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Cover: The elderly, whose numbers are increasing, are at particular risk of developing cancer, including in the head and neck region. Though necessary to the successful treatment of cancer patients, oral healthcare may not be a priority, especially when the patient is confined to a nursing home.

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Midlevel Providers Know the "What" and "How," Not the "Why" and "When"

There is no substitute for a fully trained dental professional.

Policymakers and legislators do a great disservice to the dental profession and to patients when they enable lesser-trained technicians to perform irreversible procedures independently, without the direct supervision of a dentist. The reduced education of midlevel providers (MLPs) leaves them only partially prepared to practice to the current standard of care of a dentist. While they might technically handle basic cases, they could place patients with more intricate and unusual needs at risk.

Proponents contend states should utilize MLPs to improve access to care for the underserved. However, engaging lesser-trained technicians to provide symptomatic care will not prevent future disease or remove the barriers to the utilization of existing care from dentists. As such, midlevel care does not serve patients' best interests. Hence, implementing such a proposal violates dentistry's contract with society, which, ultimately, will threaten dentistry's status as a profession.

Current dental school curricula, accreditation standards, licensing, scope-of-practice laws and the legal standard of care all evolved to set a minimum quality level and protect the public. The Commission on Dental Accreditation (CODA) sets the minimum competencies dental schools must teach and students must master as a prerequisite for the D.D.S./D.M.D. degree and, ultimately, licensure. These competencies developed for a critical reason. They progressed and advanced to ensure practitioners will apply state-of-the-art scientific knowledge safely to the public. Courts look to the principles taught in dental curricula as a basis for setting the legal standard of care. These competencies, standards, regulations and laws place the oral healthcare decision-making authority in the hands of a licensed dentist, the individual in the best position to act in the patient's best interest. Since these competencies and principles set a minimum standard of care, any program that incorporates only some, but not all, of the required standards, by definition, cannot certify its graduates competent to perform all the tasks reserved for a graduate of an accredited program.

MLPs generally receive an average of three years of dental education after high school. No doubt they learn "what" to do and "how" to do it. The key element of the advanced education and training of a doctor of dental surgery (or medicine) that distinguishes dentists from midlevel-trained technicians is the perspective attained in the broader context of the study of the biologic, dental, clinical and behavioral sciences. In essence, CODA competencies and the legal standard of care require that to earn the privilege and autonomy to perform irreversible procedures, dentists must not only possess the knowledge of "what" and "how" to perform, but also demonstrate the wisdom of understanding "why" conditions exist, "why" treatment should be rendered and "when" to take or not take certain action.

We can certainly teach MLPs the techniques to drill, fill and pull teeth. However, midlevel programs cannot prepare MLPs to competently interpret complex history, lab and radiographic findings, or manage patients with bleeding disorders, artificial heart valves, diabetes, oral malignancies and life-threatening infections, among others. We can teach, in a scaled down MLP course, techniques, observations and entry-level knowledge. We cannot develop in

the MLP the clinical judgment to know why a certain technique is appropriate, or when to revise treatment in the face of complicated dental and psychosocial problems or a compromised medical history.

Some advocates of MLPs claim such a system works in medicine, so it should succeed in dentistry. This argument fails, since the comparison is flawed. Dentistry stands as more of an unofficial surgical specialty of medicine and should be more appropriately compared to surgical medical specialties, not medicine in general. Medical MLPs, such as physician assistants and nurse practitioners, do not perform surgery on patients' brains, hearts, eyes or feet. Policymakers who take the position that MLPs can perform fillings and extractions indirectly imply that dental surgery is somehow not as complex or important as surgery in other parts of the body, which, of course, is absurd.

We can all agree organized dentistry needs to work to increase access and utilization of oral healthcare services to the underserved. However, the MLP model in dentistry offers little more than a politically expedient proposal, which will not succeed because it fails to address the removal of key barriers to utilization, such as oral healthcare literacy and prevention.

Society bestows professional status upon dentistry contingent upon the dental profession's commitment to act in the best interests of patients. Patients have a right to know they are protected and will receive the level of competency necessary for the safe delivery of oral healthcare. It seems unjust to take a position that makes potentially substandard care available to the underserved, when much more could and should be done to remove barriers to the utilization of available care.

Society expects dentistry to work with other professional organizations, government agencies and community groups to fight this fight. Together, we must not lose faith that, as we succeed in removing these barriers, the underserved will embrace our existing, high-quality oral healthcare system. All patients deserve access to dentists who not only know the "what" and "how," but also the "why" and "when."

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The Interprofessional Management of Geriatric Patients Undergoing Head and Neck Cancer Treatment in U.S. Nursing Homes

Literature Review

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ABSTRACT

Head and neck malignancies can be difficult to treat, especially in the geriatric population. A dental approach toward interprofessional management, treatment planning and rehabilitation of diagnosed individuals undergoing cancer therapies is essential. This literature review focuses primarily on: presurgery oral hygiene instructions; pre-radiotherapy/chemotherapy dental clearance; complications and management during radiotherapy/chemotherapy; post-radiotherapy/chemotherapy oral healthcare; and adjunctive measures. Each section aims to affirm that thorough evaluation is vital to understanding the assessment and safe management of patients undergoing oncology treatment. Furthermore, this project will help establish guidelines for interprofessional nursing home teams in regards to oral healthcare.

Head and neck cancer (HNC) constitutes less than 5% of all cancers, but it can have devastating outcomes in the lives of affected patients.[1-2] In 2001, there were approximately 30,000 HNC-related deaths in the United States.[1] It has been linked to several causes, including tobacco use and/or alcohol consumption. Eighty percent to ninety percent of head and neck cancers are due to tobacco and alcohol use. Traditionally, men over the age of 40 who smoke have the highest risk of developing HNC, squamous cell carcinoma (SCC). A recent study showed that development of oral cancer in smokers increases with age. Men who stopped smoking at 30 years of age had a 1.2% risk of developing SCC, while men 75 years of age and older who continued smoking had a 6.3% risk of developing cancer of the upper digestive tract.[1]

The incidence of human papilloma virus (HPV)-related squamous cell carcinoma has increased in the last 20 to 30 years. This disease usually occurs in the region of the oropharynx in patients who are 45 years or younger, so it is not usually a risk factor in the geriatric population. HPV-related SCC is discovered at a later stage, but it responds better to radiation treatment compared to non-HPV-related squamous cell carcinoma. Deficiencies of vitamin A and iron, occupational exposure to toxic chemicals and genetic abnormalities have also been linked to the development of cancer in the head and neck.[2] Immunosenescence, which is defined as the changes that occur in the immune system due to increased age, may cause an increase in the risk of cancer.[3]

It has been postulated that by the year 2030, 20% of the overall population will be composed of individuals 65 years and older.[4] The elderly, in particular, are at risk of developing cancer throughout the body, including the head and neck region. According to the American Cancer Society, people ages 55 and above represent 78% of all cancer diagnoses.[5] Treatment modalities for

cancer, such as surgery, radiation and/or chemotherapy, can take a physical, psychological and financial toll on the affected individuals.[6] Support from family, friends and nursing home staff can help these patients learn how to cope with their illness.

Nursing homes are facilities that provide long-term residential accommodations and/or healthcare for patients who do not require hospitalization but do require 24-hour assistance.[6-11] According to the Centers for Disease Control and Prevention, in 2013, the United States had 15,700 nursing homes, with 1.4 million beds out of a possible 1.7 million being occupied. While nursing homes provide healthcare to ensure a better quality of life, oral care has been found to be a low priority.[6] Nurses form an integral part of the interdisciplinary team caring for patients before, during and after treatment.[6] Dental professionals can assist nurses with patients living in these facilities.

For this study, the journals reviewed were published in English between 1975 and 2015 and contained information about the various types of cancers that occur in the head and neck region, along with treatment modalities and complications. Literature regarding dental care in nursing homes was also included. Abstracts and continuing education course materials were excluded. Relevant websites and textbooks with information related to the incidence of cancer and the care of affected patients were also used. This review will discuss pre-surgery oral hygiene instructions, pre-radiology/chemotherapy dental clearance, during-radiology/chemotherapy dental complications and management, post-radiology/chemotherapy oral healthcare and adjunctive healthcare. The aim of this review is to explore guidelines for nursing home healthcare teams in regards to oral health and interprofessional collaboration.

Presurgery Oral Hygiene

The healthcare team at a nursing home comprises family, physicians, nursing staff, social workers, rehabilitation staff, physical therapists, occupational therapists, speech therapists and other non-medical personnel.[6,12]

As the population ages, physician visits increase, while dental visits decrease for reasons ranging from the individual's lack of awareness, changes in oral hygiene habits and inability to afford dental care.[7] The nursing home staff may require more encouragement in recognizing the importance of oral care in older populations. According to a study by Coleman and Watson, five nursing homes in New York State did not meet oral hygiene standards for their residents, such as brushing their teeth for at least two minutes, flossing, performing an oral assessment, rinsing with mouthwash and wearing gloves during the performance of oral care.[13] The elderly are particularly at risk for the development of oral diseases that are gateways to other systemic disease, such as cardiovascular disease, stroke and respiratory infection.[14] Thus, it is extremely important for the team at the nursing home to administer a rigorous oral care protocol. One of the most effective ways to ensure change is to include interdisciplinary collaboration between nurses and dental professionals.

The risk of squamous cell carcinoma increases after age 65, so it is important that all staff be aware of the clinical manifestations. These include red and white mucosal lesions that cannot be wiped away and which should undergo biopsy if present for more than two weeks.[3] Non-healing ulcers present for more than two weeks should also be biopsied.[2] HNC may present as ominous-appearing nodular growths, palpable swellings, constant sore throat and otitis media that do not respond to antibiotics, as well as non-trauma-induced and uncontrolled bleeding of the mucosa.[2]

Once a patient has been diagnosed with cancer, it is important to increase oral hygiene measures. Cancer therapy can cause the oral cavity to undergo many changes, so it is best to establish a good regimen. Patients should be encouraged to cease all high-risk behaviors, such as smoking and excessive alcohol consumption.

Dental Clearance prior to Radiation and Chemotherapy

A comprehensive oral evaluation, prior to HNC treatment, is necessary to identify and eliminate active or potential oral sources

TABLE 1.
Oral and Dental Care Prior to Cancer Therapy

Dental Care Considerations	Guidelines and Action
Surgical Considerations	Vital and viable teeth should be retained for function, aesthetics, and quality of life.[70] Nonetheless, it is significant to eliminate possible sources of infection.[28] Due to the increased risk of osteoradionecrosis in irradiated bone with dental extractions or untreated infection, it is generally advised to extract teeth with poor long-term prognosis prior to radiotherapy.[71] Ideally, extractions should be undertaken up to two weeks before commencement of radiotherapy.[28,72] Teeth that should be considered for extraction include, but are not limited to:[70] • Extensive carious lesions with questionable pulpal status or involvement • Extensive peri-apical lesions • Moderate or advanced periodontal disease, with extensive attachment loss, bone loss and mobility or furcation involvement • Residual root tip that is radiolucent or if not fully covered by alveolar bone • Impacted or incompletely erupted teeth (third molars)[71, 73, 74]
Periodontal Considerations	Scaling and prophylaxis with establishment and reinforcement of good oral hygiene and dietary advice. With emphasis on adequate hydration.[75] Deep scaling and root planing (PD < 6 mm) should occur 14 days prior to radiotherapy for sufficient healing time.[28] • Recommended mouthrinse with aqueous alcohol-free chlorhexidine gluconate mouthwash for short-term use.
Restorative Treatment	Caries removal and restorations. Smoothing of irregular teeth and sharp areas on restoration. Removal and replacement of defective restorations.[74]
Prosthetic Considerations	Removable prostheses should be removed if any signs of ulceration. Ill-fitting dentures should be relined, repaired, or replaced to avoid irritation and tissue trauma.[23]
Orthodontic Appliances	Treatment should be discontinued.[76]

of infection. In cancer patients, the most frequently documented source of sepsis is the oral cavity.[15-20] Therefore, it is essential to evaluate and address dental needs for geriatric patients receiving oncology treatment. An early assessment of oral status and a means for providing care are critical in reducing potential complications associated with cancer therapy.[17]

Pre-treatment Comprehensive Oral Evaluation

Ideally, the oral evaluation should occur several weeks prior to commencement of cancer treatment, to allow for adequate healing time.[18,21,22] Medical consultation and thorough review of the patient's dental history are needed to develop an appropriate treatment plan. The medical consultation should encompass information concerning the disease/condition and associated treatment protocol, immunosuppression status, medications, allergies and contact information for the patient's oncologist.[17,23]

Dental health history must contain information regarding the patient's previous dental care, symptomatic teeth, trauma, habits, fluoride exposure, caries risk assessment and oral hygiene.[16] The assessment includes extraoral and intraoral clinical examinations, identification of existing infections and other compromised hard or soft issues, and an evaluation of relevant radiographs.[16,24]

Dental and Systemic Care prior to Oncology Therapy

Upon completion of the oral examination, it is critical that the dentist communicate the findings and associated treatment plan to the oncologist. If the radiation oncologist determines that a delay in cancer treatment will affect the potential success of disease control and patient survivability, pre-cancer dental care may not be advisable.[25,26] The main focus should be on existing infections, extractions, periodontal care and sources of tissue irritation (Table 1).[16,24,27,28] The team should educate the patient on proper oral hygiene, maintenance, nutrition and diet.[24] Oral and maxillofacial surgeons (OMS) play an important role in diagnosing HNC and patient management. The OMS is involved with cancer screenings and patient education, and surgical therapy.[29,30] The team approach will aid in reducing or eliminating complications, such as radiation mucositis, xerostomia and osteoradionecrosis.[31,32]

In addition to a complete oral examination, a comprehensive geriatric assessment (CGA) should be completed.[33] Geriatric patients often present with comorbidities, such as narrow arteries and decreased organ function, which may cause complications during surgery, radiation and chemotherapy. Chemotherapy-related toxicity is more common in elderly patients. This is caused by decreased liver and renal function, which allows abnormally high levels of chemotherapeutics to build up in the bloodstream.[29,30,33] CGA is a multidiscplinary evaluation of the patient's nutritional status, co-morbid medical conditions, cognition, psychological state and functional status. This assessment will help determine what type of treatment the patient will be able to withstand.[33] Systemic health benefits and oral care are critical to the quality of life for geriatric patients undergoing oncology treatment.[34]

Dental Complications and Management during Radiation/Chemotherapy Treatment

Oral or oropharyngeal mucositis (OM), an inflammatory disease of the oral and oropharyngeal mucosa, is commonly induced by ionizing radiation during radiation therapy (RT). Within three weeks, mucosal ulcerations manifest and become confluent.[35] These ulcerations may persist for up to six weeks after completion of RT and impair the patient's ability to eat, speak and function properly. These lesions leave patients vulnerable to microbial invasion of the bloodstream, increasing their risk of acquiring local or systemic infections.[36,37]

Therapeutic treatments, including soft laser and cryotherapy, show increasing evidence of effectiveness in preventing and managing OM. Lack of significant evidence, however, has limited their approval or utilization.

OM treatment is associated with symptom management.[38] As pain is typically present, treatment begins with topical analgesics or anesthetics, such as viscous lidocaine to help with OM.[15] Some clinicians will prescribe "magic mouthwash" to alleviate the pain associated with OM. There are many different formulas used to make magic mouthwash. Palifermin is the only FDA-approved growth factor or cytokine-approved medication to treat oral mucositis.[39-41] Alternating non-opioid oral analgesics, such as ibuprofen and Tylenol, is recommended if topical application is insufficient. If pain persists, opioids may be added, starting with the lowest effective dose and increasing as needed.[38,42]

Low-level laser therapy (LLLT) is a new modality, which may be used to reduce the effects of mucositis in patients treated with RT.[15,33,43] In a recent study by Gautam et al., LLLT treatment of the oral cavity prior to RT showed decreased OM, oral pain, weight loss and the need for analgesic in elderly patients with HNC.[15,43]

Xerostomia

Salivary glands are sensitive to RT. Xerostomia may manifest due to inflammation, fibrosis or degeneration of salivary glands. Decreased flow is continuous throughout RT and may persist post-treatment, increasing the risk of infections and the development of dental caries.[36] Current treatments for xerostomia hope to increase the existing salivary flow or replace lost secretions, maintain proper oral hygiene, control dental caries that may be present and treat any arising infections. The presence or absence of residual oral cavity secretions determines appropriate treatment.[42]

Xerostomia treatments include muscarinic acetylcholine agonists, salivary substitutes or stimulants, acupuncture and hyperbaric oxygen therapy (HBOT). Some examples of muscarinic acetylcholine agonists are pilocarpine and cevimeline. The continued administration of topical pilocarpine lozenges has been found to be superior to other treatments for improving unstimulated and stimulated salivary flow rates.[44]

Alternative treatments include mechanical, gustatory or electrical salivary stimulants. If salivary secretion stimulation fails, mouthwash or saliva substitutes that mimic natural saliva may be prescribed. Because of the increased risk for dental caries and infections, patients require frequent dental visits. Patients must be active in managing xerostomia by performing daily self-examinations for the presence of any white, red or dark-pigmented lesions, ulcerations or tooth decay.[42,44]

Infections

Radiation-induced xerostomia and mucosal ulcerations increase the risk of viral, bacterial or fungal infections. Common bacterial infections involve species of *Staphylococci* and *Streptococci*, *Klebsiella pneumonia*, *Pseudomonas aeruginosa* and *Escherichia coli*.[45] The most common infection is oral candidiasis (OC), an opportunistic fungal infection primarily associated with *Candida albicans*. The two types of acute OC found in this population are pseudomembranous and erythematous. Patients suffering from pseudomembranous OC may present with wipeable, white, patchy lesions, while erythematous OC manifests in the oral cavity as red diffused lesions on the oral mucosa. Frequently coexisting during RT, OC is often mistaken as oropharyngeal mucositis. Common symptoms are generalized burning sensations and pain.[35,46]

The treatment for OC is topical antifungal medication. Systemic antifungals are administered for more invasive infections. Antifungal agents that are fully absorbed from the GI tract, such as fluconazole, ketoconazole and itraconazole, appear to be more effective in preventing OC.[44,47]

Trismus and Fibrosis

Radiation-induced fibrosis and ischemia may lead to trismus during or post-RT. Unmanaged trismus may cause difficulty in swallowing. The main treatment for increasing mouth-opening is a constant exercise regimen.[37] Nursing home patients experiencing difficulty with exercising independently may manage exercise routines with the help of staff. Modified, custom-made mouth-opening devices could be an alternative treatment.[48]

Post-radiation/chemotherapy Complications and Management

Long-term complications of radiotherapy and chemotherapy include xerostomia, osteoradionecrosis, rampant caries and radiation-induced sarcomas. Severe cases of xerostomia can cause difficulty in speech and swallowing, making everyday tasks troublesome.[49]

Residents in nursing homes might experience extreme discomfort with removable prostheses because saliva promotes bonding between the interface of prosthesis and the oral/gingival tissue.[50,51] Xerostomia treatment includes salivary stimulants, mouth moisteners and parasympathomimetic drugs. Side effects might be sweating, headache, rhinitis, dizziness and urinary fre-

quency.[52-56] Persistent xerostomia can lead to rampant caries. Effective preventive therapies include maintenance of good oral hygiene, the use of fluoride and chlorhexidine rinse.[52] Conventional glass ionomers are the restorative material of choice because of their bond strength and fluoride release.[25]

Most cases of osteoradionecrosis occur as the result of traumatic incidents, such as tooth extraction, biopsies, periodontal disease, subgingival scaling or ill-fitting prostheses.[20] After radiotherapy, edentulous patients should not wear dentures for at least one year. Dental implants can be placed successfully in irradiated bone 12 to 18 months after completion of radiotherapy.[58] Although rare, treatment with radiation can cause post-radiation sarcomas.[57] Therefore, any suspicious lesion should be sent for biopsy. If the lesion is found to be malignant, surgical resection is often the main treatment of choice.[51] The oral and maxillofacial surgeon can aid in post-radiation/chemotherapy therapy by helping patients through the rehabilitation process.[29,30]

Prostheses are essential to help patients regain normal function and improve facial aesthetics. Prostheses include dentures, as well as treatments for other portions of the face and neck. Because tissues actively heal and change after surgery, a close follow-up of the fit of the prostheses and assessment of functional jaw improvement is recommended.[59] Patients, with the help of the nursing staff, should perform frequent oral examinations to identify any abnormal changes occurring in the oral cavity.[15] The dentist can help by performing monthly oral examinations for residents for the first six months after completion of their treatment(s) and semiannually thereafter.[59] An oral medicine specialist should be notified whenever oral pathology is suspected.[15]

Adjunctive Treatment

Innovative cancer treatments are keeping patients alive longer, resulting in complex disabilities, including fibrosis of irradiated tissue, trismus, dermatitis, and severe, acute mucositis and oropharyngeal.[53,56] Radiation-induced tissue damage occurs from injury to the endothelial cells lining small blood vessels, resulting in inflammation, ischemia and interstitial edema.[60] Edema in the head and neck causes facial disfigurement; in severe cases, swelling of eyelids and lips can lead to difficulties in eating, as well as impaired vision.[55] Traditional nursing measures, such as compression garments, ambulation and elevation, contribute to a reduction in lymphedema. Radiation dermatitis symptoms may be alleviated by skin care instructions, the use of aloe vera gels and water-based lotions, avoiding chemical irritants, and limiting sun and wind exposure. Massage and position changes can alleviate the pressure sores of bedridden patients.[60]

Patients should be aware of support services, such as physical, manual and occupational therapy, to reduce deconditioning and muscle atrophy. Manual therapy includes passive/active stretching and joint manipulation to increase range of motion (ROM) and reduce inflammation, hypoxia and contractile tensions.[61] A novel technique is trigger-point dry-needling, which decreases pain and increases cervical ROM and blood flow to the site in patients having upper myofascial pain.[62] Various jaw ROM exercises and mechanical assistance devices, such as Therabite *(Atos Medical, Sweden)*, can help increase ROM.[63] Fibrosis can result in impaired movement of the muscles of mastication, tongue, pharyngeal constrictors and larynx, resulting in swallowing dysfunction and risk for aspiration.[63-65] Tongue-stretching, as an adjunct to the supraglottic swallowing maneuver, helps with coordination of chewing and swallowing.[63]

Prevention counseling in oral hygiene, nutrition, alcohol and smoking should be offered. Smoking after a cancer diagnosis decreases the effectiveness of radiotherapy, shortens survival time and increase the risks of recurrence, second primary malignancies and treatment complication.[65] Cryotherapy (e.g., sucking on ice chips) can also lower the incidence of mucositis during infusions of chemotherapeutic agents by causing local vasoconstriction, thus reducing exposure of cells to the drug.[39,63] Pain associated with mucositis can be alleviated by using mouthrinses (Table 2) and gargling several times a day with warm salt water or a baking soda solution.[66]

TABLE 2.
Natural Adjunctive Mouthrinse Recommendations for Mucositis Secondary to Chemotherapy and/or Radiation for Cancer Treatment

Management/Therapy	Patient Diagnosis	Results	Type Study/Level Evidence	Author/Year
Rinse containing **olive leaf extract (OLE)** or benzydamine hydrochloride	Chemotherapy-induced mucositis—25 patients given mouthrinse with OLE, 25 patients given mouthrinse with benzydamine hydrochloride, 25 patients given placebo mouthrinse.	Oral mucositis rates and severity were lower in OLE and Benzydamine groups compared to placebo. Decrease in pro-inflammatory cytokine production.	Double-blind RCT, Level I	Ahmed (2012)[64]
Calendula officinalis (**English marigold**) flowers extract mouthwash as oral gel on radiation-induced oropharyngeal mucositis (OM)	38 patients with HNC undergoing radiotherapy (60 Gy) or concurrent chemoradiotherapy were randomly assigned to receive either 2% calendula extract mouthwash or placebo (20 patients in each group).	OMAS scores were significantly lower in calendula group compared to placebo at week 2, 3 and 6 of the study. According to repeated measures ANOVA test, the differences between OMAS of calendula and placebo during the weeks of evaluation were statistically significant ($p<0.001$).	RCT, Level I	Babaee (2013)[77]
Korean red ginseng (liquid concentrate) only Radiotherapy only RT+ KRG group	(20 Gy) Radiation-induced oral mucositis.	Rats in RT+ KRG group had less severe mucositis, fewer ulcerative mucosal lesions on the tongue, less hair loss, less rapid decrease in weight than did the RT-only group.	RCT, Level I	Chang (2014)[78]
Dioctahedral smectite (**natural absorbent clay** of non-systemic specific aluminomagnesium silicate) and iodine glycerin (DSIG) cream vs. topical mouthrinse (composed of saline, gentamicin and Vitamin B	18 y.o. and older with pathological confirmed malignant tumors or malignant hematological diseases, 130 intensive chemotherapy or stem cells transplantation induced OM. 67 patients received topical mouthrinse and 63 patients received DSIG cream treatment.	A favorable, lower oral assessment guideline (OAG) score was observed in DSIG cream treated patients. The iodine glycerin may function as an antifungal/antibacterial and decrease repair time. From day 2-5 topical mouthrinse patients had a higher OAG score than DSIG treated. "The mouthrinse shows a protective function prior to OM appearance."	Prospective RCT, Level I	Lin (2015)[79]
Curcumin mouthwash	20 adult cancer patients with radio-chemotherapy OM randomly divided into 2 groups.	Curcumin mouthwash was found to be better than chlorhexidine mouthwash in terms of rapid wound healing and better patient compliance.	RCT, Level I	Patil (2015)[80]
13 received an **aloe vera mouthwash**, 13 received benzydamine mouthwash.	26 HNC patients (receiving at least 50 Gy) with radiation-induced mucositis.	Aloe vera mouthwash was as beneficial as benzydamine mouthwash in alleviating the severity of radiation-induced mucositis and showed no side effects.	Triple Blind RCT, Level I	Sahebjamee (2015)[81]
16 received **polaprezinc**, 15 received azulene oral rinse (control).	31 HNC patients with radiotherapy or radiochemotherapy OM.	Incidence rate of mucositis, pain, xerostomia and taste disturbance was lower in polaprezinc group than control.	Prospective RCT, Level I	Watanabe (2010)[82]

RCT: Randomized Control Trial
Gy: Grays
OMAS: Oral Mucositis Assessment Scale
HNC: Head and Neck Cancer
OM: Oropharyngeal Mucositis
KRG: Korean Red Ginseng
RT: Radiation therapy

HNC patients are at high risk for malnutrition due to taste alterations, xerostomia from medications, parotid atrophy and radiation-induced diarrhea (RID).[65,66] The diagnosis of diminished swallowing function and odynophagia (painful swallowing) can be made by a speech language pathologist and treated by means of postures to control the bolus and other swallowing maneuvers. Twenty-nine percent to sixty-six percent of patients will experience severe oral mucositis during treatment, which is why alternative ways to provide nutrition need to be found.[67] Bolus modification by altering foods (mashed, puréed, thickened [fluids]) may make them safer to swallow.[68] Feeding tubes can also offer additional nutritional support post-treatment, when oral consumption is too painful.

Psychological support and speech rehabilitation can greatly improve the HNC patient's mental health. Frequently used alternatives include selenium, relaxation techniques, prayer, vitamin C, meditation and distraction.[69] Mild-to-moderate exercise can promote energy and boost quality of life and mood, despite feelings of tiredness.[70] Care plans should be tailored to meet the needs of each individual.

Conclusion

The objective of this review is to stress the importance of interprofessional collaboration and management for the treatment of patients undergoing various types of cancer therapy. Each healthcare provider has the knowledge to guide the geriatric population in nursing homes through the various phases of diagnosis, treatment and health maintenance post-treatment. The dentist and allied dental health professional can assist the nursing home staff with patients' oral hygiene and look for signs of recurrent disease. The goal of the interprofessional team is to increase overall health and quality of life.

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REFERENCES

1. Davies L, Welch HG. Epidemiology of head and neck cancer in the United States. Otolaryngol Head Neck Surg 2006;135:451-457.
2. Marur S, Forastiere AA. Head and neck cancer: changing epidemiology, diagnosis, and treatment. Mayo Clin Proc 2008;83:489-501.
3. Yap T, McCullough M. Oral medicine and the ageing population. Aust Dent J 60 (suppl 1): 2015. 44-53.
4. American Cancer Society Cancer facts and figures. Atlanta, GA: American Cancer Society 2015. 1-17.
5. Extermann M. Integrating a geriatric evaluation in the clinical setting. Semin Radiat Oncol 2012;22:272-276.
6. American Hospice Foundation, Roles of the family and health professionals in the care of seriously ill patients 2014. https://americanhospice.org/caregiving/roles-of-the-family-and-health-professionals-in-the-care-of-the-seriously-ill-patient/.
7. Arvidson-Bufano UB, Blank LW, Yellowitz JA. Nurses' oral health assessments of nursing home residents pre- and post-training: a pilot study. Spec Care Dentist 1996;16: 58-64.
8. Benson BH, Niessen LC, Toga CJ. Dental treatment and demand for services in a Veterans Administration nursing home care unit. J Public Health Dent 1984;44:147-155.
9. Gift HC, Cherry-Peppers G, Oldakowski RJ. Oral health care in US nursing homes, 1995. Spec Care Dentist 1998;18:226-233.
10. Logan HL, Ettinger R, McLeran H, Casko R, Secco DD. Common misconceptions about oral health in the older adult: nursing practices. Spec Care Dentist 1991;11:243-247.
11. Murray PE, Ede-Nichols D, Garcia-Godoy F. Oral health in Florida nursing homes. Int J Dent Hyg 2006;4:198-203.
12. Washington Health Care Association All about care conferences. 2015. http://www.whca.org/.
13. Coleman P, Watson NM. Oral care provided by certified nursing assistants in nursing homes. J Am Geriatr Soc 2006;54:138-143.
14. Coleman P. Opportunities for nursing-dental collaboration: addressing oral health needs among the elderly. Nurs Outlook 2005;53:33-39.
15. Elad S, Raber-Durlacher JE, Brennan MT, Saunders DP, et al. Basic oral care for hematology-oncology patients and hematopoietic stem cell transplantation recipients: a position paper from the joint task force of the Multinational Association of Supportive Care in Cancer/International Society of Oral Oncology (MASCC/ISOO) and the European Society for Blood and Marrow Transplantation (EBMT). Support Care Cancer 2015;23:223-226.
16. American Academy of Pediatric Dentistry. Guideline on dental management of pediatric patients receiving chemotherapy, hematopoietic cell transplantation, and/or radiation. Pediatr Dent 2013;35: E185-E193.
17. National Cancer Institute, Oral complications of chemotherapy and head/neck radiation (PDQ), http://www.cancer.gov/about-cancer/treatment/side-effects/mouth-throat/oral-complications-pdq. 2014. Accessed 01/22/16.
18. Scully C, Epstein JB. Oral health care for the cancer patient. Eur J Cancer B Oral Oncol 1996;32B:281-292.
19. Schubert MM, Peterson DE, Lloid ME. Oral complications. In: Thomas ED, Blue KG, Forman SJ, eds. Hematopoietic Cell Transplantation, 2nd ed. Oxford: Blackwell Science 751-763. 1999.
20. Toth BB, Martin JW, Fleming TJ. Oral complications associated with cancer therapy. An M. D. Anderson cancer center experience. J Clin Periodontol 1990;17 (suppl 1): 508-515.
21. National Institute of Dental and Craniofacial Research. Oral complications of cancer treatment: what the dental team can do. 2015. http://www.nidcr.nih.gov/OralHealth/Topics/CancerTreatment/OralComplicationsCancerOral.htm. Accessed 07/14/2015.
22. Little JW, Falace DA, Miller CS, Rhodus NL. Cancer and Oral Care of the Cancer Patient. St. Louis, MO: Elsevier-Mosby, 2012. p.459-492.
23. Padmini C, Bai KY. Oral and dental considerations in pediatric leukemic patient. ISRN Hematol 2014:895721.
24. Epstein JB, Ransier A, Sherlock CH, Spinelli JJ, Reece D. Acyclovir prophylaxis of oral herpes virus during bone marrow transplantation. Eur J Cancer B Oral Oncol 1996;32B: 158-162.
25. Andrews N, Griffiths C. Dental complications of head and neck radiotherapy: part 2. Aust Dent J 2001;46:174-182.
26. Vikram B. Importance of the time interval between surgery and postoperative radiation therapy in the combined management of head & neck cancer. Int J Radiat Oncol Biol Phys 1975;5:1837-1840.
27. da Fonseca MA. Childhood cancer. In: Casamassimo PS, Nowak AJ, eds. The Handbook of Pediatric Dentistry. 4th ed. Chicago: American Academy of Pediatric Dentistry. 2012.
28. Murdoch-Kinch CA, Zwetchkenbaum S. Dental management of the head and neck cancer patient treated with radiation therapy. J Mich Dent Assoc 2011; 93:28-37.
29. Cuddy KK, Mascarenbas W, Cobb G. Participation of Canadian oral and maxillofacial surgeons in oral, lip, and oropharyngeal cancer care. J Oral Maxillofac Surg 2015;73: 2440-2445.
30. Kim BB, Kim DD. The role of dental care providers and oral and maxillofacial surgeons in the surgical and medical management of oral cancer in the United States. Gen Dent 2013;61(7):47-53.
31. Mills K, Graham AC, Winslow BT, Springer KL. Treatment of nursing home-acquired pneumonia. Am Fam Physician 2009;79:976-982.
32. Hancock PJ, Epstein JB, Sadler GR. Oral and dental management related to radiation therapy for head and neck cancer. J Can Dent Assoc 2003;69:585-590.
33. Lalami Y, de Castro G, Bernard-Marty C, Awada A. Management of head and neck cancer in elderly patients. Drugs Aging 2009;26(7):571-583.
34. Villaret AB, Cappiello J, Piazza C, Pedruzzi B, Nicolai P. Quality of life in patients treated for cancer of the oral cavity requiring reconstruction: a prospective study. Acta Otorhinolaryngol Ital 2003;28:120-125.
35. Specht L. Oral complications in the head and neck radiation patient. Introduction and scope of the problem. Support Care Cancer 2002;10:36-39.
36. Al-Ansari S, Zecha JAEM, Barasch A, de Lange J, Rozema FR, Raber-Durlacher JE. Oral mucositis induced by anticancer therapies. Curr Oral Health Rep 2015;2:202-211.
37. Devi S, Singh N. Dental care during and after radiotherapy in head and neck cancer. Natl J Maxillofac Surg 2014;5:117-125.
38. Barasch A, Epstein JB. Management of cancer therapy-induced oral mucositis. Dermatol Ther 2011;24:424-431.
39. Lalla RV, Bowen J, Barasch A, Elting L, et al. MASCC/ISOO clinical practice guidelines for the management of mucositis secondary to cancer therapy. Cancer 2014. 15;120(10):1453-1461.
40. Otterholt R. Magic Mouthwash Prescription RX Recipes and Formulas. 2008-2017. http://www.drotterholt.com/magicmouthwash.html
41. Epstein JB, Hong C, Logan RM, Barasch A, Gordon SM, Oberle-Edwards L et al. A systematic review of orofacial pain in patients receiving cancer therapy. Support Care Cancer 2010;18:1023-1031.

42. Pinna R, Campus G, Cumbo E, Mura I, Milia E. Xerostomia induced by radiotherapy: an overview of the physiopathology, clinical evidence, and management of the oral damage. Ther Clin Risk Manag 2015;11:171-188.
43. Gautam AP, Fernandes DJ, Vidyasagar MS, Maiya AG, Guddattu V. Low level laser therapy against radiation induced oral mucositis in elderly head and neck cancer patients—a randomized placebo controlled trial. Journal of Photochemistry and Photobiology B: Biology 2015;144:51-56.
44. Lovelace TL, Fox NF, Sood AJ, Nguyen SA, Day TA. Management of radiotherapy-induced salivary hypofunction and consequent xerostomia in patients with oral or head and neck cancer: meta-analysis and literature review. Oral Surg Oral Med Oral Pathol Oral Radiol 2014;117:595-607.
45. Wong HM. Oral complications and management strategies for patients undergoing cancer therapy. Scientifc World J 2014:581795.
46. Garcia-Cuesta C, Sarrion-Pérez M-G, Bagán JV. Current treatment of oral candidiasis: a literature review. J Clin Exp Dent 2014;6:e576-e582.
47. Clarkson JE, Worthington HV, Eden OB. Interventions for preventing oral candidiasis for patients with cancer receiving treatment. Cochrane Database Syst Rev 2007; CD003807.
48. Ozdere E, Ozel GS, Aykent F Management of restricted mouth opening caused by radiation: a clinical report. J Prosthet Dent 2016;115:263-266.
49. Kam AY, McMillan AS, Pow EH, Leung KC, Luk HW. A preliminary report on patient acceptance of a novel intra-oral lubricating device for the management of radiotherapy-related xerostomia. Clin Oral Investig 2005;9:148-153.
50. Verma K, Gowda EM, Pawar VR, Kalra A. Salivary reservoir denture—a novel approach to battle xerostomia. Med J Armed Forces India 2015;71:S190-S193.
51. Vergo TJ Jr, Kadish SP. Dentures as artificial saliva reservoirs in the irradiated edentulous cancer patient with xerostomia: a pilot study. Oral Surg Oral Med Oral Pathol 1981;51:229-233.
52. Walsh LJ. Clinical assessment and management of the oral environment in the oncology patient. Aust Dent J 1981;55(suppl 1):66-77.
53. Chao KS. Protection of salivary function by intensity-modulated radiation therapy in patients with head and neck cancer. Semin Radiat Oncol 2002;12:20-25.
54. Sonis S, Elting L, Keefe D, Nguyen H, Grunberg S, Randolph-Jackson P, et al. Unanticipated frequency and consequences of regimen-related diarrhea in patients being treated with radiation or chemoradiation regimens for cancers of the head and neck or lung. Support Care Cancer 2015;23:433-439.
55. Silverman AT, Hoffman R, Cohen M, Garza R. Severe cheek and lower eyelid lymphedema after resection of oropharyngeal tumor and radiation. J Craniofac Surg 2010;21:598-601.
56. Huber MA, Terezhalmy GT. The head and neck radiation oncology patient. Quintessence Int 2003;34:693-717.
57. Pauloski BR. Rehabilitation of dysphagia following head and neck cancer. Phys Med Rehabil Clin N Am 2008;19:889-928.
58. Thiagarajan A, Iyer NG. Radiation-induced sarcomas of the head and neck. World J Clin Oncol 2014;5:973-981.
59. Schweiger JW, Salcetti MA. Dental management of the geriatric head and neck cancer patient. Gerodontology 1986;5:119-122.
60. Glastonbury CM, Parker EE, Hoang JK. The postradiation neck: evaluating response to treatment and recognizing complications. AJR Am J Roentgenol 2010;195:W164-W171.
61. Krisciunas GP, Golan H, Marinko LN, Pearson W, Jalisi S, Langmore SE. A novel manual therapy program during radiation therapy for head and neck cancer—our clinical experience with 5 patients. Clin Otolaryngol 2015. DOI: 10.1111/coa.12535.
62. Pecos-Martin D, Montanez-Aguilera FJ, Gallego-Izquierdo T, Urraca-Gesto A, Gomez-Conesa A, Romero-Franco N et al. Effectiveness of dry needling on the lower trapezius in patients with mechanical neck pain: a randomized controlled trial. Arch Phys Med Rehabil 2015;96:775-781.
63. Katranci N, Ovayolu N, Ovayolu O, Sevinc A. Evaluation of the effect of cryotherapy in preventing oral mucositis associated with chemotherapy—a randomized controlled trial. Eur J Oncol Nurs 2012;16:339-344.
64. Ahmed KM. The effect of olive leaf extract in decreasing the expression of two pro-inflammatory cytokines in patients receiving chemotherapy for cancer. A randomized clinical trial. Saudi Dent J 2013;25:141-147.
65. de Bruin-Visser JC, Ackerstaff AH, Rehorst H, Retel VP, Hilgers FJ. Integration of a smoking cessation program in the treatment protocol for patients with head and neck and lung cancer. Eur Arch Otorhinolaryngol 2012;269:659-665.
66. Bonomi M, Batt K. Supportive management of mucositis and metabolic derangements in head and neck cancer patients. Cancers 2015;7:1743-1757.
67. Ps SK, Balan A, Sankar A, Bose T. Radiation induced oral mucositis. Indian J Palliat Care 2009;15(2)92-102.

68. Swan K, Speyer R, Heijnen BJ, Wagg B, Cordier R. Living with oropharyngeal dysphagia: effects of bolus modification on health-related quality of life—a systematic review. Qual Life Res 2015;24:2447-2456.
69. Guru K, Manoor UK, Supe SS. A comprehensive review of head and neck cancer rehabilitation: physical therapy perspectives. Indian J Palliat Care 2012;18:87-97.
70. Jawad H, Hodson NA, Nixon PJ. A review of dental treatment of head and neck cancer patients, before, during and after radiotherapy: part 1. Br Dent J 2015;218:65-68.
71. Jansma J, Vissink A, Spijkervet FK, Roodenburg JL, Panders AK, Vermey A, et al. Protocol for the prevention and treatment of oral sequelae resulting from head and neck radiation therapy. Cancer 1992;70:2171-2180.
72. Tong AC, Leung AC, Cheng JC, Sham J. Incidence of complicated healing and osteoradionecrosis following tooth extraction in patients receiving radiotherapy for treatment of nasopharyngeal carcinoma. Aust Dent J 1999;44:187-194.
73. Beumer J, Harrison R, Sanders B, Kurrasch M. Osteoradionecrosis: predisposing factors and outcomes of therapy. Head Neck Surg 1984;6:819-827.
74. Schiodt M, Hermund NU. Management of oral disease prior to radiation therapy. Supportive Care Cancer 2002;10:40-43.
75. Rutkauskas JS, Davis JW. Effects of chlorhexidine during immunosuppressive chemotherapy. A preliminary report. Oral Surg Oral Med Oral Pathol 1993;76:441-448.
76. Sheller B, Williams B. Orthodontic management of patients with hematologic malignancies. Am J Orthod Dentofacial Orthop 1996;109:575-580.
77. Babaee N, Moslemi D, Khalilpour M, Vejdani F, Moghadamnia Y, Bijani A, et al. Antioxidant capacity of calendula officinalis flowers extract and prevention of radiation induced oropharyngeal mucositis in patients with head and neck cancers: a randomized controlled clinical study. Daru 2013;21:18.
78. Chang JW, Choi JW, Lee BH, Park JK, Shin YS, Oh YT, et al. Protective effects of Korean red ginseng on radiation-induced oral mucositis in a preclinical rat model. Nutr Cancer 2014;66:400-407.
79. Lin J-X, Fan Z-Y, Lin Q, Wu D-H, Wu X-Y, Chen Y-R, et al. A comparison of dioctahedral smectite and iodine glycerin cream with topical mouth rinse in treatment of chemotherapy induced oral mucositis: a pilot study. Eur J Oncol Nurs 2015;19:136-141.
80. Patil K, Guledgud MV, Kulkarni PK, Keshari D, Tayal S. Use of curcumin mouthrinse in radio-chemotherapy induced oral mucositis patients: a pilot study. J Clin Diagn Res 2015; 9:ZC59-ZC62.
81. Sahebjamee M, Mansourian A, Hajimirzamohammad M, Zadeh MT, Bekhradi R, Kazemian A, et al. Comparative efficacy of aloe vera and benzydamine mouthwashes on radiation-induced oral mucositis: a triple-blind, randomised, controlled clinical trial. Oral Health Prev Dent 2015;13:309-315.
82. Watanabe T, Ishihara M, Matsuura K, Mizuta K, Itoh Y. Polaprezinc prevents oral mucositis associated with radiochemotherapy in patients with head and neck cancer. Int J Cancer 2010;127:1984-1990.


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Stress Relief through Ergonomics for Dental Professionals

Colleen A. Watson, D.D.S.; Anupama Sangadala, D.D.S.; Peter Marino, C.P.T.

ABSTRACT

As most practitioners probably know from experience, dentistry is a physically taxing profession. Long hours spent with our heads bent over to get those crown preps just right can leave us with tense and aching muscles. Along with limited sleep, bad posture and not enough time to get to the gym, these workplace habits can leave us with chronic neck, shoulder and back pain in no time. Although limited research in occupational therapy has been specifically targeted towards dental professionals, there have been extensive studies on developing exercises to strengthen the muscles that support joints and spines. While exercises that require special machinery or gym memberships might not be realistic, considering time restraints, we have compiled a few exercises that you can try anywhere, anytime. Next time your patient cancels or you go home a little early, maybe you can squeeze in a few repetitions to help ward off the injury or pain that often seems inevitable as a dental professional.

As most practitioners probably know from experience, dentistry is a physically taxing profession. Long hours spent with heads bent over to get crown preps just right can leave a dentist with tense and aching muscles. Along with limited sleep, bad posture and not enough time to get to the gym, these workplace habits can leave dentists with chronic neck, shoulder and back pain in no time. Although limited research in occupational therapy has been specifically targeted towards dental professionals, there have been extensive studies on developing exercises to strengthen the muscles that support joints and spines. While exercises that require special machinery or gym memberships might not be realistic, considering most professionals' time restraints, we have compiled a few exercises to try anywhere, anytime.

Next time a patient cancels or you go home a little early, maybe you can squeeze in a few repetitions to help ward off injury or pain that often seems inevitable as a dental professional.

Begin by assembling the following basic equipment:

- Kettle bell. (Choose a weight appropriate for comfort and ability.)
- Rubber tubing.
- Yoga mat.
- Light dumbbells.

Upper Back and Shoulder Training


One Arm Row

Hold the kettle bell in your right hand by the handle. With your back straight and spine neutral, bend forward at the waist to a 90-degree angle. Extend your opposite (left) leg back. Extend kettle bell toward the floor and then pull it back up toward your body, until your wrist reaches your side. Repeat for 10 repetitions, then switch arms and do the same on the opposite side, extending the opposite leg. In addition to working the upper back and shoulders this exercise also works on the core muscles and balance—it is okay to wobble a little; you are training your core also. When you find you are no longer challenged and are able to balance yourself fairly well, you can increase the weight. Recommend 3 sets of 10 repetitions per side.


Bridge Pull-over with Kettle Bell

This exercise will work to strengthen the core muscles, which include both the abdominal and upper back muscles. Both sets of muscles should be balanced in strength to maintain optimal posture. It will also strengthen the shoulders, triceps and rotator cuff muscles, which are often sites of injury.

Lie on your back on the mat with your knees bent and feet flat on the floor. Hold the kettle bell centered over your body with both hands in a way that your thumbs hook through the handle and your fingers can grab the handles from the sides. Raise and maintain your hips off the floor, forming a slanting bridge with your body. Extend your arms holding the kettle bell straight away from your body. Now rotate your shoulders above your head, keeping your arms straight, so that the handle of the kettle bell touches the floor above your head. Bring the kettle bell back and maintain tension until the kettle bell reaches above the abdomen while maintaining your hips upward the entire time. Without rest, rotate the shoulders to bring the handle to the floor above your head once again. Repeat 10 times. This will work the muscles mentioned above. A variation on this is to bend at the elbow; this will eliminate the triceps from working and focus more on the back. Recommend 3 sets of 10 repetitions.

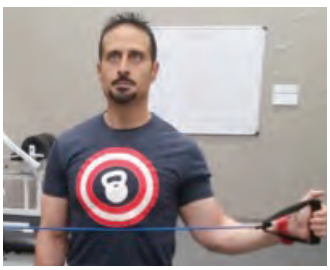

The Plank

This exercise strengthens the entire core region. Lie face down on the mat, supporting your body weight with only your toes, elbows and forearms touching the ground. Your body will be held straight, with your spine and neck linear and neutral. You may choose to keep your hands held together. Maintain tension in your midsection and do not allow your abdominals to sink towards the floor or for the hips to be out of line with the body. Another variation of this exercise includes balancing face down on only your toes and palms of your hands on the mat, with your arms extended straight to support your body weight. Hold this position for 30 seconds.

Rotator Cuff Exercises

This exercise will use resistance tubing, which includes handles. One end of the rubber tubing should be secured to a non-moving object or surface, such as a door hinge, heavy table or fixed furniture. Tubes come in a variety of resistance strengths, which should correspond with the participant's strength and ability, as appropriate. The participant will be standing during this exercise; the rubber tubing should be secured at about waist level. For exercising the external rotation motion, hold the free handle of the rubber tubing with the hand furthest from the secured end. The tubing should extend in front of your body. With the elbow next to your side, use a rotating motion to pull the handle across your body horizontally. The elbow should remain stationary, with only the forearm and hand moving across the body in a perpendicular

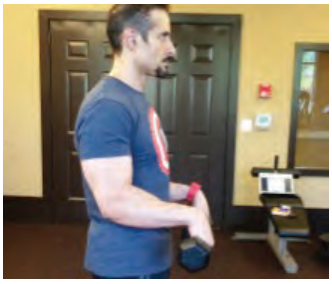
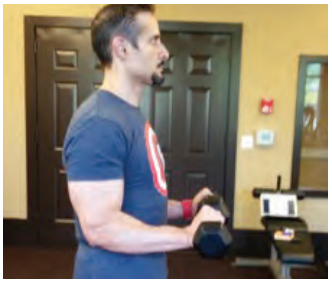
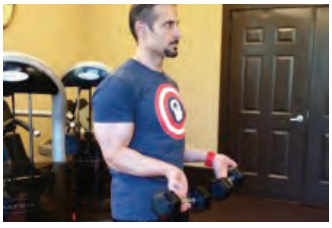


fashion. You will feel the tension increase the further you pull. For exercising the internal rotator cuff muscles, switch to the opposite hand and perform the same motion, moving internally across your body. Alternate both internal and external rotation with each hand, performing 3 sets of 10 repetitions per arm.

For another shoulder and rotator cuff exercise, hold one lightweight dumbbell in each hand while standing, with palms facing inwards towards your body. Keeping the arms straight and extended in front of you, raise your arms up to shoulder level, keeping wrists neutral. Return to starting position. Repeat this exercise for 3 sets of 10 repetitions.

Forearm Exercises


To work the top side of your forearms (wrist extensors), using lightweight dumbbells, appropriate to your strength, hold one dumbbell in each hand. Elbows and upper arms should be relaxed by your sides, and forearms should be extended in front of your body, at a 90-degree angle. Palms should be facing down toward the floor. Rotate your hands and wrists gently downward and back up. Perform 3 sets of 10 repetitions.

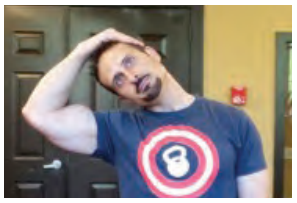
To exercise the internal part of the forearm muscles, hold one lightweight dumbbell in each hand and relax arms by your sides with palms facing out in front of your body. Rotate the hands and wrists upwards gently and back down. Perform 3 sets of 10 repetitions of this exercise as well.

Between Patients—No Equipment Necessary

Tricep Stretch

Extend one arm above your head, elbow bent, with the forearm extending back behind the neck and trapezius muscle. Use your opposite hand to stabilize this stretch by gently bringing your bent elbow deeper into the stretch. Switch arms and hold this stretch on each side for 30 seconds.

Neck Stretch



With shoulders neutral, gently tilt your head at the neck, bringing your ear over the adjacent shoulder. You may use the hand on that side to stabilize your head in this stretch, gently without pulling. To get an even deeper stretch, hold your opposite arm at your side, with the wrist flexed upward, palm facing down. Hold this stretch for 20 to 30 seconds; repeat on the other side.

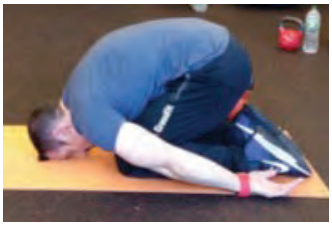

Forearm Stretch

Stand with one arm extended straight in front of you at shoulder level. Rotate the wrist downwards so that your palm is facing your body. You may use your other hand to gently extend your wrist rotation in this stretch. Then, rotate the wrist upwards, so that your palm is facing away from your body. Hold this stretch in a similar way. Switch to stretch your opposite forearm, holding each stretch for 20 to 30 seconds.

Pectoral Muscle Stretch (Chest)

Stand centered in a doorway. Extend your arms out to your sides, bent at the elbows with palms facing forward. Hold on to the sides of the doorway in this position, keeping elbows parallel to the floor. Holding your body steady, lean forward into the stretch.

Stretch Exercises




Child's Pose

This stretch will focus on your full back and shoulders. Kneel on the mat and place your hands about two feet in front of your knees, shoulder width apart, palms down, on the mat. Bring your hips back to rest above your ankles, keeping your arms extended overhead on the mat. You may choose to rest your forehead on

the mat as well. In another variation, to focus this stretch mostly on the back and less on the shoulders, you may bring your arms to your sides with your palms facing up behind you.

Conclusion

The shoulders, lower back, rotator cuffs and core muscles are most affected by poor posture. The best way to help alleviate chronic pain is to maintain a healthy lifestyle, including exercise, hydration, nutrition and sleep. These exercises can be incorporated easily into any lifestyle and adjusted according to personal preferences and physical abilities. Developing supporting muscles can help prevent muscular imbalances and undue joint stress over time.

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REFERENCES

1. Ghaderi F, Jafarabadi MA, Javanshir K. The clinical and EMG assessment of the effects of stabilization exercise on nonspecific chronic neck pain: A randomized controlled trial. J Back and Musculoskeletal Rehab BMR 2016:1-9. doi:10.3233/bmr-160735.
2. Gordon R, Bloxham S. A systematic review of the effects of exercise and physical activity on non-specific chronic low back pain. Healthcare 2016;4(2):22 doi:10.3390/healthcare4020022.
3. Hilde G, BK. Effect of exercise in the treatment of chronic low back pain: a systematic review, emphasizing type and dose of exercise. Physical Therapy Reviews 1998;3(2):107-117. Doi: 10.1179/ptr.
4. Kai Y, Gotoh M, Takei K, Madokoro K, Imura T, Murata S, Shiba N. Analysis of scapular kinematics during active and passive arm elevation. J Phys Ther Sci J Physical Therapy Science 2016;28(6):1876-1882. doi:10.1589/jpts.28.1876.
5. Kell R. Exercise as an intervention for non-specific low back pain. Physical Therapy Reviews 2010;15(1):36-37. doi:10.1179/174328810x12647087218758.
6. Kim T, An D, Lee H, Jeong H, Kim D, Sung Y. Effects of elastic band exercise on subjects with rounded shoulder posture and forward head posture. J Phys Ther Sci J Physical Therapy Science 2016;28(6):1733-1737. doi:10.1589/jpts.28.1733.
7. Swinkels A, Cochrane K, Burt A, Johnson L, Lunn T, Rees AS. Exercise interventions for non-specific low back pain: an overview of systematic reviews. Physical Therapy Reviews 2009;14(4):247-259. doi:10.1179/174328809x452917.
8. Untitled photo of dumb bells. Retrieved Sept. 10, 2016. from http://www.ebay.co.uk.
9. Untitled photo of kettle bells. Retrieved Sept. 10, 2016, from http://www.breakingmuscle.com.
10. Untitled photo of resistance tubing. Retrieved Sept. 10, 2016, from http://www.fitness1st.com.
11. Untitled photo of yoga mat. Retrieved Sept. 10, 2016, from http://www.texasrockgym.com.

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What Grows on Your Implants?

Erica Lavere, D.D.S.; Nicola Alberto Valente, D.D.S., M.S.; Sebastiano Andreana, D.D.S., M.S.

ABSTRACT

What happens to implants after we introduce them into the oral environment? Do they keep their sterility, or are they colonized by bacteria? How fast is the surface of the implants colonized by the bacteria, and which bacteria will colonize it? Do healthy implants have the same microbial populations as diseased implants? What's the role of the remaining teeth in the contamination of the dental implants? This short review of the literature will give a general overview of critical concepts in terms of peri-implant microbiology, based on the most important evidence in the literature.

Implant dentistry is a prominent topic within dentistry today. Implants are often the treatment of choice when dealing with the edentulous or partially edentulous mouth. Technology within this subject is growing, and the number of implants placed annually is growing exponentially. Despite the high rate of success, it is possible for the area around an implant to become infected, thus leading to potential failure due to inflammation-related bone resorption. The infection and consequent inflammation around an osseointegrated implant is called peri-implant mucositis, when affecting only the soft tissues, or peri-implantitis, when the disease affects the surrounding supporting bone.[1]

Microorganisms are the major players in initiation of this disease. The accumulation of gram-negative anaerobic bacteria is what causes the initiation of the breakdown of the supporting tissues of an implant.[2] There are always bacteria in the oral cavity regardless if it is in a healthy or diseased state. In a healthy state, at any site in the oral cavity, there are pioneer bacteria. Pioneer bacteria are the first colonizers to a specific surface in the oral cavity. They are the first bacteria to engage receptors to adhere to the surface.

The pioneer organisms provide a favorable environment and produce metabolic signals to attract future adhering bacteria.[3] As more bacteria attach, a biofilm is created. The signals and messages that bacteria use to communicate between one another is a phenomenon called quorum sensing. Quorum sensing is defined as the regulation of gene expression in response to fluctuations in cell population density.[4]

Bacteria use autoinducers (hormone-like molecules) to communicate with each other. The bacteria detect, release, produce and respond to these molecules. When there is a disruption in the biofilm, the bacteria use this technique to regulate themselves and their interaction.[5] They also regulate the number of certain bacteria in the environment when this occurs. This is most likely when the bacterial community changes from gram-positive to gram-negative in the case of peri-implantitis. This communication process has been found in gram-negative bacteria, which are the conductors of specific events in the infection process and formation of biofilms.[6]

Quorum sensing has also been found to be used by gram-positive bacteria. The autoinducers that gram-negative bacteria use are acylated homoserine lactones, and gram-positive micro-organisms use processed oligo-peptides to communicate.[4] In a study done on periodontal pathogens that produce quorum sensing, signal molecules *P. intermedia*, *F. nucleatum*, and *P. gingivalis* were all found to produce these autoinducer type signals.[7] These latter mentioned bacteria, responsible for the development of periodontal disease, are also prevalent in peri-implantitis cases. This provides evidence that peri-implant microflora use quorum sensing to communicate with each other and that this technique is most likely responsible for the change from implant health to disease.

As soon as the implant is introduced to the oral cavity, salivary proteins bind to it and a pellicle is formed. This allows the pioneer organisms to bind to the implant and secondary colonizers follow. Examples of these primary organisms are members of the streptococcus family such as *S. sanguinis*.[8] Once these bacteria adhere, a biofilm is able to form on the implant surface. Biofilms are composed of numerous different bacteria. They are polymicrobial, with a key feature being that each different type of bacteria requires space and nutrients to thrive. In a healthy state, the bacteria within this biofilm are in equilibrium with each other. A disturbance within this equilibrium can lead to a diseased state by allowing a more dominant type of bacteria to persistently grow.[9]

The properties of the implant surfaces, such as surface energy and roughness, allow the bacteria to adhere and, because of surface roughness, natural forces, such as saliva flow, cannot remove the bacteria from the surface irregularities of the implant.[10]

There are different types of bacteria found around healthy and diseased implants.

Microflora at Healthy Sites

Gram-positive bacteria are the microflora associated with healthy implant sites, and *S. sanguinis* is one of the primary organisms found in this population of microorganisms. A study done on the adhesion of this microorganism to rough titanium surfaces demonstrated how readily it adhered to the surface.[10] Another study analyzed the microorganisms around implants in patients who were healthy, not smokers, and did not have compromised periodontal health. The micro-organisms mainly found in this study were gram-positive cocci, *B. fragilis*, and *Prevotellaceae*.[11]

A study completed at Harvard School of Dental Medicine described the microflora detected around successfully osseointegrated implants. The most prevalent species found were oral strep-

TABLE 1.
Healthy Implants*

Author	Bacterial Species Detected	Major Findings
Shahabouee et al.[11]	*F. nucleatum* and *B. Fragilis*, Prevotellaceae, *P. gingivalis* and Gram-positive cocci	The susceptibility of implants to inflammation is the same as the susceptibility to teeth.
Mombelli et al.[19]	*Gram-positive facultative cocci*	Bacteria that colonize newly set, healthy implant sites were found.
Shibli et al.[17]	*V. parvula, S. gordonii, S. intermedius* and *F. periodonticum*	With ligature-induced peri-implantitis, time and periodontal microorganisms affect all surfaces in the same way after 2 months.
Kocar et al.[18]	*Oral streptococci*	Oral streptococci were the most prevalent type of bacteria found at healthy implant sites; in partially edentulous patients there were 4 periodontopathogenic bacteria found around implants.
Lee et al.[12]	*Oral streptocci, Capnocytophagae, V. parvula, P. micros,* and *F. nucleatum*	Microbiota on teeth influence the peri-implant microbiota.

*most significant studies

tococci, *capnocytophagae*, *V. parvula*, *P. micros*, and *F. nucleatum*.[12] A review of different biofilms associated with implants suggested that members of the yellow and purple complexes are associated with peri-implant health.[13] Yellow complexes are made of the streptococcus family, which are primary colonizers, and purple complexes consist of *A. odontolyticus* and *V. parvula*.[14] Oral streptococci species were the type of bacteria found most frequently at healthy implant sites.[10,12,15-19]

Microflora at Diseased Sites

The presence of large quantities of gram-negative bacteria is the culprit for peri-implantitis infections. In peri-implantitis, there is a greater number of red and orange complex microorganisms. Red complex organisms consist of *P. gingivalis*, *T. denticola* and *T. forsythia*. Orange complex organisms consist of *P. intermedia* and *F. nucleatum*.[14] An early study from Rams et al.[20] showed that, while the microbial population surrounding healthy implants had high rates of coccoid gram-positive cells and few spirochetes, there was an inversion tendency with increasing probing depth (PD) and gingival inflammation.

In most of the human studies assessing the peri-implant microbiota, there is a consistently high incidence of *Prevotellaceae*

TABLE 2.
Diseased Implants*

Author	In Vivo	Major Findings
Casado, et al.[36]	*A. actinomycetemcomitans, P. gingivalis, P. intermedia, T. forsythia, T. denticola*	*A. actinomycetemcomitans, P. gingivalis, P. intermedia, T. forsythia, T. denticola* are not strictly related to peri-implantitis sites.
Mombelli & Decaillet[31]	*T. forsythia, C. ochracea, Fusobacterium* spp., *P. intermedia, S. aureus*, peptostreptococci	Peri-implant infections are linked to microbiota similar to periodontitis, but some are unique to peri-implant disease, including peptostreptococci and staphylococci.
Heuer et al[15]	*A. actinomycetemcomitans* and *P. gingivalis*	"Cellular adherence of peri-implant tissue by means of hemidesmosoma, actin filaments and microvilli reduces the risk of formation of anaerobic subgingival pockets."
Shibli A et al.[17]	*P. gingivalis, T. forsythia* and *T. denticola* and *P. nigrescens*	With ligature-induced peri-implantitis, time and periodontal microorganisms affect all surfaces in the same way after 2 months.
Leonhardt et al.[23]	*A. actinomycetemcomitans, S. epidermis, P. gingivalis, P. intermedia, K. pneumoniae, P. nigrescens, Candida* spp.	Some of the species found in this study are resistant to certain antimicrobial treatments and this should be considered when treating peri-implantitis.
Costerton et al.[34]	*S. aureus*	Biofilms play a role in antimicrobial resistance and resistance between virulence genes takes place.
Rasperini et al.[37]	*Actinomyces* species and *V. parvula*	There were no differences between bacterial colonization in the two implant materials studied.
Augthun M, Conrads G[22]	*P. intermedia, P. buccae, P. oralis, P. melaninogenica, P. denticola, A. actinomycetemcomitans, F. nucleatum,* Capnocytophage *spp, E. corodens*	Bacteria from Bacteroidaceae family and actinomycetemcomitans were found most frequently, and where there were high numbers of these bacteria present there were advanced periodontal lesions.
Albertini et al.[24]	*P. gingivalis, T. forsythia, P. intermedia, T. denticola, P. aeruginosa, C. albicans* and *S. aureus*	The implant surface may be colonized with pathogens different from periodontal bacteria. Opportunistic pathogens such as P. aeruginosa, S. aureus and C. albicans may be associated with implant failure.
Botero et al.[25]	*Fusobacterium* spp, *P. gingivalis, P. intermedia, P. nigrescens, Campylobacter spp, Eubacterium* spp., *E. corrodens, gram-negative enteric rods*	The subgingival microbiota in peri-implant lesions showed high levels of periodontopathic bacteria and superinfecting bacteria compared to healthy stable implants. The role of superinfecting bacteria in the pathogenesis of peri-implant lesions needs further investigation.

*most significant studies

(*P. intermedia*, *P. buccae*, *P. oralis*, *P. melaninigenica*, *P. denticola*, *P. nigriscens*), *Porphyromonas gingivalis*, *Fusobacterium nucleatum*, *Tanneralla forsythia* and *Treponema denticola* in diseased implants, as well as coccoid gram-positives cells in healthy implants.[21-27] These findings have been confirmed by experimental studies on ligature-induced peri-implantitis on animal models, in which the characteristic shift in the microbial flora has been confirmed.[28-30]

High numbers of *A. actinomycetemcomitans* and *E. corrodens* can be also found at diseased implant sites.[13] However, the finding of *A. actinomycetemcomitans* is inconstant within the findings of the different studies. This might be due to the individual composition of the oral microflora of the patients included in the studies.[22,23]

When local factors in a biofilm change, favoring the growth of bacterial pathogens and virulence factors, the initial progression to peri-implantitis is believed to be established.[31] From the analysis of several studies concerning the microflora that adhere to and is found around implants and implant material, there is a trend that can be observed in the type of gram-negative bacteria that were reported in the above studies. Staphylococcus species were found in two different studies to adhere readily to titanium surfaces.[32,33] Consistent with these studies, different staphylococcus species were found around diseased implant sites in in-vivo studies.[23,34]

The presence of gram-positive species is not surprising considering the "initiator" function that these bacteria have on the establishment of a pathogenic biofilm. Species such as *P. gingivalis* and *P. intermedia* were almost always found in studies focusing on diseased peri-implant tissues.[17,21-23,35,36] *A. actinomycetemcomitans* was also a frequent, although inconstant, finding among the studies analyzed.[15,22,23,36,37] *T. denticola*, Fusobacterium spp, and *T. forsythia* were found in some studies as well.[17,31,36]

The colonization of the implant's surface by the microbial species starts as soon as 30 minutes after implant placement. And the bacteria load stays the same for the first week. Between the first and 12th week after surgery, the bacterial load becomes significantly higher for several species, such as *P. gingivalis*, *T. forsythia* and *T. denticola*.[27] At 12 months, the bacterial load appears to be significantly higher for some species, in particular, *T. forsythia* and, to a lesser extent, *P. gingivalis*.[26]

In conclusion, the bacterial composition of the peri-implant biofilm closely resembles that of the neighboring teeth, which implies that the microbial flora on natural teeth is the reservoir for the biofilm formation around implants. In the same way, the qualitative composition of the biofilm microflora in peri-implantitis resembles that of periodontitis, which explains why patients with active periodontal disease are at higher risk for peri-implantitis.

However, a few clinical studies on humans have shown the presence of non-periodontal microbial species, such as *P. seudomonas aeuruginosa*, *Candida albicans* and *Staphylococcus aureus*, around diseased implants.[23,24] In particular, Albertini et al.[24] found, in two patients out of 33, complete absence of periodontal microorganisms commonly found in periodontitis. A different study, aimed at assessing the presence of certain viruses in peri-implantitis sites, has found a high prevalence of human cytomegalovirus and Epstein-Barr virus in subgingival plaque of peri-implant diseased sites, suggesting a possible pathogenic role of these viruses in peri-implantitis.[38]

Conclusions

Peri-implantitis can be prevented in most cases. Maintaining good oral hygiene, mainly plaque control, is an important measure that can be taken to prevent this infection. This was shown in a study that displayed that patients with good oral hygiene tended to keep their implants longer.[39] Recall visits are crucial for peri-implant infection prevention. These visits should include examination, re-evaluation, diagnosis, motivation, reinstruction, instrumentation, treatment of infected sites, polishing, fluoridation and determining recall interval, as stated in this study. The use of an antiseptic mouthwash daily after placement of an implant is another proven effective preventative treatment of peri-implantitis.[40] Mouthrinses like chlorhexidine and those that contain essential oils increase the effectiveness of plaque control as well. Chlorhexidine often has negative side effects, like enamel

staining. Using an essential oil mouthrinse does not demonstrate the same side effects.[41]

Taking the proper preventative measures to prevent peri-implantitis is crucial because the implant is inevitably surrounded by a bacteria-containing biofilm. Due to the presence of the diverse amount of bacteria in the oral cavity and the saliva that the implant is immediately exposed to, a biofilm will always form on and around the implant. Maintaining proper oral hygiene is a way to keep the biofilm around the implant in good equilibrium so that disruption does not occur. And without disruption, there will not be a shift from the gram-positive harmless bacteria to the disease-causing gram-negative bacteria.

Queries about this article can be sent to Dr. Valente at nicolaal@buffalo.edu.

REFERENCES

1. Mombelli A, Lang NP. The diagnosis and treatment of peri-implantitis. Periodontology 2000 1998;17:63-76.
2. Mombelli A. Etiology, diagnosis, and treatment considerations in peri-implantitis. Current Opinion in Periodontology 1997;4:127-36.
3. Jenkinson HF. Oral microbial communities in sickness and in health. Trends in Microbiology 2005;13(12):589-95.
4. Miller MB, Bassler BL. Quorum sensing in bacteria. Annual Review of Microbiology 2001;55:165-99.
5. Waters CM, Bassler BL. Quorum sensing: cell-to-cell communication in bacteria. Annual Review of Cell and Developmental Biology 2005;21:319-46.
6. Hentzer M, Eberl L, Nielsen J, Givskov M. Quorum sensing:a novel target for the treatment of biofilm infections. BioDrugs:Clinical Immunotherapeutics, Biopharmaceuticals and Gene Therapy 2003;17(4):241-50.
7. Frias J, Olle E, Alsina M. Periodontal pathogens produce quorum sensing signal molecules. Infection and Immunity 2001;69(5):3431-4.
8. Heuer W, Elter C, Demling A, et al. Analysis of early biofilm formation on oral implants in man. Journal Oral Rehabilitation 2007;34(5):377-82.
9. Kreth J, Zhang Y, Herzberg MC. Streptoccal antagonism in oral biofilms: streptoccus sanguinis and streptococcus gordonii interference with streptococcus mutans. Journal Bacteriology 2008;190(13).
10. Rodriguez-Hernandez, Juarex A, Engel E, Gil FJ. Streptococcus sanguinis adhesion on titanium rough surfaces: effect of shot-blasting particles. Materials in Medicine 2011;22(8):1913-22.
11. Shahabouee M, Rismanchian M, Yaghini J, Babashahi A, Badrian H, Goroohi H. Microflora around teeth and dental implants. Dental Research Journal 2012;9(2):215-20.
12. Lee KH, Maiden MF, Tanner AC, Weber HP. Microbiota of successful osseointegrated dental implants. J Periodontol 1999;70(2):131-8.
13. Lee A, Wang HL. Biofilm related to dental implants. Implant Dent 2010;19(5):387-93.
14. Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL Jr. Microbial complexes in subgingival plaque. J Clin Periodontol 1998;25(2):134-44.
15. Heuer W, Stiesch M, Abraham WR. Microbial diversity of supra and subgingival biofilms on freshly colonized titanium implant abutments in the human mouth. Eur J Clin Microbiol Infect Dis 2011;30(2):193-200.
16. Nakazato G, Tsuchiya H, Sato M, Yamauchi M. In vivo plaque formation on implant materials. Int J Oral Maxillofac Implants 1989;4(4):321-6.
17. Shibli JA, Martins MC, Lotufo RF, Marcantonio E Jr. Microbiologic and radiographic analysis of ligature-induced peri-implantitis with different dental implant surfaces. Int J Oral Maxillofac Implants 2003;18(3):383-90.
18. Kocar M, Seme K, Hren NI. Characterization of the normal bacterial flora in peri-implant sulci of partially and completely edentulous patients. Int J Oral Maxillofac Implants 2010;25(4):690-8.
19. Mombelli A, Buser D, Lang NP. Colonization of osseointegrated titanium implants in edentulous patients. Early results. Oral Microbiol Immunol 1988;3(3):113-20.
20. Rams TE, Roberts TW, Tatum H Jr., Keyes PH. The subgingival microbial flora associated with human dental implants. J Prosthet Dent 1984;51(4):529-34.
21. Mombelli A, van Oosten MA, Schurch E Jr., Land NP. The microbiota associated with successful or failing osseointegrated titanium implants. Oral Microbiol Immunol 1987;2(4):145-51.
22. Augthun M, Conrads G. Microbial findings of deep peri-implant bone defects. Int J Oral Maxillofac Implants 1997;12(1):106-12.
23. Leonhardt A, Renvert S, Dahlén G. Microbial findings at failing implants. Clinical Oral Implants Research 1999;10(5):339-45.
24. Albertini M, Lopez-Cerero L, O'Sullivan MG, et al. Assessment of periodontal and opportunistic flora in patients with peri-implantitis. Clinical Oral Implants Research 2014 doi: 10.1111/clr.12387published [Online First: Epub Date].
25. Botero JE, Gonzalez AM, Mercado RA, Olave G, Contreras A. Subgingival microbiota in peri-implant mucosa lesions and adjacent teeth in partially edentulous patients. Journal Periodontology 2005;76(9):1490-5.
26. Salvi GE, Furst MM, Lang NP, Persson GR. One-year bacterial colonization patterns of Staphylococcus aureus and other bacteria at implants and adjacent teeth. Clinical Oral Implants Research 2008;19(3):242-8.
27. Furst MM, Salvi GE, Lang NP, Persson GR. Bacterial colonization immediately after installation on oral titanium implants. Clinical Oral Implants Research 2007;18(4):501-8.
28. Hickey JS, O'Neal RB, Scheidt MJ, Strong SL, Turgeon D, Van Dyke TE. Microbiologic characterization of ligature-induced peri-implantitis in the microswine model. Journal Periodontology 1991;62(9):548-53.
29. Hanisch O, Cortella CA, Boskovic MM, James RA, Slots J, Wikesjo UM. Experimental periimplant tissue breakdown around hydroxyapatite-coated implants. Journal of Periodontology 1997;68(1):59-66.
30. Charalampakis G, Abrahamsson I, Carcuac O, Dahlen G, Berglundh T. Microbiota in experimental periodontitis and peri-implantitis in dogs. Clinical Oral Implants Research 2014;25(9):1094-8.
31. Mombelli A, Decaillet F. The characteristics of biofilms in peri-implant disease. Journal Clinical Periodontology 2011;38(s11):203-13.
32. Harris LG, Meredith DO, Eschbach L, Richards RG. Staphylococcus aureus adhesion to standard micro-rough and electropolished implant materials. J Mater Sci Mater Med 2007;18(6):1151-6.
33. Konig DP, Perdreau-Remington F, Rutt J, Stossberger P, Hilgers RD, Plum G. Slime production of Staphylococcus epidermidis: increased bacterial adherence and accumulation onto pure titanium. Acta Orthop Scand 1998;69(5):523-6.
34. Costerton JW, Montanaro L, Arciola CR. Biofilm in implant infections: its production and regulation. Int J Artif Organs 2005;28(11):1062-8.
35. Kuula H, Kononen E, Lounatmaa K, Konttinen YT, Kononen M. Attachment of oral gram-negative anaerobic rods to a smooth titanium surface: an electron microscopy study. Int J Oral Maxillofac Implants 2004;19(6):803-9.
36. Casado PL, Otazu IB, Balduino A, de Mello W, Barboza EP, Duarte ME. Identification of periodontal pathogens in healthy periimplant sites. Implant Dent 2011;20(3):226-35.
37. Rasperini G, Maglione M, Cocconcelli P, Simion M. In vivo early plaque formation on pure titanium and ceramic abutments: a comparative microbiological and SEM analysis. Clinical Oral Implants Research 1998;9(6):357-64.
38. Jankovic S, Aleksic Z, Dimitrijevic B, Lekovic V, Camargo P, Kenney B. Prevalence of human cytomegalovirus and Epstein-Barr virus in subgingival plaque at peri-implantitis, mucositis and healthy sites. A pilot study. Int J Oral Maxillofac Surg 2011;40(3):271-76.
39. Pjetursson BE, Helbling C, Weber HP, et al. Peri-implantitis susceptibility as it relates to periodontal therapy and supportive care. Clinical Oral Implants Research 2012;23(7):888-94.
40. Ciancio SG, Lauciello F, Shibly O, Vitello M, Mather M. The effect of an antiseptic mouthrinse on implant maintenance: plaque and peri-implant gingival tissues. J Periodontol 1995;66(11):962-5.
41. Stoeken JE, Paraskevas S, van der Weijden GA. The long-term effect of a mouthrinse containing essential oils on dental plaque and gingivitis: a systematic review. J Periodontol 2007;78(7):1218-28.


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Idiopathic Gingival Fibromatosis

A Case Report and Review of the Literature

Divya Khera, D.D.S.; Elizabeth Philipone, D.M.D.; Richard Yoon, D.D.S.

ABSTRACT

Gingival fibromatosis is a rare condition that is characterized by progressive, varying expansion of submucosal gingival connective tissue. The authors present the case of an adolescent patient with unilateral idiopathic gingival fibromatosis, along with a literature review of the condition and treatment.

There are few case reports in the recent dental literature covering gingival fibromatosis, a rare benign condition characterized by a slow, progressive enlargement in the connective tissue elements of the gingival corium.[1] Gingival enlargements originate from local irritation, certain medications,[2] genetic syndromes and/or inheritance.[3,4]

Hereditary gingival fibromatosis is prevalent in 1 in 750,000 people and is phenotypically equal among males and females.[5,6] A diagnostic distinction is made between hereditary gingival fibromatosis and idiopathic gingival fibromatosis (IGF), the latter being a diagnosis of exclusion,[7] with no family history and no outward cause. Clinically, the gingival enlargement is normal in color, of firm consistency and varies in degree of growth.[6] The authors present the clinical, radiographic and histopathological key features of a patient with IGF, along with a review of the dental literature.

Case Description

A 10-year-old black male in excellent health was seen in December 2013 by the oral pathologist for a "gingival swelling." During the intraoral examination, palpation revealed the presence of a firm, pink, well-perfused, right unilateral, painless, non-erythematous gingival enlargement. The upper and lower right oral mucosa was pink, well-perfused, with unilateral, painless, non-erythematous gingival enlargement. Although the patient was aware of the mass, questioning indicated he had no associated symptoms.

A panoramic radiograph was performed and revealed a slightly hyperostotic border of the right mandible, with no visual expansion (Figure 1). An aspiration was performed to determine if the enlargement was soft tissue or bony in origin. A tentative diagnosis of hyperplastic gingiva was made, and the patient was asked to return for normal periodic oral hygiene maintenance.

Three years later, the patient, still in excellent health, presented for a "gingival swelling" with associated sensitivity upon toothbrushing and mastication. Intraoral examination and pal-

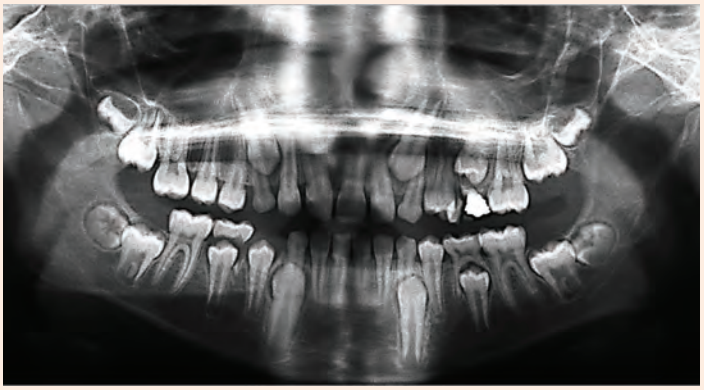

Figure 1. Panoramic radiograph of child in mixed dentition with no pathology or abnormality noted.

Figure 2. Clinical photo demonstrating pink, well-perfused, unilateral, buccal-lingual gingival overgrowth on upper and lower right quadrants. Positive, thick voluminous accretions noted throughout, particularly on posterior occlusal surfaces and lingual smooth surfaces.

pation revealed the presence of a firm, pink, well-perfused, right unilateral, painless, non-erythematous gingival enlargement and thick voluminous accretions on the smooth and occlusal surfaces of the permanent teeth (Figure 2). Follow-up periapical radiographs revealed no pathology or abnormalities.

Given the patient's history, an incisional biopsy was performed for histopathology. The gingival enlargement histologically proved to be gingival fibromatosis (Figure 3) or IGF, based on lack of family history. Neither palisading nor streaming of cells (indicative of neuromas or neurilemomas) nor sparsely distributed neuronal axons (typical of neurofibroma) were observed in the tissue. The surface epithelium appeared to be a benign-appearing, stratified, squamous epithelium with overlying keratotic material and thin, elongated rete pegs.

Subgingival scaling and prophylaxis were completed. The authors believe that an additional transitory source of discomfort may have been the normal eruption of the second permanent molars. The family declined surgery to remove excess gingival tissue, and periodic oral hygiene maintenance follow-up was made.

Review of the Literature

Most of our understanding of gingival fibromatosis originates from case reports,[4,6,7] and treatment often depends upon the degree of gingival enlargement. With respect to hereditary gingival fibromatosis, the majority demonstrate an autosomal dominant mode of inheritance,[8-10] with differences in disease extent and presentation concomitant with a syndrome or an isolated finding.[8,11] Mutations on chromosome 2, resulting in early termination of a protein, have been identified as one cause.[12-16] Alternate chromosomes have since been identified in syndromic forms.[11]

In syndromic forms, the most common characteristic of hereditary gingival fibromatosis is hypertrichosis.[6] Associated findings include syndromic deafness, growth hormone deficiency, hyper-

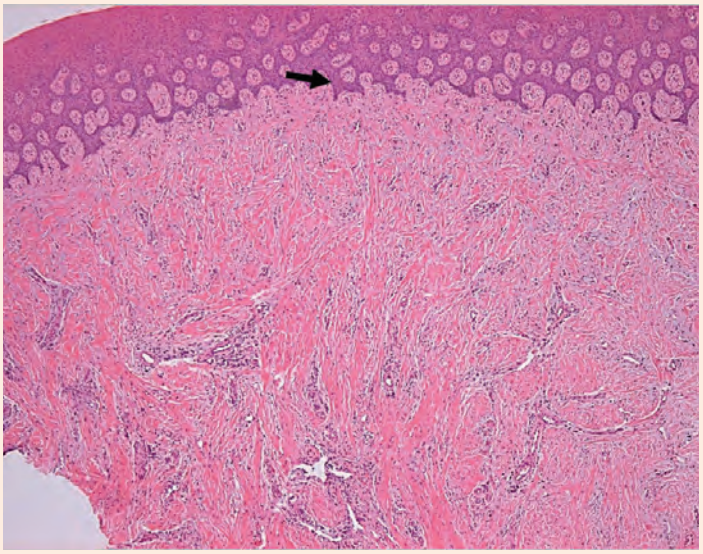

Figure 3. Histology (40x micrograph) revealed densely collagenous fibrous connective tissue sparsely populated with fibroblasts. Surface epithelium was observed to be benign-appearing, stratified, squamous epithelium with overlying keratotic material and thin, elongated rete pegs.

TABLE 1.
Syndromes Associated with Gingival Fibromatosis[4,6,8,11]

Syndrome or Condition	Inheritance	Summary of Findings
Gingival Fibromatosis	AD/AR	Hypertrichosis, intellectual disability, muscular hypotonia
Zimmerman-Laband	AD	Syndactyly, hypoplasia of the nails and terminal phalanges, joint hyperextension, hepatosplenomegaly
Cross	AR	Microphthalmia, hypopigmentation, intellectual disability, cloudy corneas, athetosis
Juvenile Hyaline Fibromatosis	AR	Multiple subcutaneous hyaline fibromas, osteolysis of terminal phalanges, osteolytic/osteoclastic skeletal lesions, sclerodermiform atrophy, stunted growth, premature death
Rutherford	AD	Delayed tooth eruption, corneal dystrophy, intellectual disability, dentigerous cysts
Ramon	AR	Cherubism, intellectual disability, epilepsy, hypertrichosis, stunted growth, juvenile rheumatoid arthritis, ocular abnormalities
Jones	AD	Progressive deafness
Costello	AD	Delayed development, loose skin folds, large mouth, arrhythmias and hypertrophic cardiomyopathy, papillomas
Tetra-amelia	AR	Absence of all four limbs, underdeveloped lungs
Prune-belly	X-linked	Absence of abdominal muscles, cryptorchidism, urinary tract abnormalities, club foot, facial dimorphism
Neurofibromatosis Type I	AD	Café au lait, lisch nodules, hypertension, stunted growth, macrocephaly
Donohue	AR	Hirsutism, acanthosis nigricans, large mouth, thick lips, hepatic cholestasis, lack of lymphatic tissue, distended abdomen
Schinzel-Giedion	AR	Hypertrichosis, alacrima, abnormal fontanelles, macroglossia, hypertelorism, skeletal abnormalities, mental retardation
Cowden	AD	Hamartomas, neoplasms
Goltz-Gorlin	AD	Form of ectodermal dysplasia, odontogenic keratocysts, neoplasms
Tuberous Sclerosis	AD	Hamartomas of brain, kidney, skin, heart, eyes, mental retardation
Francois	AD/AR	Xanthomatous nodules, corneal dystrophy

*AD autosomal dominant ; **AR autosomal recessive

telorism, intellectual disability, supernumerary teeth and generalized aggressive periodontitis. Table 1 presents syndromes associated with gingival fibromatosis and include Zimmerman-Laband, Cross, juvenile hyaline fibromatosis, Rutherford, Ramon, Jones, Costello, Tetra-amelia, Prune-belly and neurofibromatosis type 1.[4,6,8,11]

Table 2 summarizes 21 reported cases of both hereditary (non-syndromic) gingival fibromatosis and IGF, as well as dental treatment performed. Clinically, both hereditary and IGF range from localized (maxillary tuberosities and labial gingiva around the mandibular molars) to the more common generalized presentation (bilateral, affecting labia, lingual and palatal gingiva).[3,6] Typically painless, the gingival tissues are normal in color, with a firm, stippled consistency.[1,11] While plaque build-up may contribute to gingival inflammation and enlargement, the severity of gingival fibromatosis is dependent upon the penetrance of the associated gene.[8,11,17] Pseudopockets and periodontal issues are a derivative of an inability to maintain oral hygiene, as radiographically the alveolar bone remains unaffected.[11,18]

Gingival enlargement results from an increase in extracellular matrix.[4] Histologic section usually shows hyperkeratotic epithelium with elongated rete ridges and increased parallel collagen fiber bundles with few fibroblasts. Increased presence of fibroblasts suggests an increased chance of recurrence. Inflammatory cells may be seen in areas of pseudopockets but are local findings.[11,19]

Gingival enlargement is graded on extent and degree: grade zero (none); grade one (interdental papilla alone); grade two (papilla and marginal gingiva); grade three (covering greater than three-quarters of the crown).[19,20] Growth prominence occurs during transitional exchange periods of the dentition, but can occur with primary dentition eruption as well, with little to no growth seen in adolescence.[5,21] In a rare case, gingival fibromatosis was present at birth; however, it appears the presence of teeth is essential given the noted changes during eruptive periods. Edentulous areas demonstrate a recession of the condition.[6,22] Gingival enlargement can extend up to the mucogingival junction and cause delayed eruption, ectopic eruption, overretained primary teeth, difficulty in mastication, and/or changes in facial appearance (e.g., lip protrusion).[6,11,23] Varying degrees of severity have both esthetic and functional consequences.

Minimal enlargement is treated with scaling, prophylaxis and oral hygiene maintenance with periodic follow-up.[6,7] Advanced enlargement requires removal of excess tissue, while in the most severe cases, in conjunction with aggressive periodontitis, extractions and excision of tissue may be required.[6,24] Removal of bulk tissue involves gingivectomy (external or internal bevel) with gingivoplasty, apically positioned flaps, electrocautery or diode lasers.[6,25-27,29]

Treatment involves minimizing the risk of recurrence and interference with mastication, speech, primary tooth retention and psychological ramifications.[6] Recurrence tends to occur in children and adolescent patients. Consequently, the recommenda-

TABLE 2.
Tabulation of 21 Cases of Gingival Fibromatosis

Author	Diagnosis	Age-Sex	Clinical Findings	Treatment
Ramanaryan[4]	IGF	13-F	Lip incompetence, difficulty in mastication	Gingivectomy
Meera[30]	IGF	10-F	Lip incompetence, altered tongue position	Gingivectomy
Shetty[31]	IGF	13-F	Gingiva covering incisal third, mobility lower incisors	Gingivectomy
Jadhav[32]	IGF	30-F	Grade III mobile teeth, aggressive periodontitis	Extraction of Grade III mobile teeth, planned gingivectomy
Shetty, Gupta[33]	IGF	19-F	Grade III mobile teeth, aggressive periodontitis	Scaling and root planing, systemic anti-microbial therapy (amoxicillin TID and metronidazole BID x 7 days), extraction of mobile teeth, gingivectomy
Jayachandran[34]	IGF	30-F	Mobile teeth	Gingivectomy
Millet[35]	IGF	65-F	Mobile teeth, malpositioned teeth, difficulty in mastication	Extraction of mobile teeth + gingivectomy
Martelli[36]	IGF	10-M	Delayed eruption	Gingivectomy, chlorohexidine .25% BID x 14 days
Martelli[36]	IGF	30-M	Difficulty in mastication	Gingivectomy
Ganpat, Pol[37]	IGF	14-F	Delayed eruption, clinically submerged teeth	Gingivectomy
Tripathi, Dete[38]	HGF	18-F	Difficulty in mastication	Gingivectomy
Michaud, Patel[39]	HGF	57-M	Difficulty in speech, mobile teeth, bony exostoses	Extraction, gingivectomy, ostectomy, alveoplasty
Bansal[3]	HGF	17-F	Delayed eruption of permanent dentition, malpositioned teeth	Gingivectomy
Mohan[40]	HGF	22-M	Difficulty in mastication, syndactyly of middle and ring finger	Gingivectomy
Aghili[41]	HGF	9-F	Excessive overjet, protrusion of upper teeth	Orthodontics with repeat gingivectomy
Khan[19]	HGF	15-M	Tissue covering crowns of teeth	Gingivectomy
Arabi[42]	HGF	14-F	Tissue covering crowns of teeth	Gingivectomy, open-flap debridement
Dhadse[43]	HGF	12-F	Complete coverage of anterior dentition	Gingivectomy
Camilotti[25]	HGF	7-F	Anterior open bite, lip incompetence, complete coverage of teeth	Gingivectomy
Zhou[44]	HGF	28-F	Difficulty in speech and mastication	Scaling and root planing; gingivectomy
Chaurasia[23]	HGF	10-F	Difficulty in speech, mastication, overretained primary teeth	Planned gingivectomy

* IGF Idiopathic Gingival Fibromatosis; **HGF Hereditary Gingival Fibromatosis

tion for surgical intervention is upon eruption of the permanent dentition.[5,6,28,29] In this case, the patient did not want to pursue gingivectomy and gingivotomy at this time.

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REFERENCES

1. Zegarelli EV, Kutscher AH, Lichtenthal R. Idiopathic gingival fibromatosis. Am J Dig Dis 1963;8:782-8.
2. Khera P, Zirwas MJ, English JC III. Diffuse gingival enlargement. J Am Acad Dermatol 2005;52:491-9.
3. Bansal A, Narang S, Sowmya K, Sehgal N. Treatment and two-year follow-up of a patient with hereditary gingival fibromatosis. J Indian Soc Periodontol 2011;15(4):406-9.
4. Ramnarayan BK, Sowmya K, Rema J. Management of idiopathic gingival fibromatosis: report of a case and literature review. Pediatr Dent 2011;33:431-6.
5. Fletcher J. Gingival abnormalities of genetic origin: a preliminary communication with special reference to hereditary generalized gingival fibromatosis. J Dent Res 1966;45:597-612.
6. Coletta RD, Graner E. Hereditary gingival fibromatosis: a systematic review. J Periodontol 2006;77:753-64.
7. Ko YK, Farr JB, Yoon A, Philipone E. Idiopathic gingival fibromatosis: case report and review of the literature. Am J Dermatopathol 2016;38:e68-e71.
8. Hart TC, Pallos D, Bozzo L. Evidence of genetic heterogeneity for hereditary gingival fibromatosis. J Dent Res 2000;79:1758-64.
9. Goldblatt J, Singer SL. Autosomal recessive gingival fibromatosis with distinctive facies. Clin Genet 1992;42:306-8.
10. Singer SL, Goldblatt J, Hallam LA, Winters JC. Hereditary gingival fibromatosis with a recessive mode of inheritance: case reports. Aust Dent J 1993;38:427-32.
11. Häkkinen L, Csiszar A. Hereditary gingival fibromatosis: characteristics and novel putative pathogenic mechanisms. J Dent Res 2007;86(1):25-34.
12. Fryns JP. Gingival fibromatosis and partial duplication of the short arm of chromosome 2 (dup (2) (p13→p21)). Ann Genet 1999;39:54-5.
13. Shashi V, Pallos D, Pettenati MJ, et al. Genetic heterogeneity of gingival fibromatosis on chromosome 2p. J Med Genet 1999;36:683-6.
14. Hart TC, Pallos D, Bowden DW, Boylard J, Pettenati MJ, Cortelli JR. Genetic linkage of hereditary gingival fibromatosis to chromosome 2p21. Am J Hum Genet 1998;62:876-83.
15. Xiao S, Bu L, Zhu L, et al. A new locus for hereditary gingival fibromatosis (GINGF2) maps to 5q13-q22. Genomics 2001;74:180-5.
16. Hart TC, Zhang Y, Gorry MC, et al. A mutation in the SOS1 gene causes hereditary gingival fibromatosis type 1. Am J Hum Genet 2002;70:943-54.
17. Ye X, Shi L, Cheng Y, Peng Q, et al. A novel locus for autosomal dominant hereditary gingival fibromatosis, GINGF3, maps to chromosome 2p22.3-p23.3. Clin Genet 2005;68(3):239-44.
18. Bittencourt LP, Campos V, Moliterno LF, Ribeiro DP, Sampaio RK. Hereditary gingival fibromatosis: review of the literature and a case report. Quintessence Int 2000;31:415-18.
19. Khan U, Mustafa S, Saleem Z, Azam A, Khan ZA. Hereditary gingival fibromatosis diagnosis and treatment. Pakistan Oral & Dent J 2012;32(2):226-31.
20. DeAngelo S, Murphy J, Claman L, Kalmar J, Leblebicioglu B. Hereditary gingival fibromatosis—a review. Compend of Cont Educat in Dent 2007;28(3):138-43.
21. Bozzo L, de Almedia OP, Scully C, Aldred MJ. Hereditary gingival fibromatosis: report of an extensive four-generation pedigree. Oral Surg Oral Med Oral Pathol 1994;78:452-4.
22. Anderson J, Cunliffe WJ, Roberts DF, Close H. Hereditary gingival fibromatosis. BMJ 1969;3:218-9.
23. Chaurasia A. Hereditary gingival fibromatosis. Natl J Maxillofac Surg 2014;5(1):42-6.
24. Cuestas-Carnero R, Bornancini CA. Hereditary generalized gingival fibromatosis associated with hypertrichosis: Report of five cases in one family. J Oral Maxillofac Surg 1988;46:415-20.

25. Camilotti RS, Jasper J, Ferreira TB, Antonini F, Poli VD, Pagnocelli RM. Resection of gingival fibromatosis with high-power laser. J Dent Child 2015;82(1):47-52.
26. Gontiya G, Bhatnagar S, Mohandas U, Galgali SR. Laser-assisted gingivectomy in pediatric patients: a novel alternative treatment. J Indian Soc Pedod Prev Dent 2011;29(3):264-9.
27. Mavrogiannis M, Ellis JS, Seymour RA, Thomason JM. The efficacy of three different surgical techniques in the management of drug-induced gingival overgrowth. J Clin Periodontol 2006;33(9):677-82.
28. Takagi M, Yamamoto H, Mega H, Hsieh KJ, Shidoa S, Enomotos S. Heterogeneity in the gingival fibromatoses. Cancer 1991;68:2202-12.
29. Emerson TG. Hereditary gingival hyperplasia: a family pedigree of four generations. Oral Surg Oral Med Oral Pathol 1965;19:1-9.
30. Meera G, Navneet P, Ankit G. Idiopathic gingival fibromatosis. J Res Med Den Sci 2013;1(1):23-6.
31. Shetty AK, Shah J, Patil MA, Jhota KN. Idiopathic gingival enlargement and its management. J Indian Soc Periodontol 2010;14:263-5.
32. Jadhav AS, Marathe SP. Recurrent idiopathic gingival fibromatosis with generalized aggressive periodontitis: a rare case report. J Indian Soc Periodontol 2015;19: 93-5.
33. Shetty A, Gupta N, Shetty D, et al. Idiopathic gingival enlargement associated with generalized aggressive periodontitis in a 19-year-old female. J Indian Soc Periodontol 2014;18:244-8.
34. Jayachandran M, Kapoor S, Mahesh R. Idiopathic gingival fibromatosis rehabilitation: a case report with two-year follow-up. Case Rep Dent 2013;1-5.
35. Millet C, Rodier P, Farges JC, Labert N, Duprez JP. Surgical and prosthetic treatment in an elderly patient affected by unilateral idiopathic gingival fibromatosis: a case report. Gerodontology 2012;29:1185-9.
36. Martelli H, Santos SMC, Guimaraes AL, et al. Idiopathic gingival fibromatosis: description of two cases. Minerva Stomatol 2010;59:143-8.
37. Pol DG, Lobo TM, Pol SD. Idiopathic gingival fibromatosis with asymmetrical presentation and electrosurgical management. J Indian Soc Periodontol 2016;20(1):98-102.
38. Tripath AK, Dete G, Saimbi CS, Kumar V. Management of hereditary gingival fibromatosis: A 2 year follow-up case report. J Indian Soc Periodontol 2016;19(3):342-4.
39. Michaud PL, Patel A. Hereditary gingival fibromatosis with extreme ridge thickness and insufficient interarch distance: a clinical report of surgical and prosthetic management. J Prosthet Dent 2016;116(1):15-20.
40. Mohan RP, Verma S, Agarwal N, Singh U. Non-syndromic hereditary gingival fibromatosis. BMJ Case Rep 2013:1-4.
41. Aghili H, Moghadam MG. Hereditary gingival fibromatosis: a review and report of a rare case. Case Rep Dent 2013:1-4.
42. Arabi SR, Ebrahimzade Z, Mahdi K, Jourdehi SA. Hereditary gingival fibromatosis: a case report. Res J Biol Sci 2011;6(3):104-7.
43. Dhadse PV, Yeliwar RK, Pandilwar PK, Gosavi SR. Hereditary gingival fibromatosis. J Indian Soc Periodontol 2012;16(4):606-9.
44. Zhou M, Xu L, Meng HX. Diagnosis and treatment of a hereditary gingival fibromatosis case. Chin. J Dent Res 2011;14(2):155-8.


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