.3% patients showed initial periodontal pocket at 12th week and 20 % patients showed the same at 6th month observation. But in case of sanitary Pontic none of the patient was found with pocket formation at 12th week and 6th month observation. Periodontal indices showed significant differences at all follow up visits.

This study did not get any significant differences in periodontal pocket depth index at 6th week. Nevertheless, at 12th week and 6th month follow up increased pocket depth observed in modified ridge lap Pontic than sanitary Pontic. Regarding periodontal pocket depth index, 53.3% patients presented with grade 2 pocket depth in case of modified ridge lap. On the other hand, in case of sanitary Pontic 53.3% patients presented with grade 1 pocket depth at 6th week follow up visit. At 12th week 33.3% patients found in grade 3-pocket depth in case of modified ridge lap, but in case of sanitary Pontic 6.7% patients found in grade 3-pocket depth. At 6th month 13.3 % patients developed grade 4-pocket depth in case of modified ridge lap, while no patients found in grade 4 pocket depth with sanitary Pontic. This observation might be considered as an outcome of increase food accumulation and gingival inflammation.

This result correlates with the study of Khan MMA (2015) who found that 45% patients had grade 1 pocket depth at 6th week, 20% had grade 3 pocket depth at 12th week and 10% had grade 4 pocket depth at 6th month observation in case of modified ridge lap Pontic⁹.

According to Sinaidi (2014) and Sinha N. et al (2017), the abutment teeth are to be more vulnerable of increasing pocket depth than non-abutment teeth. In this study, also pocket depth of abutment increased in both groups of patients from the baseline up to 6th month follow up. However, at 12th week and 6th month follow up period the grade of periodontal pocket depth indices increased more in modified ridge lap Pontic than sanitary Pontic^{6,7}.

The present study did not get any significant differences of root caries in between two groups of Pontic at 6th week, 12th week and 6th month follow up. Regarding root caries index, in case of modified ridge lap Pontic 73.3% patients found grade 1 and in case of sanitary Pontic 86.7% patients found grade 1 at 6th week follow up. At 12th week and 6th month follow up period, grade 1 was predominant in both groups of Pontic. At 6th month observation 26.7% patients developed grade 2 root caries and 13.3% developed grade 3 root caries with modified ridge lap Pontic while 20% patients found grade 2 root caries and only 6.7% developed grade 3 root caries with sanitary Pontic group.

The study conducted by Valderhaug et al. (1993) showed that caries recorded in 3.3% of the abutment tooth surfaces at the 5th year, 10.0% at the 10th year and 12.0% at the 15th year examination. However, they did not mention type of Pontic design incorporated in their study. They observed prevalence of caries might have correlation with duration of the prosthesis¹⁸. Talabani (2016) showed that 34.5% patients developed carious lesion in the abutment²¹. Dina et al. (2013) detected root decay occur in the 18% abutments in case of ridge lap Pontic. The above studies conducted over a long observation period where root caries detected. However, the present

study period was relatively short to develop significant root caries¹⁷.

In this study, patients instructed to follow the brushing technique and to brush the teeth twice after meal. Alqabbaa LM and Rayyan MR (2018) stated that gingival disease and periodontal inflammation of fixed partial denture could prevented by meticulous hygiene regime and regular maintenance. However, differences in ethnicity, lifestyle and other factors may influence the maintenance oral hygiene²².

Conclusion and Recommendation

Based on the obtained clinical findings of the study it can be concluded that sanitary Pontic provide better periodontal health and less adverse effect on abutment teeth compared to modified ridge lap Pontic of posterior three unit fixed partial denture. Sanitary Pontic causes less inflammation of gingiva than modified ridge lap Pontic. Periodontal pocket depth was more to measure in modified ridge lap Pontic. No remarkable root caries detected in both groups of Pontic.

Within the limitations of this study, recommended that the clinician can use sanitary Pontic in posterior fixed partial denture to maintain better periodontal health. Additional study with large sample size should done with long-term follow up.

References

1. Rosenstiel, S. F., Land, M. F., & Fujimoto, J. (2006). Contemporary fixed prosthodontics - E-book (4th ed.). London, England: Mosby.
2. Tylman, S. D. (1989). Theory and practice of fixed prosthodontics (8th ed.). Saint Louis, MI: MDMI.
3. Johnson, G. K., & Leary, J. M. (1992). Pontic design and localized ridge augmentation in fixed partial denture design. Dental Clinics of North America, 36(3), 591-605. doi:10.1016/s0011-8532(22)01818-3

4. Tjan, A. H. (1983). A sanitary-arc-fixed partial denture: concept and technique of pontic design. *The Journal of Prosthetic Dentistry*, 50(3), 338-341. doi:10.1016/s0022-3913(83)80087-0. Nagarsekar, A., Gaunkar, R., & Aras, M. (2016). Knowledge, attitude, and practice of dental professionals regarding the effect and management of food impaction associated with fixed partial denture prostheses: A survey. *Journal of Indian Prosthodontic Society*, 16(4), 372-379. doi:10.4103/0972-4052.191286
5. Nagarsekar, A., Gaunkar, R., & Aras, M. (2016). Knowledge, attitude, and practice of dental professionals regarding the effect and management of food impaction associated with fixed partial denture prostheses: A survey. *Journal of Indian Prosthodontic Society*, 16(4), 372-379. doi:10.4103/0972-4052.191286
6. Al-Sinaidi, A., & Preethanath, R. S. (2014). The effect of fixed partial dentures on periodontal status of abutment teeth. *The Saudi Journal for Dental Research*, 5(2), 104-108. doi:10.1016/j.ksujds.2013.11.001
7. Sinha, N., Gupta, N., Reddy, K. M., & Shastry, Y. M. (2017). Versatility of PEEK as a fixed partial denture framework. *Journal of Indian Prosthodontic Society*, 17(1), 80-83. doi:10.4103/0972-4052.197941
8. Becker, C. M., & Kaldahl, W. B. (1981). Current theories of crown contour, margin placement, and pontic design. *The Journal of Prosthetic Dentistry*, 45(3), 268-277. doi:10.1016/0022-3913(81)90387-5
9. Khan, M. A. A., Rahman, M., Islam, K. Z., Mohsina, N., Iqbal, M. A., & Jahan, M. S. (2015). A comparative study on tissue response under the ridgelap and modified ridgelap pontic. *Update Dental College Journal*, 5(1), 15-20. doi:10.3329/updcj.v5i1.25823
10. Zareen, N., & Gounder, D. R. (n.d.). Retrieved September 25, 2022, from Globalresearchonline.net website: <https://globalresearchonline.net/journalcontents/v40-1/55.pdf>
11. Khajuria, R. R., Safaya, R., & Singh, R. (2017). Pontic design in mandibular posteriors: An original research. *Annals of Dental Specialty*, 5(3), 101-103.
12. Nagib, M. A., Abhinav, A., Naeem, A., Abhishek, G., Kaushik, P., & Mariyam, A. (2017). All about dental pontics: Bridging the gap - A review. *Journal of Science*, 7(8), 294-298.
13. Oswal, M., & Oswal, M. (2016). Unconventional pontics in fixed partial dentures. *Journal of Dental and Allied Sciences*, 5(2), 84. doi:10.4103/2277-4696.192970
14. Cavazos, E., Jr. (1968). Tissue response to fixed partial denture pontics. *The Journal of Prosthetic Dentistry*, 20(2), 143-153. doi:10.1016/0022-3913(68)90137-6
15. Chansoria, S., & Chansoria, H. (n.d.). Abutment selection in fixed partial denture. doi:10.9790/0853-1703010412
16. Parkinson, C. F., & Schaberg, T. V. (1984). Pontic design of posterior fixed partial prostheses: is it a microbial misadventure? *The Journal of Prosthetic Dentistry*, 51(1), 51-54. doi:10.1016/s0022-3913(84)80105-5
17. Dina, M. N., Mărgărit, R., & Andrei, O. C. (2013). Pontic morphology as local risk factor in root decay and periodontal disease. *Revue Roumaine de Morphologie et Embryologie [Romanian Journal of Morphology and Embryology]*, 54(2), 361-364.
18. Valderhaug, J., Ellingsen, J. E., & Jokstad, A. (1993). Oral hygiene, periodontal conditions and carious lesions in patients treated with dental bridges. A 15-year clinical and radiographic follow-up study. *Journal of Clinical Periodontology*, 20(7), 482-489. doi:10.1111/j.1600-051x.1993.tb00395.x
19. Tolboe, H., Isidor, F., Budtz-Jørgensen, E., & Kaaber, S. (1987). Influence of oral hygiene on the mucosal conditions beneath bridge pontics. *European Journal of Oral Sciences*, 95(6), 475-482. <https://doi.org/10.1111/j.1600-0722.1987.tb01963.x>
20. Hirshberg, S. M. (1972). The relationship of oral hygiene to embrasure and pontic design-A preliminary study. *The Journal of Prosthetic Dentistry*, 27(1), 26-38. [https://doi.org/10.1016/0022-3913\(72\)90170-9](https://doi.org/10.1016/0022-3913(72)90170-9)
21. Talabani, Ranjdar. (2016). Influence of Abutment Evaluation on Designing of Fixed Partial Denture: A Clinical Study. *International Journal of Oral Health and Medical Research*. 3.
22. AlQabbaa, L., & Rayyan, M. (2018). Oral hygiene and maintenance habits among fixed partial denture wearers. *Saudi Journal of Oral Sciences*, 5(2), 115. https://doi.org/10.4103/sjos.sjoralsci_12_18

Does irradiation affect the mechanical properties of dentine?

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Abstract

Background and purpose: Radiation therapy is widely used for head-and-neck cancer treatment. However, reports suggest radiation affects oral hard tissues like enamel and dentine. Therefore, this study aimed to evaluate the effects of γ -radiation on the Hardness (H) and Elastic Modulus (E) of dentine. **Methods:** Ten sound third molars were randomly divided into two experimental groups according to the variable- radiation exposure (no irradiation: Normal dentine; irradiation with 60-Gy of γ -radiation: Irradiated dentine). All teeth were sectioned longitudinally to prepare 1 mm thick dentine slices. One slice from each tooth was selected, and the internal surface was sequentially finished and polished. After 24 hours of room drying, the H and E of the samples were tested. Statistical analysis was done by Independent Samples t Test ($\alpha = 0.05$). **Results:** A significant difference in mean H ($t=2.671 = 46, p = 0.01$) and E ($t=13.581 = 37.38, p < 0.001$) between normal and γ -ray irradiated dentine was observed. Both values were found to be significantly higher in normal dentine. **Conclusion:** γ -radiation at a dose of 60-Gy significantly reduces dentine's Hardness and Elastic Modulus.

Key Words: Irradiation, Mechanical Property, Hardness, Elastic Modulus, Dentine. (J Cont Dent Sci 2020;8(2):17-21)

Introduction

Radiotherapy is one of the effective methods of cancer treatment. Therefore, radiation therapy is widely used for head-and-neck cancer treatment. However, despite advances in radiation technology, radiotherapy in the targeted areas can still adversely affect dentition.¹ Dental hard tissue undergoes degeneration when exposed to 30-Gy radiation. The damage worsens with an accumulation of 60-Gy dose and a significant

reduction in ultimate tensile strength and microhardness.^{2,3} The irradiation dosage between 40 and 70-Gy is standard for the head and neck region⁴. Therefore, preventing radiation-induced damage to dental hard tissues like enamel and dentin is impossible.

Dentine comprises an organic matrix, primarily of Type I collagen and an inorganic phase made up of carbonate-rich apatite crystal. However, these components are not uniformly distributed in dentine's microstructure, contributing to a hypermineralized (~95 vol.%) peritubular dentine (PTD), interposed to a less mineralized collagen-rich (~30 vol.%) intertubular dentine (ITD)⁵. PTD contributes only 10-20% of human dentine⁶. Therefore, the mechanical characteristics of human dentine mainly depend on the ITD matrix⁷.

After radiation exposure, the crystal structures of dental hard tissues change. Radiation also demineralizes dentine's inorganic and organic components, as shown by a decreasing Calcium/Phosphorus ratio⁴. This alteration may lead to a reduction in the surface hardness of the substrate. Further, dentine is an intrinsically hydrated tissue. Its mechanical properties are affected by its hydration status⁸⁻¹⁰. Radiation decreases the amount of salivation¹¹. Reduced salivation could affect dentine's hydration status, altering mechanical properties. An ultra

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microhardness tester can assess such alterations in mechanical properties because the device allows the simultaneous measurement of Hardness and Elastic Modulus on small amounts of materials based on indentations' load-displacement data¹²⁻¹⁵.

The objective of this study was to evaluate the effects of 60-Gy γ -radiation on the Hardness and Elastic Modulus of dentine. The null hypothesis was that the irradiation would not affect the: 1) Hardness (H) and 2) Elastic Modulus (E) of dentine.

Materials and methods

This investigation followed the Declaration of Helsinki of 1975, revised in 2013. Accordingly, The institutional ethics committee approved the study protocol. An overview of the study design is shown in Figure 1.

Specimen preparation

Ten sound human third molar teeth were used for this study. After the patient's informed consent, the teeth were collected and stored in an aqueous solution of 0.5% Chloramine- T at 4- C and employed within three months of extraction¹⁶. The teeth were free from any signs of caries, cracks, or fractures. The teeth were randomly divided into two experimental groups according to the variable- radiation exposure (no irradiation: Normal dentine; irradiation with 60-Gy of γ -radiation: Irradiated dentine).

The Radiotherapy protocol consisted of a single radiation dose of 60-Gy. Radiation therapy was performed by a linear accelerator using 6 MV X-rays, a dose rate of approximately 6-Gy/min and a source-surface distance of 80 cm between the irradiation cycles. Five molars were subjected to 60-Gy of γ -radiation. The teeth were stored in distilled water during radiotherapy.

All teeth from both groups were then sectioned longitudinally to prepare 1 mm thick dentine slices by a low-speed diamond saw (IsoMet 1000, Buehler, Lake Bluff, IL, USA). One slice from

each tooth was selected, and the internal surface was sequentially finished with no. 1000-, 1200-, and 2000-grit SiC paper (Sankyo-Rikagaku Co., Saitama, Japan) under running water; and polished with ^{6, 3,} and 1- μ m diamond pastes (DP-Paste, Struers, Denmark). After every finishing and polishing step, the specimens were cleaned in an ultrasonic unit with distilled water for 3 mins.

H and E Tests

Pilot studies were done to establish the testing parameters for the H and E tests of the dentine slices. After 24 hours of room drying (22-24- C, 30% RH), the dentine slices were sequentially fixed on glass slides (Matsunami Glass Ind. Ltd., Osaka, Japan) and tested with an ultra microhardness tester (DUH-211, Shimadzu, Japan) having a triangular pyramidal diamond indenter with a tip angle of 115 and a radius 0.1 μ m (Figure 1).

Intertubular dentin was targeted, and indentations were performed at a constant speed of 0.2926 mN/s, with a 10 s holding at peak load. The maximum loads employed were 5.04 mN. At least a 10 μ m distance between adjacent indentations was maintained. Poisson's ratio was 0.30. Data were discarded when a part of any indentation included a dentinal tubule. H and E values were obtained from the default software of the ultra microhardness tester. Three H and E data from each dentine slice were recorded.

Statistical analysis

The normality and homogeneity of H and E data were tested using the Shapiro-Wilk and Levene's tests. The data were then analyzed by Independent Samples t-Test with the significance level set at 0.05. All statistical analyses were done using SPSS 25.0 for Windows (SPSS, Chicago, IL, USA).

Results

Independent Samples t Test revealed a significant difference in mean H between normal and γ -ray irradiated dentine ($t=2.671$, $df=46$, $p=0.01$).

Table 1. Mean - Standard Deviation of Hardness and Elastic Modulus of normal and 60-Gy γ -radiated dentine in MPa

Hardness (n = 15/group)		Elastic Modulus (n = 15/group)	
Normal dentine	Irradiated dentine	Normal dentine	Irradiated dentine
660 - 50.8 ^a	614.4 - 67 ^b	20725 - 1245 ^a	16713 - 738 ^b
Different superscript lower-case letters indicate significant differences [Independent Samples t Test; p - 0.05].			

The mean H of normal dentine (660 - 50.8 MPa) was significantly greater than that of irradiated dentine (614.4 - 67 MPa). A significant difference in mean E between normal and irradiated dentine was also observed ($t=13.581$, $p=0.001$). The mean E of normal dentine (20725 - 1245 MPa) was significantly higher than that of irradiated dentine (16713 - 738 MPa). The mean H and E values of the tested irradiated and nonirradiated human dentine are shown in Table 1.

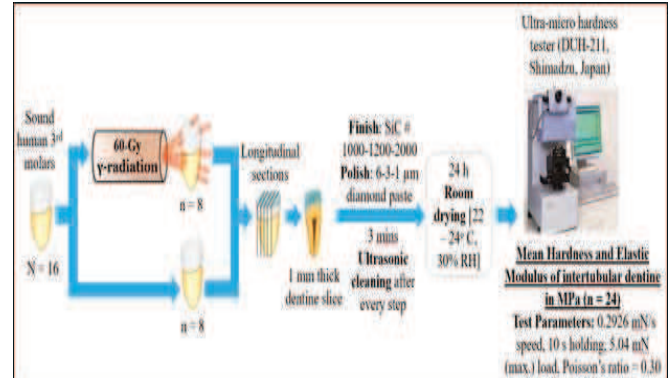
Discussion

As dentine's mechanical properties mainly depend on the ITD matrix⁷, we used ITD to determine dentine's H and E.

An ultra microhardness tester can simultaneously evaluate the H and E of a material by the Oliver and Pharr method¹⁷. Nevertheless, dentine's unique moist characteristic challenges testing because the ultra microhardness tester was designed for testing dry samples. Our pilot studies helped to address and overcome this drawback.

Marshall et al.¹⁸ reported that the hardness of hydrated intertubular dentine (ITD) lie between 0.15 ± 0.51 GPa. However, according to another report, H values increased to 0.6 ± 0.7 GPa when samples were tested in completely dry conditions⁵. The H values observed in the present study in 24-hour room-dried (22-24°C, 30% RH) normal human dentine samples (~ 0.6 GPa) are within this range. Upon drying, the collagenous matrix of dentine collapses, compressing the loose extrafibrillar mineral. This increases the rigidity of dentine, leading to higher surface hardness¹⁹. Previous studies reported that E values of ITD

range from 17.7 to 21.1 GPa¹⁸. The E values 20.5 GPa observed in the present study are also within this range.

**Figure 1:** Schematic of specimen preparation and test set-ups for determining the Hardness and Elastic Modulus of Normal and Irradiated human dentine.

Previous reports employing 60-Gy γ -radiation on dentine reported a significant decrease in ultimate tensile strength²⁰ and microhardness²¹. Similarly, in the present study, irradiation reduced human dentine's H significantly ($p=0.01$). In addition, we observed a significant decrease in dentine's E ($p<0.001$). Therefore, both null hypotheses have to be rejected. The significant reduction of H and E of irradiated dentine might have resulted from combined effects of altered type I collagen structure and decreased protein/mineral ratio²¹, fissure formation and obliterated tubules²².

Oyen ML reported that in the case of mineralized tissues, E also increases with the increase of H²³. In an indentation technique, H measures the material's resistance to deformation by surface indentation. On the other hand, E is the ratio of stress to strain when deformation is totally elastic. However, in the case of mineralized tissues, the contributions from elastic and plastic deformation can be similar. Therefore, H is directly dependent on E, meaning an increase or decrease in E contributes to a rise or fall of H. Angker et al. found similar results when they examined carious primary dentine²⁴. Chang et al.²⁵ and Zysset et al.²⁶ reported similar findings with alveolar bone and femur.

The mechanical properties of dentine can affect the bonding outcome of a restoration.²⁷ The significant changes in dentine's H and E reported in the present study might influence the selection of dental materials. In addition, they may help to prevent radiation-related caries in root dentine. Based on our observations, it could be extrapolated that restorative treatments can be performed using adhesive procedures in irradiated dentine. Still, it is preferable to complete the restorations before the irradiation protocol to guarantee better adhesive properties for restoration.

Conclusion

Within the limitations of the present study, we conclude that γ -radiation at a dose of 60-Gy significantly reduces human dentine's Hardness and Elastic Modulus.

References

- Andrews N, Griffiths C. Dental complications of head and neck radiotherapy: part 1. *Aust Dent J*. 2001; 46: 88-94
- Gon-alves LM, Palma-Dibb RG, Paula-Silva FW, Oliveira HF, Nelson-Filho P, Silva LA, Queiroz AM. Radiation therapy alters microhardness and microstructure of enamel and dentin of permanent human teeth. *J Dent*. 2014; 42: 986-992
- de Siqueira MT, Palma-Dibb RG, de Oliveira HF, Garcia Paula-Silva FW, Nelson-Filho P, da Silva RA, da Silva LA, de Queiroz AM. The effect of radiation therapy on the mechanical and morphological properties of the enamel and dentin of deciduous teeth-an in vitro study. *Radiat Oncol*. 2014; 9: 30.
- Velo MMAC, Farha ALH, da Silva Santos PS, Shiota A, Sansavino SZ, Souza AT, Hon-rio HM, Wang L. Radiotherapy alters the composition, structural and mechanical properties of root dentin in vitro. *Clin Oral Investig*. 2018; 22: 2871-2878
- Ziskind D, Hasday M, Cohen SR, Wagner HD. Young's modulus of peritubular and intertubular human dentin by nano-indentation tests. *J Struct Biol*. 2011; 174: 23-30
- Goldberg M, Kulkarni AB, Young M, Boskey A. Dentin: structure, composition and mineralization. *Front Biosci (Elite Ed)*. 2011; 3: 711-735
- Kinney JH, Pople JA, Marshall GW, Marshall SJ. Collagen orientation and crystallite size in human dentin: a small angle X-ray scattering study. *Calcif Tissue Int*. 2001; 69: 31-37
- Jameson MW, Hood JA, Tidmarsh BG. The effects of dehydration and rehydration on some mechanical properties of human dentine. *J Biomech*. 1993; 26: 1055-1065
- Sano H, Ciucchi B, Matthews WG, Pashley DH. Tensile properties of mineralized and demineralized human and bovine dentin. *J Dent Res*. 1994; 73: 1205-1211
- Bertassoni LE, Swain MV. Influence of hydration on nanoindentation induced energy expenditure of dentin. *J Biomech*. 2012; 45: 1679-1683
- Frank RM, Herdly J, Philippe E. Acquired dental defects and salivary gland lesions after irradiation for carcinoma. *J Am Dent Assoc*. 1965; 70: 868-883
- Sadr A, Shimada Y, Lu H, Tagami J. The viscoelastic behavior of dental adhesives: a nanoindentation study. *Dent Mater*. 2009; 25: 13-19
- Pedreira AP, Pegoraro LF, de Goes MF, Pegoraro TA, Carvalho RM. Microhardness of resin cements in the intraradicular environment: effects of water storage and softening treatment. *Dent Mater*. 2009; 25: 868-876
- Shahdad SA, McCabe JF, Bull S, Rusby S, Wassell RW. Hardness measured with traditional Vickers and Martens hardness methods. *Dent Mater*. 2007; 23: 1079-1085
- Zhang YR, Du W, Zhou XD, Yu HY. Review of research on the mechanical properties of the human tooth. *Int J Oral Sci*. 2014; 6: 61-69
- Armstrong S, Breschi L, -zcan M, Pfefferkorn F, Ferrari M, Van Meerbeek B. Academy of Dental Materials guidance on in vitro testing of dental composite bonding effectiveness to dentin/enamel using micro-tensile bond strength (μ TBS) approach. *Dent Mater*. 2017; 33: 133-143
- Oliver WC, Pharr GM. An improved technique for determining hardness and elastic modulus using load and displacement sensing indentation experiments. *J Mater Res*. 1992; 7: 1564-1583
- Marshall GW Jr, Marshall SJ, Kinney JH, Balooch M. The dentin substrate: structure and properties related to bonding. *J Dent*. 1997; 25: 441-458
- Bertassoni LE, Habelitz S, Kinney JH, Marshall SJ, Marshall GW Jr. Biomechanical perspective on the remineralization of dentin. *Caries Res*. 2009; 43: 70-77
- Soares CJ, Castro CG, Neiva NA, Soares PV, Santos-Filho PC, Naves LZ, Pereira PN. Effect of gamma irradiation on ultimate tensile strength of enamel and dentin. *J Dent Res*. 2010; 89: 159-164.

21. Reed R, Xu C, Liu Y, Gorski JP, Wang Y, Walker MP. Radiotherapy effect on nano-mechanical properties and chemical composition of enamel and dentine. *Arch Oral Biol.* 2015; 60: 690-697
22. Gonoalves LM, Palma-Dibb RG, Paula-Silva FW, Oliveira HF, Nelson-Filho P, Silva LA, Queiroz AM. Radiation therapy alters microhardness and microstructure of enamel and dentin of permanent human teeth. *J Dent.* 2014; 42: 986-992
23. Oyen ML. Nanoindentation hardness of mineralized tissues. *J Biomech.* 2006; 39: 2699-2702
24. Angker L, Swain MV, Kilpatrick N. Characterising the micro-mechanical behaviour of the carious dentine of primary teeth using nano-indentation. *J Biomech.* 2005; 38: 1535-1542
25. Chang MC, Ko CC, Liu CC, Douglas WH, DeLong R, Seong WJ, Hodges J, An KN. Elasticity of alveolar bone near dental implant-bone interfaces after one month's healing. *J Biomech.* 2003; 36: 1209-1214
26. Zysset PK, Guo XE, Hoffler CE, Moore KE, Goldstein SA. Elastic modulus and hardness of cortical and trabecular bone lamellae measured by nanoindentation in the human femur. *J Biomech.* 1999; 32: 1005-1012
27. Perdigão J. Dentin bonding-variables related to the clinical situation and the substrate treatment. *Dent Mater.* 2010; 26: e24-37

Acknowledgements: The authors acknowledge the technical support provided by Dr Shirin Shila for this research.

Nonsurgical endodontic retreatment of an incisor with a large periapical lesion: management of an endodontic failure case

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Abstract

Periapical lesions and subsequent external root resorption are pathological conditions that can lead to tooth loss if not treated properly. The presented case reflects a more conservative retreatment of a large periapical lesion, avoiding surgical procedures in an endodontically treated maxillary incisor. In this retreatment case, pure calcium hydroxide was used as an intracanal medicament after adequate biomechanical preparation. The patient was recalled at regular intervals. Multiple visits provided the opportunity for repeated intracanal medication. Clinically, the teeth became symptom-free within 3 weeks. Complete resolution of the periapical lesion with bone regeneration was observed within 14 months of treatment initiation.

Key Words: Endodontic Failure, Periapical Lesion, Nonsurgical Retreatment, Re-root Canal Treatment, Calcium Hydroxide.

(J Cont Dent Sci 2020;8(2):22-28)

Introduction

Endodontic treatment procedures have improved tremendously, showing remarkable promise in nonsurgical management of failure cases. The leading causes of endodontic treatment failure are microleakage, incomplete cleaning and obturation, retreatment, the complex anatomy of the tooth, and occlusal trauma¹⁻⁶. Bacterial colonization in root canals generally plays a vital role in the pathogenesis of periapical lesions⁷. This may progress into external resorption of the root and subsequent tooth loss.

Teeth with inadequate root canal obturation and asymptomatic periapical lesions usually harbour obligate anaerobic microorganisms despite sound coronal restorations^{8,9}. These microorganisms may induce the periapical lesions^{7,10}, characterized by bone resorption, followed by substitution by granulomatous tissue and a dense collection of polymorphonuclear leukocytes. Less commonly, there is an epithelial plug at the apical foramen to block the penetration of microorganisms into the extra-radicular tissues. Only a limited number of endodontic pathogens can penetrate these barriers. However, microbial products and toxins can penetrate these barriers to initiate and establish periradicular pathosis. Periapical radiolucencies are these lesions' most frequent clinical signs¹¹.

The endodontic treatment of teeth with periapical lesions varies according to the lesion size and the root resorption severity if present¹². Most periapical lesions heal after meticulous nonsurgical endodontic treatments¹³. When the initial root canal treatment fails to promote healing, the treatment options to save the tooth include nonsurgical endodontic retreatment with or without apical surgery, intentional replantation, transplantation, and extraction followed by an implant¹⁴. If the tooth is restorable, nonsurgical treatment is usually preferred because the procedure is less invasive than surgical endodontics, replantation, transplantation and extraction and replacement with implant¹⁵.

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Therefore, in the presented case, we opted for a nonsurgical approach to manage an endodontic treatment failure in a maxillary incisor tooth with a large periapical lesion.

Case report

A 37-year-old woman with a noncontributory medical and allergic history was referred to the Department of Conservative Dentistry & Endodontics, Sapporo Dental College & Hospital, to treat her maxillary right central incisor (tooth # 11) associated with localized pain and discharging sinus. The patient had a history of endodontic treatments followed by porcelain fused metal crowns on the maxillary left central incisor (tooth # 21), maxillary right central incisor (tooth # 11), and maxillary right lateral incisor (tooth # 12) due to severe attrition. The crowns were joined together. The treatments were done 4 years back.

The patient was moderately built and nourished and was afebrile during examination. Intraoral examination revealed pain on percussion present on teeth # 11, 12, and 21. Buccal and palatal marginal gingiva adjacent to the porcelain fused metal joint crowns on teeth # 11, 12, and 21 were inflamed.

The patient reported to our outpatient department with an intraoral periapical radiograph (Fig. 1 A), which revealed inadequate obturation on teeth # 11, 12, and 21. A large, oval-shaped periapical radiolucency was associated with tooth # 11. A small periapical radiolucency was associated with tooth # 21 (Fig. 1 A). Based on the patient's complaints, clinical examination, and radiological findings, our diagnoses were endodontic treatment failure due to inadequate obturation with chronic periapical abscess in tooth # 11, inadequate obturation with chronic periapical periodontitis in tooth # 21 and inadequate obturation in tooth # 12.

The treatment procedures were explained to the patient that the retreatment procedure would be continued without removing the porcelain crown for aesthetic purposes. The patient was further informed that periapical surgery might be

necessary if the nonsurgical retreatment procedure fails. The patient consented to the treatment plan.

At this initial appointment, access to teeth # 11, 12, and 21 root canals was established by preparing access cavities through the porcelain fused to metal crowns and removing Gutta Percha using Gutta Percha Solvent (NEOSOL/orikam, India) and Hedstrom Files (M access/Dentsply Sirona, Switzerland). The periapical lesion around the apex of tooth # 11 was measured at 7.5 x 6.8 mm (Fig. 1 B). Cotton rolls and a saliva ejector were used for isolation. A thin, purulent discharge from the canal of tooth # 11 was noticed. The canals of teeth # 11, 12, and 21 were then gently irrigated with Normal saline (Normal/The ACME Laboratories Ltd. Dhaka, Bangladesh). Cotton pellets were placed within the pulp chambers, the access cavities were kept open, and the patient was discharged with the advice of warm saline gargling as frequently as possible over the next 48 hours. A 3rd generation of Cephalosporin (Cefixime 400 mg, 12 hourly for seven days) was also prescribed to aid in periradicular microbial control associated with analgesic (Ibuprofen 400 mg, twice daily as necessary), and antiulcerant (Omeprazol 20 mg twice daily to be used with the analgesic). The materials and instruments used for the re-root canal treatment are listed in Table 1 and Table 2.

Table 1. Materials used for re-root canal treatment

Material	Name/Manufacturer
Gutta Percha Solvent	NEOSOL/orikam, India
Lubricating Agent	Glyde File Prep/Dentsply, USA
Normal Saline	Normal/The ACME Laboratories Ltd. Dhaka, Bangladesh
Paper Point (4%)	Absorbent Paper Points/DiaDent, Korea
Pure Calcium Hydroxide Powder	Calcium Hydroxide/Deepti Dental Products, Ratnagiri, India
Zinc Oxide Cement	e-Temp/DiaDent, Korea
Calcium Hydroxide Sealer	Sealapex/SybronEndo, Glendora, USA
Gutta Percha Points	Guttapercha Points/DiaDent, Korea

Table 2. Instruments used for re-root canal treatment

Instrument	Name/Manufacturer
Hedstrom File	M access/Dentsply Sirona, Switzerland
Kerr File	M access/Dentsply Sirona, Switzerland
Endodontic Rotary File	V-taper Gold/FANTA, China
Lentulospiral Paste Fillers	PASTE CARRIERS/ Mani, Japan

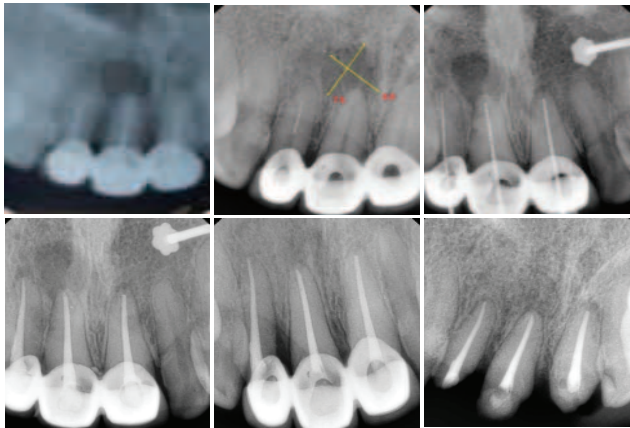


Fig. 1 A - F. Re-root canal treatment of failed endodontically treated teeth # 11, 12, and 21, follow-ups, and final restorations. A, Pre-operative intraoral periapical view showing a large oval-shaped radiolucency around the apex of tooth # 11 and a small radiolucency around the apex of tooth # 21; B, Establishing the access to the root canals and removal of the gutta percha obturating material from # 11, 12, and 21. The periapical lesion around the apex of tooth # 11 is measured at 7.5 x 6.8 mm; C, Working length determination; D, Obturation with Gutta Percha; E, 13 and half-months-follow-up showing bone regeneration and complete periapical healing; F, Removal of the faulty indirect restorations for restoration correction.



Fig. 2. Insertion of corrected porcelain fused to metal crowns on teeth # 11, 12, 21

On the next visit, pain on percussion persisted in teeth # 11, 12, and 21. The buccal and palatal marginal gingiva adjacent to the porcelain fused metal joint crowns on teeth # 11, 12, and 21 remained inflamed. Working length was determined by the # 40 Hedstrom File in tooth # 11 (18.5 mm), # 20 Hedstrom File in tooth # 12 (18 mm), and # 40 Kerr File (M access/Dentsply Sirona, Switzerland) in tooth # 21 (19 mm) (Fig. 1 C). Periodontal thickening became apparent in intraoral periapical views taken by the Radiovisiography technique in teeth # 11, 12, and 21 (Fig. 1 B - C), probably resulting from the faulty margins of porcelain fused metal joint crowns. After establishing working length, the canals were instrumented up to the apical size 55 in tooth # 11 and 21 and apical size 40 in tooth # 12 with Hedstrom Files. Normal saline was used as an irrigating solution. Then, cotton pellets were placed within the pulp chamber. The cavities were kept open again, and the patient was discharged with the advice of warm saline gurgling frequently over the next 48 hours.

After 48 hours, there was very mild pain on percussion in teeth # 11, 12, and 21. Buccal and palatal gingiva remained inflamed. Therefore, we performed scaling of the upper anterior region. Subsequently, biomechanical preparation was done using 4% Endodontic Rotary Files (V-taper Gold/FANTA, China) until dry dentine was observed. Glyde File Prep/Dentsply, USA, was used as a lubricating agent, and normal saline was used as an irrigating solution. The canals were dried with the help of sterile paper points (Absorbent Paper Points/DiaDent, Korea). Pure calcium hydroxide powder (Calcium Hydroxide/Deepti Dental products, Ratnagiri, India) was mixed with normal saline to be used as an intracanal medicament and placed in the canals with Lentulospiral Paste Fillers (PASTE CARRIERS/Mani, Japan). After applying cotton pellets in the pulp chambers, temporary restorations were placed (E-temp/DiaDent, Korea). The patient was discharged with the advice of meticulous oral hygiene maintenance associated with warm saline gurgling as frequently as possible over the next 7 days.

advice of meticulous oral hygiene maintenance associated with warm saline gurgling as frequently as possible over the next 7 days.

After one week, only mild discomfort was felt on percussion in tooth # 11. The buccal and palatal gingiva appeared normal. Copious irrigation was done with normal saline. Calcium hydroxide paste was placed after drying the canals. After applying cotton pellets in the pulp chambers, temporary restorations were placed. The patient was asked to visit after 7 days.

On the next visit, the teeth were found asymptomatic. The discharging sinus on tooth # 11 disappeared. The root canals were found dry. The canals were obturated after thorough irrigation, cleaning, and drying with sterile paper points. Obturation was done with thermoplastic Gutta Percha Point (Gutta-percha Points/DiaDent, Korea) used as filler and calcium hydroxide (Sealapex/SybronEndo, Glendora, USA) used as a sealer (Fig. 1 D). The patient was advised to revisit the hospital after 1 week.

On the next visit, the patient had no complaints. Radiographically, the apical radiolucency was more or less the same size. The patient was discharged with oral hygiene instructions. The next visit was scheduled after 1 month.

After one month, the teeth appeared normal clinically. However, the apical radiolucency was more or less the same size. The next visit was scheduled after 3 months. However, the patient came after 12 months for a follow-up visit. The patient had no complaints. The teeth appeared normal clinically. Radiographically, complete periapical healing was observed with bone regeneration (Fig. 1 E - F). Mild gingival inflammation was observed adjacent to the porcelain fused metal joint crowns. Therefore, the crowns were removed, and scaling was performed in the upper anterior region. The patient was instructed on meticulous oral hygiene maintenance for one week. After one week, there was no gingival inflammation, and periodontal condition also improved. Therefore, impressions were taken,

and new, corrected porcelain fused metal crowns were constructed and fixed on teeth # 11, 12, and 21 (Fig. 1 F and Fig. 2).

Discussion

Nonsurgical endodontic treatment with proper infection control can promote the healing of large lesions¹⁶. In such circumstances, the outcome of apical surgery is less consistent than nonsurgical treatment. A review revealed that the chance of teeth with apical periodontitis to completely heal after initial treatment or retreatment is 74 - 86%, and their probability to be functional over time is 91 - 97%. On the contrary, the chance of teeth with apical periodontitis completely healing after apical surgery is 37 - 85%, and their probability of being functional over time is 86 - 92%¹⁷. Considering the favourable outcome, nonsurgical endodontic therapy is justified. Therefore, it should be attempted when an excellent restorative and periodontal prognosis is projected. Hence, we chose nonsurgical endodontic treatment in the case reported.

The teeth were previously endodontically treated in the presented case but showed signs of periapical inflammation. The causes of endodontic treatment failure may include lack of proper tooth isolation, inadequate cleaning, shaping and irrigation, and incomplete obturation¹⁸. Bacteria, tissue debris, and other irritants not removed during cleaning and shaping constitute a potential source of irritation to periapical tissues and hamper healing, leading to chronic periapical lesions and endodontic failure¹⁹.

Periapical inflammation may result in radicular external resorption. The severity of resorption is proportional to the duration of the periapical inflammation. Histological studies show that the external resorption of cementum and dentine is due to the activity of granulation tissue in the area of chronic inflammatory process²⁰. Therefore, complete chemo-mechanical preparation is essential in root canal disinfection. However, the complete elimination of bacteria is difficult to

accomplish. The intracanal medicament may help to eliminate surviving bacteria when placed between appointments²¹. Calcium hydroxide has been widely used as an intracanal medicament for retreatment of failed cases²². In the presented case, we considered scheduling multiple appointments for the treatment process to benefit from repeated intracanal medication and irrigation to aid in canal disinfection.

Calcium hydroxide has been recommended as an intracanal medicament because of its antibacterial, anti-resorptive, and tissue-dissolving properties²³. It has two main bioactive mechanisms: killing bacteria in inaccessible areas of the root canal system and healing periapical lesions²⁴. Since calcium hydroxide has an alkaline pH, it actively influences the local environment around a resorptive area by reducing osteoclast activity and stimulating repair. The diffusion of hydroxyl ions released by calcium hydroxide through the dentinal tubules that directly communicate with periodontal ligament space would increase the pH of periodontal ligament space from 6.0 to 7.4-9.6^{23,25}. In this case, we observed that acute symptoms subsided following proper cleaning, shaping, and irrigation, followed by repeated application of calcium hydroxide as an intracanal medicament.

Endodontic therapy's success depends on various factors, including proper isolation, shaping and cleaning, irrigation, and complete obturation²⁶. Regarding irrigation, sodium hypochlorite is known to aid in achieving the sterility of the canals²⁷. However, as we did not use a rubber dam, sodium hypochlorite was not used to irrigate canals in the presented case. Fortunately, in our case, the flushing action of the normal saline irrigation, together with the use of calcium hydroxide as an intracanal medicament on multiple visits, proved enough for the disinfection of the canals, leading to the complete resolution of the periapical lesions after obturation within 14 months following treatment initiation (Fig. 1 E- F).

Cvek²⁸ reported a 96% success rate utilizing calcium hydroxide as an intracanal medicament and gutta-percha for obturation. Total removal of a resorption granuloma, calcium hydroxide treatment, and adequate root canal sealing are paramount for long-term endodontic success. Calcium hydroxide as a sealer also has antimicrobial and periapical healing properties²⁹. Therefore, in the reported case, we used calcium hydroxide sealer to benefit from the abovementioned properties.

For assessing periapical healing and endodontic treatment success, at least a 6-month³⁰ to 12-month³¹ period after root canal treatment should be considered. Nonetheless, complete healing of the periapical lesions might take up to four years in some cases, while signs of initiated but incomplete healing can be visible in most healing roots after 1 year³¹. In the presented case, complete periapical recovery was achieved with bone regeneration within 14 months of treatment initiation (Fig. 1 E – F).

Conclusion

In the reported endodontic treatment failure case, nonsurgical re-endodontic treatment with repeated calcium hydroxide intracanal medication has been proven sufficient for the induction of complete periapical healing with regeneration of bone. Calcium hydroxide sealer might have also contributed to the outcomes. After almost 14 months of treatment initiation, the teeth showed remarkable clinical and radiographical improvements.

References

1. Jokinen MA, Kotilainen R, Poikkeus P, Poikkeus R, Sarkki L. Clinical and radiographic study of pulpectomy and root canal therapy. *Scand J Dent Res*. 1978; 86: 366-373
2. Lindhe J, Nyman S, Ericsson I. Trauma from occlusion. In Lindhe J, Karring T, Lang NP eds. *Clinical Periodontology and Implant Dentistry*. 3rd edn. Blackwell Munksgaard 1998; 279-94

3. Pekruhn RB. The incidence of failure following single-visit endodontic therapy. *J Endod.* 1986; 2: 68-72
4. Petersson K, Lewin B, Hakansson J, Olsson B, Wennberg A. Endodontic status and suggested treatment in a population requiring substantial dental care. *Endod Dent Traumatol.* 1989; 153: 153-158
5. Swartz DB, Skidmore AE, Griffin JA. Twenty years of endodontic success and failure. *J Endod.* 1983; 9: 198-202
6. Torabinejad M, Ung B, Kettering JD. In vitro bacterial penetration of coronally unsealed endodontically treated teeth. *J Endod.* 1990; 16: 566-569
7. Kakehashi S, Stanley H, Fitzgerald RJ. The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. *Oral Surg Oral Med Oral Pathol.* 1965; 20: 340-349
8. Cheung GS, Ho MW. Microbial flora of root canal-treated teeth associated with asymptomatic periapical radiolucent lesions. *Oral Microbiol Immunol.* 2001; 16: 332-337
9. Sundqvist G, Figdor D, Persson S, Sj-gren U. Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1998; 85: 86-93
10. Moller AJ, Fabricius L, Dahl'n G, Ohman AE, Heyden G. Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys. *Scand J Dent Res.* 1981; 89: 475-484
11. Ramachandran Nair PN. Light and electron microscopic studies of root canal flora and periapical lesions. *J Endod.* 1987; 13: 29-39
12. Gunraj MN. Dental root resorption. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1999; 88: 647-665
13. Figdor D. Apical periodontitis: a very prevalent problem. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2002; 94: 651-652
14. Torabinejad M, White SN. Endodontic treatment options after unsuccessful initial root canal treatment: Alternatives to single-tooth implants. *J Am Dent Assoc.* 2016; 147: 214-220
15. Salehrabi R, Rotstein I. Epidemiologic evaluation of the outcomes of orthograde endodontic retreatment. *J Endod.* 2010; 36: 790-792
16. Mahmud MS, Shila S, Islam MN, Muktadir AMG, Sarker UK, Mahmud AA, Chowdhury AFMA. Management of a Discoloured Incisor Tooth with Large Periapical Lesion- A Case Report. *J Contemp Dent Sci.* 2019; 7: 28-32
17. Friedman S, Mor C. The success of endodontic therapy-healing and functionality. *J Calif Dent Assoc.* 2004; 32: 493-503
18. Allen RK, Newton CW, Brown CE Jr. A statistical analysis of surgical and nonsurgical endodontic retreatment cases. *J Endod.* 1989; 15: 261-266
19. Caliřkan MK. Nonsurgical retreatment of teeth with periapical lesions previously managed by either endodontic or surgical intervention. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2005; 100: 242-248
20. Sreeja R, Minal C, Madhuri T, Swati P, Vijay W. A scanning electron microscopic study of the patterns of external root resorption under different conditions. *J Appl Oral Sci.* 2009; 17: 481-486
21. Fuss Z, Tsesis I, Lin S. Root resorption-diagnosis, classification and treatment choices based on stimulation factors. *Dent Traumatol.* 2003; 19: 175-182
22. Sj-gren U, Figdor D, Persson S, Sundqvist G. Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. *Int Endod J.* 1997; 30: 297-306
23. Tronstad L, Andreasen JO, Hasselgren G, Kristerson L, Riis I. pH changes in dental tissues after root canal filling with calcium hydroxide. *J Endod.* 1981; 7: 17-21
24. Bystrom A, Claesson R, Sundqvist G. The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals. *Endod Dent Traumatol.* 1985; 1: 170-175
25. Esberard RM, Carnes DL Jr, Del Rio CE. pH changes at the surface of root dentin when using root canal sealers containing calcium hydroxide. *J Endod.* 1996; 22: 399-401

26. Bogen G, Handysides R. Retreatment. In Torabinejad M, Walton RE, Fouad AF eds. Endodontics Principles and Practice. 5th edn. Elsevier 2015; 355-375
27. Mohammadi Z, Shalavi S, Moeintaghavi A, Jafarzadeh H. A Review Over Benefits and Drawbacks of Combining Sodium Hypochlorite with Other Endodontic Materials. Open Dent J. 2017; 11: 661-669
28. Cvek M. Endodontic Treatment of Traumatized tooth. In Andreasen JO eds. Traumatic injuries of the Teeth. 2nd edn. Munksgaard 1981; 321-384
29. Desai S, Chandler N. Calcium hydroxide-based root canal sealers: a review. J Endod. 2009; 35: 475-480
30. Torres-Lagares D, Segura-Egea JJ, Rodr  guez-Caballero A, Llamas-Carreras JM, Guti  rrez-P  rez JL. Treatment of a large maxillary cyst with marsupialization, decompression, surgical endodontic therapy and enucleation. J Can Dent Assoc. 2011; 77: b87
31. Orstavik D. Time-course and risk analyses of the development and healing of chronic apical periodontitis in man. Int Endod J. 1996; 29: 150-155

Acknowledgements:

The authors would like to thank all the Department of Conservative Dentistry & Endodontics members, Sapporo Dental College & Hospital, Uttara, Dhaka, for their cooperation.

Management of Amelogenesis Imperfecta- A case Report

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Abstract

Background: Any divergence from the normal enamel formation leads to amelogenesis imperfecta which is a hereditary condition and resulted in sensitivity, loss of esthetics and impaired function. Rehabilitation of patient with amelogenesis imperfecta is greater challenge to the clinician. **Case presentation:** Based on clinical and radiological examination and family history, the presented case was diagnosed as hypoplastic smooth autosomal dominant type amelogenesis imperfecta. The treatment plan included patient motivation for the treatment and maintenance of oral hygiene, endodontic treatments of several teeth, onlay denture for correction of occlusion, vertical height of occlusion, mandibular deviation, and midline shifting, full veneer crown, fixed partial denture and direct composite veneer on anterior teeth to restore function and aesthetics. This treatment plan involved conservative dentistry and endodontic, and prosthodontics department. **Conclusion:** Amelogenesis imperfecta requires utmost care, evaluation, perfect treatment planning. Multidisciplinary treatment approach is essential for a better and successful treatment of AI. Patient education and patient co-operation are the basic requirements of a successful and desirable treatment plan.

Key Words: Amelogenesis imperfecta, Hypoplastic enamel, Autosomal dominant, Rehabilitation, Multidisciplinary treatment approach. (J Cont Dent Sci 2020;8(2):29-32)

Introduction

Enamel is the hardest part of the human body. In enamel formation, the protein structure of the enamel interacts with calcium phosphate minerals, creating elongated, parallel, and bundled apatite crystals that exhibit the greatest harness¹. Any divergence from the normal enamel formation leads to amelogenesis imperfecta (AI) which can be manifested in the oral cavity.

Amelogenesis imperfecta (AI) was first reported in 1890 and was not considered a clinical entity distinct from dentinogenesis imperfecta until 1938². AI is a term used to describe a group of hereditary conditions that have an effect on the structure and appearance of dental enamel, often

in conjunction with changes in other intra-oral and/or extra-oral tissue, in both primary and secondary dentition. The term amelogenesis imperfecta is also used to describe enamel phenotypes in other syndromes³.

AI can be classified in different ways. According to the mode of inheritance, according to molecular basis, according to the biochemical outcome, according to phenotype (type I-hypoplastic, type II- hypomaturational, type III- hypocalcified, type IV- hypomaturational hypoplastic with taurodontism)⁴. Type I is characterized by a hypoplastic structure with reduced enamel thickness, rough surface, and various extensions of defects. Type II shows mottled and softer enamel and chipping of the enamel from the dentin. Type III shows normal enamel thickness at the time of eruption but the enamel rapidly wears down. Type IV manifests as a mixed appearance of hypoplastic-hypomaturational combined with taurodontism⁵.

Case report

A 15-year-old male patient reported to the diagnosis department of Sapporo Dental College Hospital, Uttara, Dhaka with chief complaints of yellowish discoloration and broken back teeth with sensitivity.

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He could not chew food properly. His father gave a history of yellowish discoloration of deciduous teeth. He has no comorbidity. His father and two siblings presented with similar discoloration of teeth. His past dental history disclosed that he underwent filling on his few back teeth, root canal treatment of one of the back teeth due to decay and sensitivity, and extraction of another back tooth due to broken. The patient was referred to the Department of Conservative Dentistry and Endodontics and the Department of Prosthodontics for treatment and rehabilitation.

Extra-oral examination disclosed normal mouth opening, no temporomandibular joint problems, mandibular deviation to the right side, and class iii skeletal pattern. On intraoral examination, the Patient had permanent dentitions with missing 24 and congenitally missing 15,25. All teeth showed yellowish-brown discoloration. All remaining posterior teeth showed attrition. Caries was present on 12,22,27. Broken and attrited fillings were present on 14,16,17,26,34,36,37,44,45,46,47. The consistency of enamel in anterior teeth was hard and chipping of enamel was not present. The occlusal plane was uneven due to worn-down posterior teeth. The lower midline shifted to the right with mandibular deviation. Cross-bite was present in the anterior segment and right posterior segment. The vertical height of occlusion was decreased. The periodontal examination revealed normal healthy gingiva with no abnormalities (Figure 1,2,3).

Radiographic investigations included an orthopantomogram (OPG) and full-mouth intraoral periapical (IOPA) radiographs. The OPG showed the presence of all unerupted mandibular third molars and the absence of maxillary 3rd molars, as well as a seemingly normal pattern and timing of eruption of teeth [Figure 4]. Root canal treatment was found on 36. Examination of the IOPA radiographs revealed a normal pulp chamber and root canal spaces with no signs of obliteration.

Based on clinical and radiological examination and family history, it was diagnosed as hypoplastic smooth autosomal dominant type amelogenesis imperfecta. Based on the arrival diagnosis, a full

mouth rehabilitation was planned. A well-formulated treatment plan involving conservative dentistry and endodontic, and prosthodontics department was charted out to furnish a functional occlusion with good esthetics and further prevent tooth loss. The entire treatment plan was explained to the patient and informed consent was obtained. The initial phase of the treatment consisted of endodontic therapy.

Treatment protocol:

• Objectives of treatment-

- ❑ Prevention of further destruction of posterior teeth as well as restoration of function.
- ❑ Correction of mandibular deviation, midline shifting, and decreased vertical height occurred due to posterior teeth attrition.
- ❑ Improvement of aesthetics.

• Treatment plan-

- ❑ Patient motivation for the treatment and maintenance of oral hygiene,
- ❑ Endodontic treatments of # 14,16,17,26,27, 34,35,36,37,44,45,46,47
- ❑ Onlay denture for correction of occlusion, vertical height of occlusion, mandibular deviation, and midline shifting
- ❑ Full veneer crown on # 14,16,17,34,35,36, 37,44,45,46,47
- ❑ Fixed partial denture to replace 24. Retainer on abutment #23,
- ❑ Direct composite veneer on anterior teeth.
- ❑ Periodic follow-ups at 6 months, 1- and 2-year intervals followed by augmentation of oral hygiene measures.

Endodontic treatment was performed under local anesthesia. The working lengths for all teeth were obtained using hand K files and confirmed by diagnostic x-ray (RVG). Cleaning and shaping of all the canals were performed with hand K files and Protaper rotary files under copious irrigation with 3% sodium hypochlorite and Normal saline.



Figure 1: Frontal view



Figure 2: Maxillary occlusal view



Figure 3: Mandibular occlusal view



Figure 4: Preoperative OPG



Figure 5 showing radiograph after endodontic treatment



Figure 6- Onlay denture in corrected occlusion



Figure 7- Corrected occlusion after 6 months wearing onlay denture



Figure 8- Preoperative



Figure 9- Postoperative

EDTA was used as a chelating agent. Retreatment was performed with respect to # 26. Calcium hydroxide was used as intracanal medicament. All the access cavities were given temporary seal dressing with a cotton pellet and temporary filling material. All the obturation was completed using gutta-percha cones with non-eugenol, calcium hydroxide polymeric root canal sealer. Post-endodontic restoration of #14,16,17,26,27,34, 35,36,37,44,45,46,47 with light-cured composite resin (Figure 5).

An onlay denture was given to the patients at increased vertical height with a corrected midline position of teeth (Figure 6). After six months, the patient had a corrected occlusion (Figure 7) Porcelain fused to metal crowns were fabricated on # 14,16,17,34,35,36,37,44,45,46,47 and a fixed partial denture to replace 24. Teeth preparation was done with the appropriate cutting instruments. After fluid control and gingival retraction with a retraction cord, impressions were made by condensation silicone impression material. Provisional restorations were cemented in increased occlusal height and corrected occlusion. Evaluation of prosthesis was done for adequate proximal contact, marginal integrity, stability, and occlusion. All prostheses were cemented with glass ionomer cement. The direct veneer was done on #11,12,21,22,31,32,41,42 with light-cured composite resin.

Discussion

Enamel hypoplasia, hypomineralization (hypomaturation and hypocalcification), or a mixed phenotype are the most common clinical signs in amelogenesis imperfecta (AI). The characteristics of AI can be transferred by autosomal-dominant, autosomal recessive mode, and X-linked inheritance. This presented case was hypoplastic autosomal dominant type AIs. As the patient had a history of similar discoloration of one of his parents and siblings, the patient was diagnosed as autosomal dominant. The prevalence of various types of AI is known to differ between different populations. A study conducted in

Sweden revealed that a major percentage of cases were inherited as autosomal-dominant. Conversely, a study conducted in the Middle East revealed that the most common type of AI was autosomal recessive⁶.

A few genes such as amelogenin, enamelin, ameloblastin, amelotin, KLK4, MMP-20 etc. have been indicated as candidate genes for AI⁷⁻¹¹. The enamelin (ENAM) gene is a tooth-specific gene found at high level in the enamel organ, and, at a low level, in odontoblasts. The ENAM gene has been mapped within this location by radiation hybrid analysis and fluorescent in situ hybridization in autosomal dominant cases. So, this gene was considered a candidate gene for autosomal dominant type of AI⁸.

Mottling, opaque white-brownish yellow discoloration, highly brittle enamel was found in the hypomaturation type AI. Radiographically it was shown that the density of enamel is identical to dentin. Pigmented, softened, and loosened enamel was found in hypocalcification type AI. Radiographic analysis of this defect shows normal enamel thickness, but reduced density which is less than that of the dentin.

The normal enamel maturation but reduced thickness of enamel was found radiographically in the hypoplastic type AI¹². This presented case was diagnosed as hypoplastic type AI which also showed same feature of radiographic finding. Dentinal sensitivity, loss of vertical dimension, and esthetics were the common clinical features found in AI cases. Treatment planning in AI involved a comprehensive diagnosis and treatment using an interdisciplinary approach of periodontal, endodontic, prosthodontic and restorative treatment.¹³ A review literature concluded that the treatment plan for AI should be aimed at protecting the entire stomatognathic system and restoration of hard tissues. The final treatment modality till date still revolves around either full coverage restorations or, more of adhesive restorations¹⁴.

Several authors suggested the porcelain fused to metal restorations as the treatment modality for AI patients. With the increasing need for better esthetics and ability of bonding to dentin, porcelain fused to metal crowns has been well acceptable by the practitioners to restore the form and function to a satisfactory level^{14,16}. In this present report, porcelain fused to metal crown and light cured composite resin was used to increase vertical height of occlusion, prevent further destruction of posterior teeth, restore function, and improve aesthetics.

During treatment planning several various factors were taken into consideration, such as age of the patient, oral hygiene status, quality of life, internal anatomy of teeth, remaining tooth structure, Occlusion, temporomandibular joint, endodontic consideration etc. The treatment modalities may vary with the different factors and aim of treatment.

Conclusion

Rehabilitation and prevention of further damage of the patient with amelogenesis imperfecta is a challenge to the clinician. It requires utmost care, evaluation, perfect treatment planning. Multidisciplinary treatment approach is essential for a better and successful treatment of AI. Patient education and patient co-operation are the basic requirements of a successful and desirable treatment plan.

References

1. Pandya M, Diekwisch TGH. Amelogenesis: Transformation of a protein-mineral matrix into tooth enamel. *J Struct Biol*. 2021;213(4):107809.
2. Finn SB. Hereditary Opalescent Dentin. I. An Analysis of the Literature on Hereditary Anomalies of Tooth Color. *The Journal of the American Dental Association and The Dental Cosmos*. 1938;25(8):1240-9.
3. Gadhia K, McDonald S, Arkutu N, Malik K. Amelogenesis imperfecta: an introduction. *Br Dent J*. 2012;212(8):377-9.
4. Aldred MJ, Savarirayan R, Crawford PJM. Amelogenesis imperfecta: a classification and catalogue for the 21st century: Amelogenesis imperfecta, classification. *Oral Dis*. 2003;9(-):19-23.
5. Sidney B. Finn. Hereditary Opalescent Dentin. I. An Analysis of the Literature on Hereditary Anomalies of Tooth Color. *The Journal of the American Dental Association and The Dental Cosmos*. 1938;25(8):1240-1249
6. Aldred MJ, Savarirayan R, Crawford PJ. Amelogenesis imperfecta: a classification and catalogue for the 21st century. *Oral Diseases* 2003;9:19-236)
7. Hart TC, Hart PC, Gorry MC, Michalec MD, Ryu OH, Uygur C, et al. Novel ENAM mutation responsible for autosomal Amelogenesis imperfecta Chaudhary, et al. *JOMFP: Vol. 13 Issue 2 Jul - Dec 2009*
8. Kim JW, Seymen F, Lin BP, Kiziltan B, Gencay K, Simmer JP, Hu JC. ENAM mutations in autosomal-dominant amelogenesis imperfecta. *J Dent Res* 2005;84:278-82.
9. Ozdemir D, Hart PS, Firatli E, Aren G, Ryu OH, Hart TC. Phenotype of ENAM mutations is dosage-dependent. *J Dent Res* 2005;84:1036-41.
10. Toyosawa S, Fujiwara T, Shintani S, Sato A, Ogawa Y, Sobue S, et al. Cloning and characterization of the human ameloblastin gene. *Gene* 2000;256:1-11
11. Mårdh-Kärrman C, Backman B, Simmons D, Golovleva I, Gu TT, Holmgren G, et al. Human ameloblastin gene: genomic organization and mutation analysis in amelogenesis imperfecta patients. *Eur J Oral Sci* 2001;109:8-13.
12. Canger EM, Celenk P, Yenisey M, Odyakmaz SZ. Amelogenesis Imperfecta, hypoplastic type associated with some dental abnormalities: a case report. *Braz Dent J*. 2010;21(2):170-4.
13. Toksavul S, Ulusoy M, Türkün M, Kümbüloğlu Ö. Amelogenesis imperfecta: the multidisciplinary approach: a case report. *Quintessence Int*. 2004;35:11-4.
14. Turagam N, Mudrakola DP. Restoring esthetics and function in a patient with amelogenesis imperfecta—a multidisciplinary approach. *Dentistry*. 2015;5:3.
15. Roma M, Hegde S. Amelogenesis imperfecta: a review of the literature. *J Pharm Sci Res*. 2016;8(9):1042-4
16. Gökçe K, Canpolat C, Özel E. Restoring function and esthetics in a patient with amelogenesis imperfecta: a case report. *J Contemp Dent Pract*. 2007;8:90-101



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