Cukurova Medical Journal
ÇUKUROVA ÜNİVERSİTESİ TIP FAKÜLTESİ DERGİSİ

Cukurova Med J 2017;42(4):780-784
DOI: 10.17826/cutf.326817

OLGU SUNUMU / CASE REPORT

Radiation caries in an irradiated patient with mucoepidermoid carcinoma of parotid gland

Işın tedavisi görmüş parotid bezi mukoepidermoid karsinomlu hastada radyasyon çürüklerinin oluşumu

Devika S Pillai[1], Subhas G Babu[1], Renita Lorina Castelino[1], Sachin Ram G[1], Kumuda Rao[1], Jasmine Shanthi Kamath[1]

[1]A B Shetty Memorial Institute of Dental Sciences, Nitte University, Department of Oral Medicine and Radiology, Mangalore, Karnataka

Cukurova Medical Journal 2017;42(4):780-784

Abstract
Radiation caries is a common clinical finding in patients who receive therapeutic radiation for head and neck carcinomas. Radiotherapy induced effects are most commonly seen in the oral mucosa, salivary glands, bone, teeth, and muscles of head and neck. Radiotherapy given for head and neck carcinomas cause salivary gland dysfunction and xerostomia which will further increase the risk for dental caries. There is rampant destruction of teeth, involving all surfaces. Here we present a case report of a patient with radiation caries after treatment for mucoepidermoid carcinoma of the parotid gland.
Key words: Radiotherapy, teeth, caries, carcinoma.

Öz
Radyasyon çürüğü, baş boyun kanserlerinde terapötik radyasyon uygulanan hastalarda sık görülen bir klinik bulgudur. Radyoterapiye bağlı etkiler en çok oral mukoza, tükrük bezleri, kemik, dişler ile baş ve boyun kaslarında görülür. Baş-boyun kanserleri için verilen radyoterapi, tükrük bezi disfonksiyonuna ve ağız kuruluğuna (kserostomi) neden olarak diş çürüğü riskini daha da arttırarak dişlerin tüm yüzeylerini kapsayan yaygın bir tahribata yol açar. Burada, parotis bezi mukoepidermoid karsinomu tedavisi sonrası radyasyon çürükleri olan bir hastanın olgusu aktarılmıştır.
Anahtar kelimeler: Radyoterapi, dişler, çürük, kanser.

INTRODUCTION

Salivary gland carcinomas are rare and constitutes only up to 5% of all head and neck carcinomas and 0.3% of all malignancies. Almost 75% of all salivary gland tumours occur in the parotid gland and most of them are benign. Mucoepidermoid carcinoma is the commonly seen malignancy that accounts for about 40-50% of the malignant tumours. Management of these tumours commonly involves surgical removal followed by postoperative radiation therapy[1]. Oral cavity is highly prone to adverse effects of radiation. This can be due to high turnover rate of oral mucosal cells, complex oral micro flora and constant trauma to the tissues during normal functioning. One of the earliest problems that develop in the oral cavity after radiotherapy is radiation induced caries. Irradiated patients are at increased risk of developing a rapid rampant carious process called radiation caries[2].

CASE

A twenty nine year old female patient reported to the Department of Oral Medicine and Radiology with the chief complaint of decay in the upper and lower front teeth since four years. On eliciting the history, the patient reported that she had undergone a surgery for mucoepidermoid carcinoma of the parotid gland followed by radiation therapy five years back. The radiation therapy with a divided dose of 30 Gy, once in a week was given for a period of three months. Drug allergy was non – contributory. The patient's mother and maternal

Yazışma Adresi/Address for Correspondence: Dr. Devika S Pillai, A B Shetty Memorial Institute of Dental Sciences,Nitte University, Department of Oral Medicine and Radiology, Mangalore, Karnataka
E-mail: spillaidevika@gmail.com
Geliş tarihi/Received: 20.01.2017 Kabul tarihi/Accepted: 23.02.2017

uncle had a history of thyroid carcinoma. On extra oral examination, the patient did not have any gross facial asymmetry and had a straight profile with competent lips. A scar was present in the region of left posterior triangle of the neck, of about 0.5 x 2 cm in dimension, extending anteriorly about 1cm away from the anterior border of sternocleidomastoid muscle to posteriorly, 0.5 cm below and in front of the ear, superiorly about 2cm below the lower border of the mandible and inferiorly at the level of the hyoid bone (post-surgery).

Figure 1A. Intra oral clinical photograph of the patient showing rampant carious destruction involving the mesial, distal and cervical aspect of mandibular teeth

Figure 1B. Intra oral clinical photograph of the patient showing, the rampant caries process involving the maxillary teeth, and root stumps in the region of 17 and 18

On intra oral examination, generalised physiologic pigmentation was noticed on the right and left buccal mucosa and the gingiva. A decrease in salivary flow was also noticed. Fissured tongue was present. On further examination of the hard tissues there were carious lesions involving the mesial, distal and cervical area of the mandibular anteriors and caries involving all the surfaces of the teeth in maxillary and mandibular posteriors and root stumps were present with respect to 18,17,28,38 (Figure 1A and 1B).

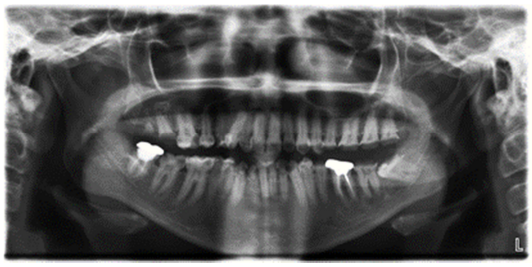

Figure 2. An orthopantamogram showing coronal radiolucency involving enamel, dentin and pulp were present in all teeth. Also, radio opacity involving the coronal part of the tooth extending till the apical one- third with respect to 36 and 47 suggestive

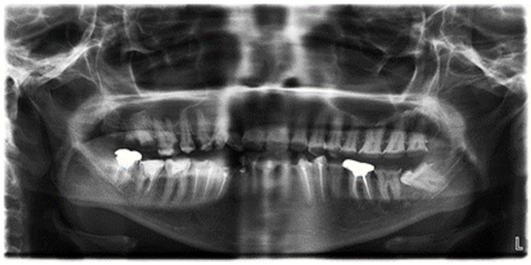

Figure 3. A post-operative orthopantamogram showing completed endodontic treatment of seven teeth

A provisional diagnosis of radiation caries was given. The patient was further subjected to an orthopantomogram to know about the extent of carious lesion. The OPG revealed normal condylar and coronoid morphology with coronal radiolucency involving enamel, dentin and pulp in all the teeth. Also, radio opacity involving the coronal part of the tooth extending till the apical one- third noted with respect to 36 and 47 suggestive of endodontic restorative material (Figure 2). The patient was then referred to the Department of Endodontics for restoration of the affected teeth. The patient was instructed to follow oral hygiene measures for xerostomia, artificial saliva (Saleva^{R}) was prescribed thrice daily for a period of 15 days. The patient was also advised to continuously sip water whenever the mouth felt dry. The patient is currently undergoing pulp space therapy of the affected teeth followed by full mouth rehabilitation in the Department of Endodontics. As the treatment is done phase wise, seven teeth have been endodontically treated and the patient has been

given appointment for continuation of the treatment in the coming days (Figure 3). The patient is kept on a regular follow- up.

DISCUSSION

Radiation caries is defined as the development of rampant caries in patients undergoing radiotherapy in the head and neck region[3]. In 1939 Del Regato observed that xerostomia was a complication of radiation therapy and postulated that this modification of saliva secretion can lead to development of caries[3]. In addition to xerostomia, an increase in the viscosity and a decrease in the pH of saliva were also noticed after radiation therapy[2]. As a consequence of irradiation, three forms of dental defects were described by Frank et al in 1965 and Baden in 1970[3] . The first type is a characteristic caries like lesion usually completely encircling the neck of the tooth[3]. Amputations of the crowns may occur due to this type of lesion. Sometimes, extensions to labial, buccal or lingual surfaces are also observed[3]. The second type of lesion begins as brown to blackish discoloration of the crown[3]. The occlusal surface of posterior teeth and incisal edges of anterior teeth wear off. The third type of lesion begins as a spot depression which spreads from incisal or occlusal edges on the labial or buccal and lingual surfaces[3]. Enamel shell is also destroyed and coronal dentin becomes partially disintegrated leaving the crown reduced to an irregularly shaped discoloured stump projecting over the gingiva[3]. Xerostomia, one of the major complications of post radiation therapy is considered as the major factor leading to the development of this rampant caries process[2].

The radiation to the salivary glands and teeth results in weakened enamel- dentin bonds[2]. Even a low dose of 20Gy can cause changes in the consistency and amount of saliva. The properties of saliva become altered just after 4-5 fractions of radiation[2]. The similar kind of presentation was seen in our case, with a reduced salivary flow at a radiation dosage of 30 Gy with a rampant carious destruction of teeth. It's also related to the location and size of the tumor and the method used for radiotherapy[2]. Newer methods such as intensity modulated radiotherapy (IMRT) reduce the adverse effects by avoiding larger doses of radiation and thus retaining their function[2]. The changes noticed in the saliva include increase in viscosity, reduced buffering capacity, changes in the anti-bacterial system, alteration in the composition of saliva and changes in the salivary electrolyte concentration[5]. As the salivary pH and buffering capacity are reduced, the dissolution of the mineral component of enamel and dentin take place easily[2]. Thus the remineralisation of the dental hard tissues is altered leading to progression of the caries process. Also there is reduced oral clearance of the food debris due to alteration in the flow of saliva[2,5]. This leads to changes in the oral flora after radiation therapy. There is an increase in the levels of cariogenic and acidogenic microorganisms like Streptococcus mutans, Lactobacillus and Candida species[2]. These changes are manifested from the onset of radiotherapy to 3 months after completion and can remain constant thereafter[2]. All these factors like shift in the oral microflora, altered salivary composition and reduced flow undoubtedly leads to an enormous increase in the amount of caries and risk for periodontal infections[2,5].

A study was conducted by Springer[4] et al, which concluded that radiation is thought to have direct destructive effect on dental hard tissue, especially at the dentinoenamel junction (DEJ). Also there are morphometric differences in the colour of the irradiated enamel signifying that it is less resistant to acid attack[4,6]. There is hypovascularity resulting in a decrease in the circulation through the pulpal tissue in the teeth located in the irradiated region. This decrease in the blood flow also has an effect in the rampant destruction of the teeth. He also concluded that there is a significant increase in the collagen cross links lysyl pyridinoline and hydroxylysyl pyridinoline in dialyzed and ultra-filtrated probes of pulpal tissue of irradiated teeth as compared to non-irradiated ones[4]. This shows that there is a significant increase in the amount of collagen caused by direct radiogenic destruction, which further contributes to decreased vascularity and secondary fibrosis, thereby impairing the odontogenic metabolism[4]. This degeneration of the odontoblast will further lead to obliteration of the dentinal tubules is due to the direct cell damage effects of radiation. This alteration in the metabolism along with latent damage of the parenchyma ultimately leads to functional disturbances. This acts as a causative factor for the progression of rampant destruction of the teeth[4]. An increase in the stiffness of enamel and dentin were observed near the DEJ. This is made on the basis of the hypothesis that there is radiation induced decrease in the protein content, with a reduction in the enamel content.

These alterations in the chemical composition and mechanical properties lead to biomechanical failure at the DEJ and enamel delamination[4]. The minimal amount of tooth damage is seen below 30 Gy, with a 2-3 times increased risk of tooth damage between 30-60 Gy, most likely due to effect on salivary glands[2]. A 10 times increased risk of tooth damage is seen when the dosage is above 60 Gy[2]. These findings suggest a positive correlation with the effects of radiation and tooth damage[6]. Clinically, the progression of radiation caries begins at the labial surface, at the cervical areas, followed by caries affecting the smooth surfaces including the mandibular anterior teeth, which are the areas most resistant to caries in non-irradiated populations[2,6]. This is because, in non-irradiated teeth, there is mechanical cleansing of these surfaces by the continuous flow of saliva which is severely affected in radiation due to hyposalivation[2,6]. The progression of the lesion mostly affects the cervical areas of the teeth, indicating that this area is prone more caries. Consequently there are changes in the color and translucency of the enamel which causes increased breakdown and friability of the tooth with complete amputation of the crown[2]. Our case also presented with similar carious lesions at the cervical areas of the mandibular anteriors along with smooth surface carious lesion involving all the teeth.

Clinically three different patterns of the progression of the caries lesion have been identified[2].

Type 1- The most commonly seen pattern, which affects the cervical aspects of the teeth and extends to the cement -enamel junction. A circumferential decay develops and amputation of the crown is seen often.

Type 2- It appears as areas of demineralization on all the surfaces of the teeth. Generalized erosion with wearing away off the incisal and occlusal surfaces are seen.

Type 3- It is the least common pattern, with changes in the color of dentin seen. The crown becomes dark brown to black with incisal or occlusal wear.

Till now no evidences suggestive of microscopic differences between intial radiation carious lesions and healthy incipient lesions have been reported[2]. According to the above mentioned classification system, our case can be categorized as Type 2.

The management should mainly focus on the factors that may lead to the radiation induced caries rather than the caries itself, including xerostomia. The preventive measures that can be carried out before the initiation of radiation therapy include a thorough clinical and radiographic examination including examination of the mucosa, dentition and periodontium[2]. Periodontal evaluation and restoration of the carious tooth should be carried out. Also teeth with severe pulpal or periodontal infection should be extracted prior to the radiation therapy to reduce the risk of osteoradionecrosis. The patients should also be educated about the home oral hygiene methods including use of dental floss and mouth wash, restricted cariogenic diet and use of self applicable topical fluoride[7,8]. Radiotherapy by sparing the salivary glands, use of muscarinic agonists such as pilocarpine and use of cytoprotective drugs will help to reduce the effect of radiation induced xerostomia to a greater extent[6]. Also, surgical transfer of submandibular glands can be done following the Management Guidelines and Quality of Life Recommendations as given by the American Society of Clinical Oncology clinical and practise guidelines by Brennan[9] *et al.* (Oral Care Study Group 2010). Salivary output has shown to increase in patients undergoing parotid sparing IMRT when compared to conventional radiotherapy techniques.

The use of certain cytoprotective drugs also decreases the damage to salivary gland tissue by making it less sensitive to radiation damage[8]. These drugs, like Amifostine (WR-271, Ethyol –R) after entering the blood stream will get converted to its active form WR – 1065 by rapid hydrolyzation by endothelial alkaline phosphatise. The drug acts as a scavenger against free radicals and prevents radiation damage to the DNA. According to a study by Seikaly H et al, the incidence of acute xerostomia reduced from 78% to 51% and chronic xerostomia from 57% to 34% after administration of the drug[2]. Normally during head and neck radiotherapy, about 60-65 Gy of radiation is delivered to major salivary glands. Submental region receives only scattered dose of the radiation, of about only 5% of the total radiation. Surgical procedure that involves the transfer of a single submandibular salivary gland to the submental space was introduced in the early 1980s. But this can be employed in patients who have a negative cervical lymph node on the contra lateral side.

During and after the phase of radio therapy, patient education should be done regarding maintenance of

good oral hygiene[8]. This includes the use of a soft bristled tooth brush, brushing about 2- 4 times/ day and the use of supplementary aids like dental floss and mouth wash. Fluoride prophylaxis can also be done. Salivary substitutes and sialogoguges can also be used for relief of symptoms. The use of pilocarpine increases the salivary flow rates. Cevemeline, a new anticholinergic drug when given at a dosage of 30-45 mg three times a day for 52 weeks increases the production of unstimulated saliva. Sugar free gums like xylitol can also be used which stimulates the salivary flow rate, sugar clearance and buffering capacity[2,8]. Salivary substitutes can also be used, in those patients who do not respond to the pharmacological management. These substitutes are mainly based on aloe vera, carboxy methyl cellulose, xanthan gum etc but these provides only short term relief[2].

After completion of the radiation therapy, frequent recall and follow up should be schedulded. Appropriate treatment includes oral prophylaxis and restorations should be carried out. Extractions have to be avoided after the radiation therapy to avoid the risk of osteoradionecrosis[10]. As our patient presented with carious lesion involving all the teeth, root canal therapy was advised followed by full mouth rehabilitation and the patient is currently under treatment. Also diet counselling and oral hygiene instructions have been given to the patient. Patients should be made aware of the need of well maintained oral health following radiation therapy. They should be kept under regular supervision to minimize the risk of caries. Head and neck radiotherapy causes several alteration in the oral environment leading to conditions like radiation mucositis, xerostomia, changes in the salivary flow rate and buffering capacity, alteration in the oral microflora which will finally lead to rampant caries process causing destruction of teeth. In order to maintain the health of the patient it is advised to maintain proper oral hygiene.

REFERENCES

1. Chen AM, Lau VH., Farwell DG., Luu Q, Donald P J. Mucoepidermoid carcinoma of the parotid gland treated by surgery and postoperative radiation therapy: clinicopathologic correlates of outcome. Laryngoscope. 2013;123:3049–55.
2. Gupta N, Pal M, Rawat S, Grewal MS, Garg H, Chauhan D et al. Radiation-induced dental caries, prevention and treatment: a systematic review. Natl J Maxillofac Surg. 2015;6:160-6.
3. Shafer EG, Hine MK, Levi BM. A textbook of Oral Pathology.6th Ed. (Eds R Rajendran, Sivapathasundaram):440-3. Philadelphia, WB Saunders, 2009.
4. Springer IN, Niehoff P, Warnke PH, Böcek G, Kovács G, Suhr M et al. Radiation caries – Radiogenic destruction of dental collagen. Oral Oncol. 2005;41:723-28.
5. Grötz KA, Duschner H, Kutzner J, Thelen M, Wagner W. New evidence for the etiology of so-called radiation caries: proof for directed radiogenic damage of the enamel-dentin junction. Strahlenther Onkol. 1997;173:668-76.
6. De Moor R. Direct and indirect effects of medication (including chemotherapy) and irradiation on the pulp. Rev Belge Med Dent. 2000;55:321-33.
7. Vissink A, Jansma J, Spijkervet FK, Burlage FR, Coppes RP. Oral sequelae of head and neck radiotherapy. Crit Rev Oral Biol Med. 2003;14:199-212.
8. Rao K, Castelino RL,Shetty U, Babu GS. A review of post-radiotherapy complications necessitating urgency for further advances and innovations in radiotherapy. Austin Journal of Radiology. 2015;2:1026.
9. Brennan MT, Shariff G, Lockhart PB, Fox PC. Treatment of xerostomia: a systematic review of therapeutic trials. Dent Clin North Am. 2002;46:847-56.
10. Lakshman AR, Kanneppady SK, Castelino RL. Radiation caries in irradiated patient of nasopharyngeal carcinoma: a case report. Pacific Journal of Medical Sciences. 2013;11:88-94.