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Editorial

Role of Journals in Life Long Learning

As time goes by we become acutely aware of the world changing around us. Opportunities, problems and knowledge base seems to emerge and disappear like the opening and closing of automatic doors. The half life of knowledge is the time that elapses before half of the knowledge in a field is superseeded or shown to be untrue . In this fast changing world the half life of knowledge in medicine and dentistry is suggested to be five to seven years . Continual change can be either stimulating or stress provoking. The key to facing this challenge is to be ready to detect,absorb and implement acquire new tools and knowledge and bring it to our field ,continually through the pursuit of lifelong learning. The information and experience so acquired should be communicated to our colleagues as well , for the benefit of the patients , profession and society as a whole. Keeping abreast of the latest information requires that clinicians involve themselves in a process that may be as informal as regular dialogue with colleagues about shared experiences or as formal as the pursuit of higher education, continuing education courses, books and scientific journals.

Since research and evidence based practice is the scientific basis for health care-related practice, exposure to research information and understanding the process is very important for translating these findings into clinical practice. Clinicians are expected to provide safe and effective care based on the evidence. In spite of computerized databases, continuing education meetings, and study clubs , Scientific journals still remains the gold standard in disbursing and providing , information and assistance in finding the best evidence .

TheTrivandrum Dental Journal, the official publication of the Indian Dental Association , Trivandrum Branch, is intended to be a research periodical that aims to inform its readers of ideas, opinions, developments and key issues in dentistry - clinical, practical and scientific - stimulating interest, debate and discussion and an opportunity for life long learning ,amongst dentists of all disciplines. All papers published in the TDJ are subject to rigorous peer review by our excellent review board. We have tried to design the journal in such a way that the readers can find the relevant information fast and easily.

By creating and sustaining a critical and reactive journal the Trivandrum branch of the Indian dental association intends to construct a space where its members irrespective of their field of practice ,can learn how to "test the waters" academically, create sophisticated and informed arguments and ruminate on their invaluable experiences in dentistry.

In short TDJ is envisaged to be an excellent conduit for scientifically updating ourselves , sharing information and and enhancing our performance through a process of lifelong learning.

I take this opportunity to request the support of the esteemed members of the IDA Trivandrum branch in sustaining Trivandrum Dental Journal by contributing articles, letters ,opinions, and last but not the least ,finance .

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Editor in Chief
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CASE REPORT

Comprehensive report of a case of central odontogenic fibroma

Bindu.J.Nair[1], Sivakumar T.T.[2], Sooraj.S.[3], Freeda Mary.S[4]

ABSTRACT

A case of central odontogenic fibroma, a benign neoplasm of odontogenic origin is reported here. It can present histologically as fibrous tissue associated with or without odontogenic epithelium called the simple type. Another type is when fibrous tissue is associated with dysplastic dentin and cementum like material called the WHO type. Histopathological differential diagnosis is also listed out which highlights the importance of adequate expertise for diagnosing a fibrous lesion of the jaw microscopically.

KEYWORDS

Benign neoplasm, Odontogenic origin, Central odontogenic fibroma.

Introduction

The Central Odontogenic fibroma is a rare benign neoplasm considered to be derived from the mesenchymal tissue of dental origin.It is a poorly defined tumour of the jaw bone which has only been infrequently reported in the literature[1].It could appear to mimic a dentigerous cyst,odontogenic keratocyst,and or other odontogenic tumours.Central odontogenic fibroma resembling endodontic lesions have also been reported[2,3]. The Central Odontogenic fibroma consist of fibrous connective tissue containing varying amounts of odontogenic epithelium[4]. Sometimes calcified material resembling dentin or cementum like material is present[5]. Clinically, the Central Odontogenic fibroma could appear as an asymptomatic expansion of the buccal or lingual cortical plate, occurring in the mandible and in the maxilla with equal frequency. In the maxilla the lesion appears frequently to involve the anterior region, whereas in the mandible the lesion tends to be located in the posterior area involving the premolar and molar areas. The radiological examination of this lesion appear as an area of radiolucency or as an area with mixed radiodensity, most of the time the lesion has well defined borders [6]. The Central Odontogenic fibroma has been described as an unilocular[7,8] or a multilocular radiolucent lesion; root resorption and displacement have also been reported.

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Case Report

A twelve and a half years old female was referred to the outpatient department of PMS college of Dental Science and Research with a chief complaint of painless swelling in the right side of face since 2 years. The swelling was slowly increasing in size. Her medical history was non contributory.

Extra orally there was a bony hard non tender enlargement of the right side of the mandible. Intra

oral examination revealed a partially erupted, lingually displaced second premolar, missing first molar and an unerupted second molar on the right side. Expansion of the buccal cortical plate in relation to the mandibular right second premolar was noticed.

Radiographic evaluation showed the presence of a unilocular radiolucent area in association with impacted mandibular right first and second molars. The right lower border of the mandible showed an outline similar to that of thumb impression. The serum calcium and serum alkaline phosphatase were within normal limits. The mandibular axial and coronal CT scan revealed an expansile cystic lesion in posterior aspect of right half of mandible in the region of molar tooth. The radiographic picture and the CT scan gave a possible differential diagnosis of a dentigerous cyst, fibro-osseous lesion, unilocular ameloblastoma, adenomatoid odontogenic tumour or a traumatic cyst.

An incisional biopsy was taken and the histological examination of the lesion was done under hematoxylin and eosin stain. Section showed moderately collagenous fibrous tissue with prominent fibroblasts. In certain areas the cellularity was more composed of plump proliferating fibroblasts. Fibroblasts did not show any pleomorphism or mitotic figures.

The histopathological picture was suggestive of a proliferative fibrous lesion. The differential diagnosis histopathologically were

a. Aggressive fibromatosis
b. Desmoplastic fibroma of bone
c. Desmoplastic ameloblastoma
d. Central odontogenic fibroma

Subsequently the excision of lesion under general anaesthesia was carried out in the department of Oral and Maxillofacial surgery, PMS college of Dental Science & Research.

Histopathological examination revealed bundles of collagen fibres showing proliferating fibroblasts which are spindle shaped with wavy nuclei. Strands and small nests of cells with deeply staining nuclei were seen scattered among the collagen fibres.

Immunohistochemical staining on the paraffin embedded section with pancytokeratin and CD 34 found that the cells were positive for pancytokeratin confirming that these cells with the deeply staining nuclei are odontogenic epithelial cells. Hence the diagnosis of Central Odontogenic fibroma – simple type was made.

Discussion

Central Odontogenic fibroma is a central tumour of the jaws which is seen so infrequently that little is known about the neoplasm. The

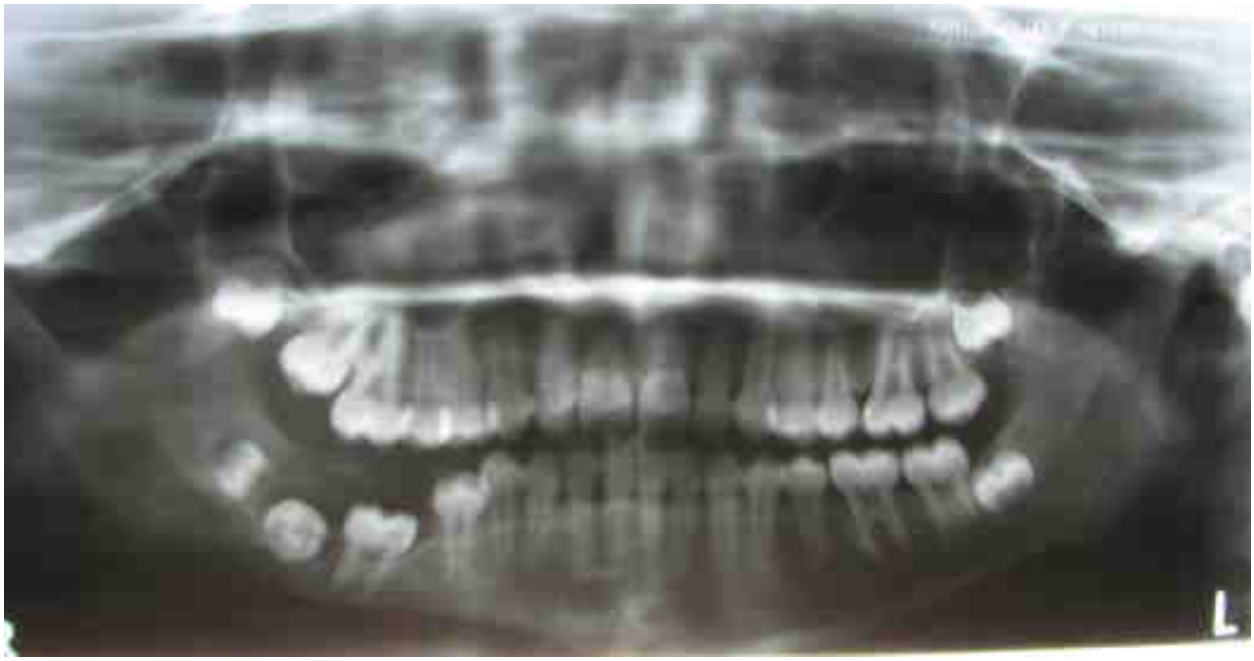

Fig-I : OPG showing impacted 46, 47 and unilocular radiolucency associated with 46, 47.

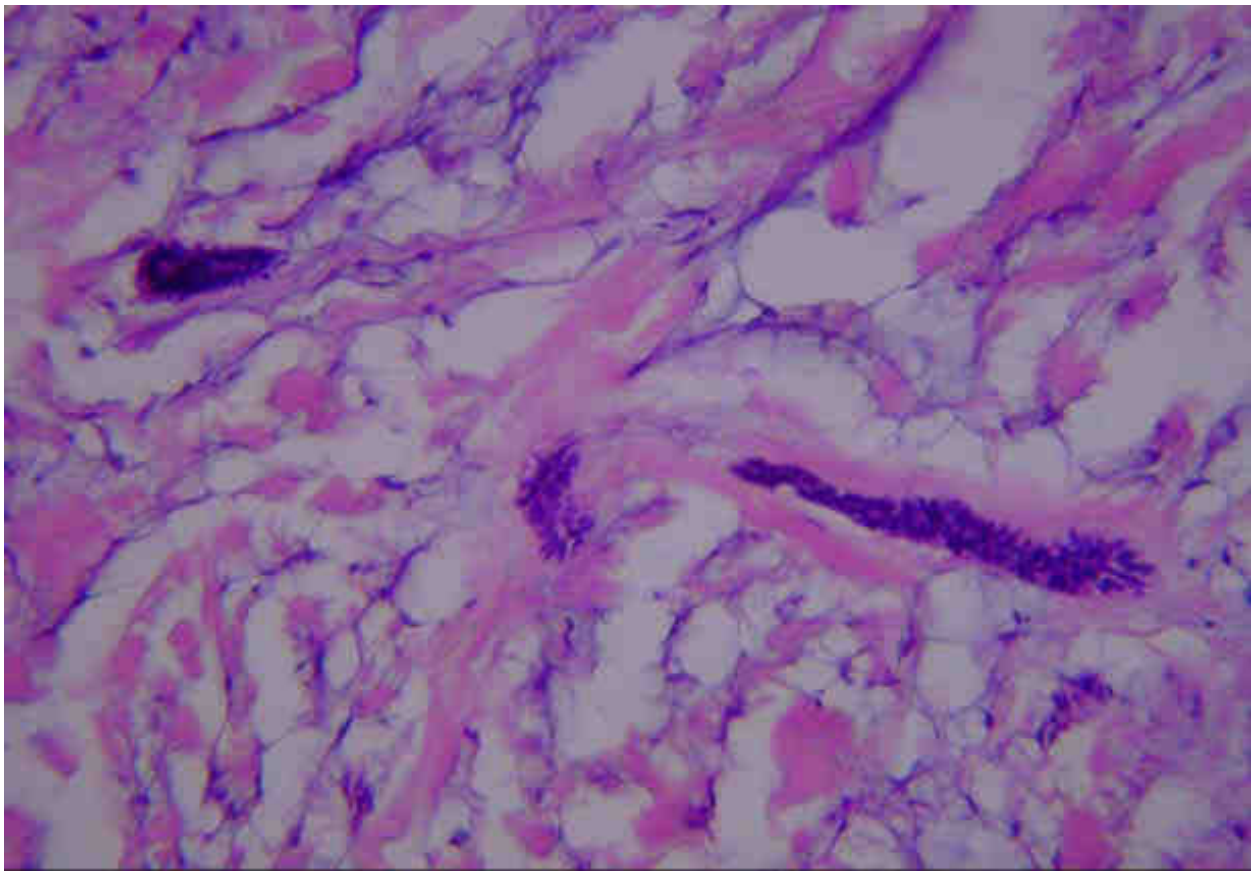

Fig. II : Photomicrograph (40x) showing fibroblasts in a moderately dense connective tissue with islands of odontogenic epithelium (H&E stain).

Central Odontogenic fibroma lesion is often diagnosed in the second and third decade of life. In International literature a considerable age range was noted. Some authors reported a distribution related to gender with 2.2:1 ratio for women is to men[9]. The most frequently observed sign was swelling and dislocation of adjacent teeth. Clinical symptoms such as pain and paraesthesia were uncommon.

The radiographic features of Central Odontogenic fibroma were compatible with radiolucent lesions with well defined borders. The radiographic unilocular or multilocular aspect of the lesions seemed to be dependent on the dimension of the lesion. Small fibromas (about 2cm) had a radiographic unilocular appearance whereas large lesions (about 4cm) had a radiographic multilocular appearance[10]. Many clinical cases of odontogenic fibroma showed a dislocation of adjacent teeth and sometimes root resorption. The case reported in this study showed a unilocular radiolucent area with well defined borders associated with dislocation of adjacent teeth without signs of root resorption. This was similar to the findings reported by Hewang et al [11]. The radiographic feature of Central Odontogenic fibroma are similar to other peripheral odontogenic tumours such as traumatic bone cyst, ameloblastoma, odontogenic cyst and central giant cell granuloma[13]. It was originally thought that most of these lesions have multilocular radiolucencies but current reports show that they are more unilocular than multilocular radiolucencies[12].

The histologic differential diagnosis is based on the presence of reactive fibrous hyperplastic tissue, which may contain occasional inactive odontogenic cells. Mineralised tissues like bone, dentinoid or even cementum –like material may be found, sometimes associated with the odontogenic epithelium.

Modified WHO classification groups Odontogenic fibroma as a benign lesion derived from 'odontogenic ectomesenchyme with or without odontogenic epithelum'. Gardner[13] reviewed the information concerning Central Odontogenic fibroma, identifying lesions with different histologic patterns. The first type,

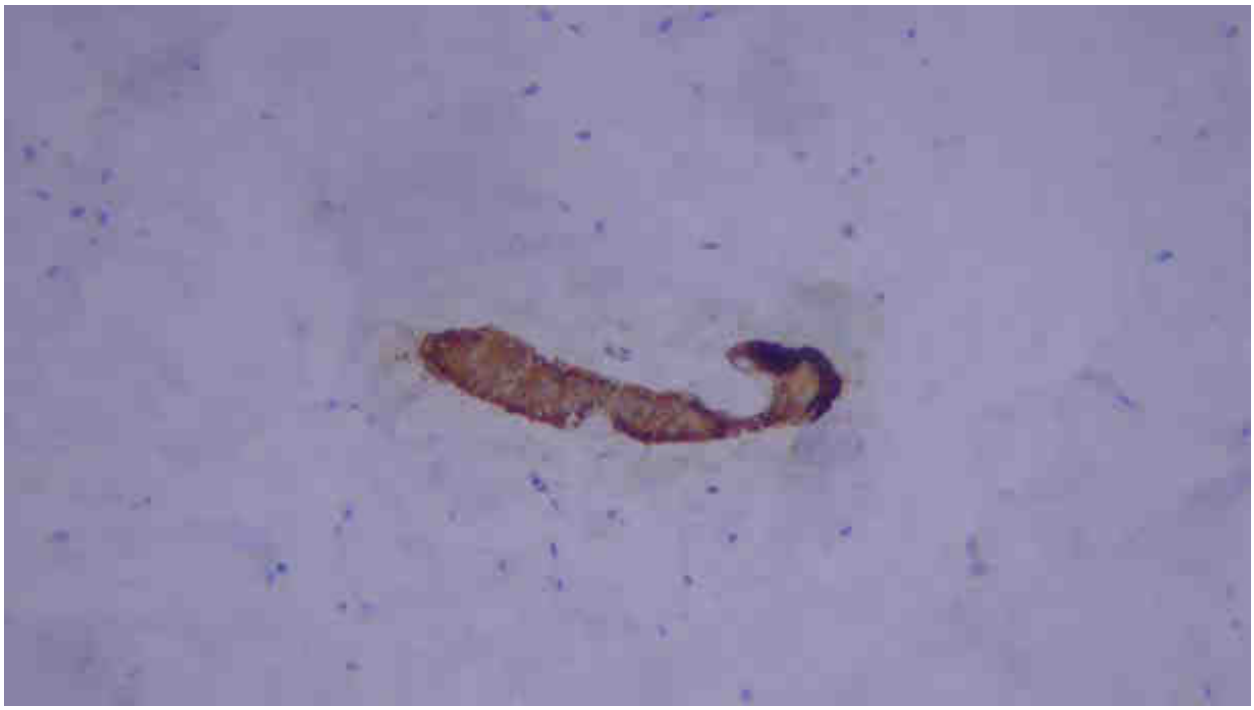

Fig. III : Photomicrograph (40x) showing odontogenic epithelium stained positively with pancytokeratin.

classified as simple, contained fibrous tissue with various amounts of collagen and the second type contained fibrous tissue with myxoid area associated to odontogenic epithelium. Moreover dysplastic dentin and cementum like material could be found. This latter type was designated as Central Odontogenic fibroma-WHO type.

Central Odontogenic fibroma occurring in the maxillary right canine region in a 26 year old female was reported by Daniel[14] and here also he found an absence of odontogenic epithelium.

Though recurrence is not established in Central Odontogenic fibroma, few clinical cases reported in literature had a recurrence which required more extensive surgical excision[4]. A clinical case which had recurrence 9 years after surgery has also been reported[15]. In another clinical study it was found that 2 cases out of 15 showed recurrence[16].

In conclusion clinical radiological and histological aspects of the case reported here is consistent with the diagnosis of Central Odontogenic fibroma – simple type. This paper also highlights the importance of adequate pathological expertise for histological examination of fibrous lesion of the mandible.

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CASE REPORT

Malignant Schwannoma Masquerading As An Innocuous Facial Swelling : A Case Report

Manoj. S. Nair

ABSTRACT

Sarcoma arising within a peripheral nerve has been termed variously as Malignant Schwannoma, neurofibrosarcoma and malignant peripheral nerve tumour. The neoplasm which has a rather debated histogenesis requires extensive histologic examination for diagnosis. A case of malignant schwannoma in a 40 year old male patient presenting as a localized facial swelling is described.

KEYWORDS

Facial swelling, malignant schwannoma, peripheral nerve tumor

Introduction

Sarcoma arising within a peripheral nerve has been termed malignant schwannomma, neuro fibrosarcoma and malignant peripheral nerve sheath tumour. Malignant schwannomas are rare and are classified among the soft tissue sarcomas, accounting for about 10% of all such tumours. It is extremely rare for a malignant schwannoma to occur in the oral cavity[1]. This neoplasm is often associated with Von-Recklinghausen's disease (neurofibromatosis). It can occur as a solitary malignant schwannoma also. In a study conducted by Ducatman et al, 52% of the cases were found to be associated with Von-Recklinghausen's disease and 48% were found to occur as solitary malignant schwannoma[2]. In a study conducted by Sordillo and his associates it was found that 4 to 29% of the patients with neurofibromatosis may have malignant schwannoma at sometime in their lives[3]. Reports of malignant schwannoma occurring after irradiation has also been published[4]. Malignant schwannoma, was found to occur in the age group of 7 to 75, mean age being 34[2]. Solitary malignant schwannoma was found to be distributed to all parts of the body while neoplasms associated with Von-Recklinghausen's disease was found to be centrally located[3]. 23% of the cases studied by Ducatman et al was found to occur in the head and neck region[2]. In a study conducted by Leu et al on head and neck schwannomas, 49% of malignant schawannomas were found to occur in face, scalp and external ear canal and 18% of the cases were located in oral and nasal cavity[5]. In the soft tissue malignant schwannoma, within oral cavity, the tongue, lip, gingiva, palate and buccal mucosa have been sites of involvement. Here a rare case of solitary malignant schwannoma of the face is presented.

Case Report

A 40 year old man presented with swelling in the right cheek of 6 months duration (Fig.1). The mass

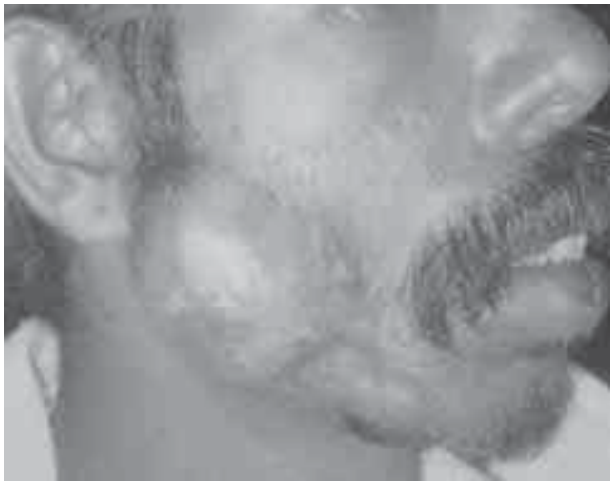

Fig. 1 Photograph showing facial swelling.

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which had a size of 7x5cms was painless, non tender, firm, ovoid in shape, well circumscribed and was not attached to the underlying structures. The skin over the swelling was smooth and free. A scar was present anterior to the lesion near the lower border of the mandible. Intra orally no swelling was visible. On general examination no similar swelling was found anywhere in the body. Patient gave a history of a similar swelling in the same site which was removed three years back. No hospital records were available for the same. A clinical diagnosis of benign soft tissue tumour was made. An incision was done under local anaesthesia. Histopathologic features were suggestive of a neurolemomma. Later excision of the tumour was done under general anaesthesia. The tissue was submitted for histopathologic examination. Serial section was made and stained with H&E.

Fig.2 High power magnification, H&E, 40X, showing cellular pleomorphism, nuclear hyperchromatism and abnormal mitotic figures.

Histopathology

Microsection consisted of two bits showing a slightly different picture. One bit showed densely packed cells with fibrous capsule. Cells were uniformly arranged without streaming pattern. Nuclei appeared vesicular, less hyperchromatic, ovoid to elongate at certain regions. Vascular spaces were seen between tumour cells. Intercellular stromal component was present sparsely (Fig.2). The other bit was composed of densely packed spindle shaped cells partly covered by fibrous capsule (Fig.3). Proliferating cells at one region showed pleomorphism, hyperchromatic nuclei of ovoid spindle shaped pattern and numerous abnormal mitotic figures (Fig. 2). A diagnosis suggestive of malignant schwannoma was made.

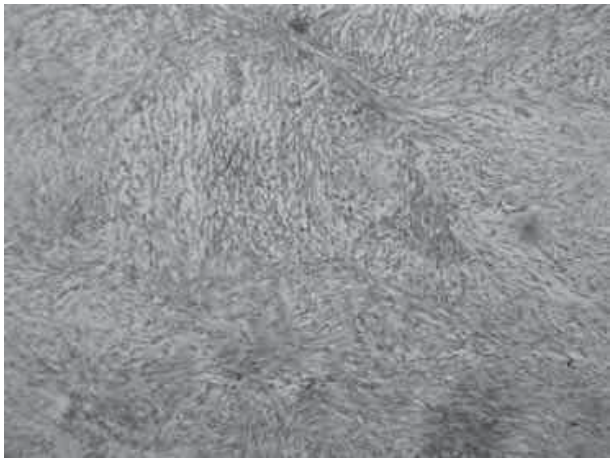

Fig.3 Low power magnification, H&E, 10x.

Discussion

The microscopic appearance of malignant schwannoma is often similar to fibrosarcoma. The differential diagnosis of malignant schwannoma includes fibrosarcoma, malignant melanoma, malignant fibrous histiocytoma, leiomyosarcoma and rabdomyosarcoma[2]. D. Augustino[6] and Stout[7] have proposed a criteria for diagnosis of malignant Schwannoma. This include (a) tumour origin from a nerve, (b) presence of contiguous neurofibroma and to lesser extend (c) an association with Von-Recklinhausen's disease. In this case the incision biopsy finding was typical of Neurolemmoma. The presence of dysplastic features disclosed by the serial sections of the excision biopsy material made us reconsider the case and formulate a diagnosis of malignant schwannoma. Considerable debate exists regarding the histogenesis of malignant schwannoma. Certain morphological features such as wavy cytoplasmic and nuclear configuration and finding of cellular palisading suggested a Schwann cell derivation. There is certain evidence which suggest that malignant schwannoma does not arise from Schwann cells alone. Many tumours show a predominantly herring bone pattern characteristic of fibrosarcoma[2]. The transition of neurolemmoma to malignant schwannoma has been reported to be extremely rare[8].

The diagnosis of malignant schwannoma requires extensive histologic examination. Mitotic index, cellularity, nuclear atypia and necrosis are the most reliable criteria. Immunohistochemical markers, including S-100 protein, leucin 7 and myelin basic protein help separate malignant schwannoma from

other spindle cell type tumours[10]. Jeffery et al has reported S-100 protein to be strongly positive in a case of malignant schwannoma involving trigeminal nerve[11]. S-100 protein is very helpful in differentiating histologically closely resembling lesions such as fibrous histicytoma, fibrosarcoma and synovial cell sarcoma from malignant schwannoma[12]. Solitary malignant schwannoma though less differentiated has a better prognosis compared to that of malignant schwannoma associated with Von-Recklinghausen's disease[3].

The recommended treatment for malignant schwannoma is radical excision with as wide a margin of normal tissue as is feasible and removal of all but the most vital structure. When radical removal is not possible, excision combined with high dose radiation therapy seems to be the best alternative[9]. The role of chemotherapy remains unsettled.

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PRACTICE

Managing Your Orthodontic Practice

R Roopesh

ABSTRACT

In this difficult economy, are you yourself responsible for your declining practice …. Having little or no business management background, many orthodontists tend to latch onto nuggets of perceived wisdom picked up from colleagues. Unfortunately, many of these "nuggets" turn out to be lies. If acted upon, they can be very counterproductive. At a time when orthodontists are down anywhere from 5 to 10% because of the economy, you can't afford to be sidetracked by 'ortho' lies. Here are some that orthodontists commonly tell themselves. Know them. Avoid them!

KEYWORDS

Orthodontic Practice, Practice Management

In this difficult economy, are you yourself responsible for your declining practice ….

Having little or no business management background, many orthodontists tend to latch onto nuggets of perceived wisdom picked up from colleagues. Unfortunately, many of these "nuggets" turn out to be lies. If acted upon, they can be very counterproductive. At a time when orthodontists are down anywhere from 5 to 10% because of the economy, you can't afford to be sidetracked by 'ortho' lies.

Here are some that orthodontists commonly tell themselves. Know them. Avoid them!

1. "Dentists need me. I don't need them."

Wrong! Without referrals from general dentists, your practice couldn't survive. It's not something many orthodontists want to admit or verbalize, but it's the truth. Referrals from your general dentists make up the majority of your patient base. Sure, you may receive referrals from patients, but could you build a thriving, growing practice around just patient referrals? Not really. Patient referrals are important but your referring doctors are even more valuable. You need dentists more than they need you—that realization is the foundation of a successful referral marketing program.

2. "Marketing is not for me."

This mentality may have worked 10 years ago but in this uncertain economy—not a chance! Most dentists have three or four options on where to send patients who need orthodontic treatment. You can sit back and wait for dentists to come knocking at your door. But you can bet your competitors won't be doing that. They'll be working to expand their referral base by targeting your referring doctors. Taking your referring dentists for granted is no longer an option. For orthodontists, implementing a referral marketing system that emphasizes regular, consistent contact with current and potential referring doctors.

3. "My dentist would never refer his patients to another orthodontist."

Don't be so sure. The relationship between orthodontists and their referring dentists can be a fragile one. Some orthodontists assume their long-term relationships with referring doctors are written in stone. Don't make that mistake. You should have regular contact with your referring dentists, whether

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it be a monthly phone call, a quarterly visit or a recurring lunch at a favorite restaurant.

4. "I don't have the time to market my practice."

You have to find time off your schedule to be in touch with the dentist who refer patients, or better still have a professional public relations service provider do the marketing for you.

5. "Meeting with referring doctors isn't a great use of my time."

If you think these meetings are unproductive, how do you think your referring doctors feel about them? If you harbor negative feelings about these get-togethers, isn't it possible that you unconsciously telegraph such thoughts to the referring doctor? Consider your last meeting with a referring doctor—did you do everything possible to make it a good experience? Were you friendly and outgoing? Were you a good listener and not just a good talker? Did you pick the appropriate venue to meet? Meeting with referring doctors can be an enjoyable and productive experience. It's up to you to make it that way.

6. "I am in touch with them THAT'S enough."

Do you think that is sufficient ? Don't you want to exceed expectations? Why be just another vanilla orthodontist who says hello to them over telephone once in a while? You can—and should—do better. And what about the rest of the year? Do your top referring practices only send you patients once a year? Of course not. Neither should your marketing efforts be just a one-time thing.

Exceeding expectations is the key to building long-term referral relationships.

7. "When things get better, I won't need to bother much with marketing."

This is a very common sentiment—and a dangerous one. Inconsistent marketing leads to inconsistent results. A good leader is always thinking ahead. You don't want to play "catch-up" when you don't have to. Consistent referrals should always be your goal. An effective referral marketing program is how you get them.

Conclusion

To have a growing orthodontic specialty practice, you must tackle these fallacies head-on. These facts (and others) must be identified and shrugged off, not clung to. The success of your practice in a fragile economy hangs in the balance!

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REVIEW ARTICLE

Photodynamic Therapy : The beginning of an end ?

V Vivek

ABSTRACT

Neoplasia often can be described as an evolution in real time; a moving target for therapeutic intervention. Proper management of patients with cancer demand multi disciplinary approach with the treatment plan for each patient representing the optimal combination of surgery, radiotherapy, chemotherapy and support services. Newer approaches to the treatment of neoplasm aim at potentially curative procedures with minimal host toxicity. The only current modality that approaches this goal seems to be photodynamic therapy.

KEYWORDS

Photodynamic therapy, Photo chemotherapy, Photosensitizers

Cancer is one of the leading causes of death in the world. Over one hundred forms of malignancies have been recognized, each with different clinical and biologic characteristics. It is this heterogenicity of the neoplastic process combined with the early appearance of metastasis and rapid mutations that makes treatment complicated. Neoplasia often can be described as an evolution in real time; a moving target for therapeutic intervention. Proper management of patients with cancer demand multi disciplinary approach with the treatment plan for each patient representing the optimal combination of surgery, radiotherapy, chemotherapy and support services. Conventional modalities of treatment like ionizing radiation, chemotherapy and surgery, inspite of having some curative potential, are nonselective with chances of recurrence. More over both radiation and chemotherapeutic agents are in themselves carcinogenic. Newer approaches to the treatment of neoplasm aim at potentially curative procedures with minimal host toxicity. The only current modality that approaches this goal seems to be photodynamic therapy.

Photodynamic therapy also called PDT, photo radiation therapy, phototherapy or photo chemotherapy is based on the discovery in 1900 that one celled organism treated with photo sensitizing drugs, will die when exposed to light at a particular frequency, while the same organism could survive in the dark[1]. Tappeiner and Jodlebauer introduced the German term "photodynamisagerscheinung" (photodynamic reaction) for a biologic system that required oxygen, a light absorbing sensitizer and light[2]. PDT probably gained its entry into the field of cancer treatment when Von Tappeiner and Jasionek , used topical eosin and a combination of sunlight and artificial light for successfully treating three patients with skin cancer [2,3]. The current era of PDT began with a series of observations by RichardLipson and SamSchwartz[1]. The recent surge in clinical and research interest stem from the work of Dougherty in the 70`s, which showed that PDT can selectively necrose tumors[3].

Photodynamic therapy is based on a biologic system that requires a light absorbing sensitizer, light and oxygen. It aims to provide local selective tumor destruction without damage to surrounding tissue. It essentially involves injecting the patient with a photosensitizer (PS) that is partially retained in the tumor for about 48-72 hours by a complex pharmacokinetic process. When illuminated with a 600-800nm wave length activating light , this

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retained dye by a process of photoexcitation absorbs light energy and converts it into chemical energy that can induce tumor necrosis . Tumor necrosis in PDT can occur due to induction of blood stasis, by direct killing of tumor cells or a combination of both. The absence of drug toxicity in the absence of light eliminates the possibility of adverse reaction at non neoplastic sites of sensitizer accumulation[4]. More over the long wave length light is harmless in the absence of the drug.

The most common feature of all sensitizers is the possibility that these compounds can transform absorbed light energy into chemical energy(photo excitation). Ideally the sensitizer should be chemically pure, have minimal dark toxicity, high tumor selectivity, and sufficiently large absorption coefficient at high wave length and optimal volume light penetration[5]. The most common photo sensitizing agents used are hematoporphyrin derivative and its more refined form, photofrin[1,6]. But these have several problems associated with their use like skin photosensitization,and inefficient use of light at poorly penetrating wavelength. Hence several second generation sensitizers like chlorines, purpurins, meta-tetrahydroxyphenyl porphine, pthalocyanines, benzoporphyrins, texaphyrins, delta amino levulanic acid, ATX-S10(Na), were introduced to alleviate the problems associated with photofrin and HPD[7]. Inspite of all these compounds,several studies have indicated that photofrin / HPD can give acceptable clinical results. Hence photofrin may represent the major photosensitizers in PDT for several years to come.

One of the major advantages suggested for PDT as a treatment modality for cancer is that it can localize specifically in the tumor. Several studies using isotope labeled photosensitizers have indicated that most photosensitizers localize more in the tumor cells than in the surrounding normal tissue[8,9].

Various photo sensitizer delivery mechanisms to the tumor sites have been proposed namely lipoprotein carrier system, macromolecular carrier system, monoclonal antibody based conjugates, microsphere based conjugates and macromolecular carrier systems[7]. But studies, regarding the photo sensitizer delivery mechanisms to the tumor site ,are still in its infancy.

The clinical effectiveness of PDT for the treatment of tumors depend on the appropriate delivery of both light and PS to the target tumor tissue.Various light sources ranging from mercury lamps to new generation lasers have been tried[10,11]. Xenon lamps 5000W have been used to treat breast carcinoma, but with the advent of lasers PDT came to rely more on lasers as light source. Lasers generate powerful monochromatic collimated light source suitable for efficient coupling into small optical fibres. In spite of the development of advanced laser systems like gold vapour laser, copper vapour pumped dye laser, flash lamp pumped dye laser, exciemer laser, and the recently introduced diode lasers, the argon pumped –dye laser still remains as the standard light source for clinical PDT. For surface irradiation wave length filtered lamps are considered as light source. Depending on the surface to be irradiated different types of light delivery systems have been designed, like bare fiber with microlens tips, flat fiber tips, double balloon catheters, spherical diffusing tips, cylindrical diffusers etc[7].

Once the PsS is localized in the tumor tissue destruction is expected. The mechanism of tissue destruction in PDT has been the subject of considerable investigation and debate. Damage to the tumor and surrounding tissue microvasculature play an important role in tumor destruction rather than cytotoxicity in PDT[12]. Some authors have suggested that PDT can cause immediate effects by causing damage to the cell membrane, mitochondrial or lysosomal sites or by inducing apoptosis[13].

Head and neck tumors may especially be amenable to PDT because they are likely to be readily accessible to illumination with current laser technology. Eventhough some authors have questioned the value of PDT in the treatment of large tumors, possibilities for eradication of wide spread preneoplastic and neoplastic tissue are appealing. Excellent response rates have been obtained with PDT in the treatment of intralaryngeal and upper digestive tract tumors[14]. However most studies have shown PDT to be effective only in short term cancer control, hence some researchers have suggested that PDT be employed as an adjuvant to radiotherapy[15].

Adverse reaction to PDT reported include persistant skin photosensitization,local

edema,transient pain, and superficial infections[16]. Newer sensitizers will help to circumvent some of the the adverse reactions. Problems like edema, and pain could be controlled conventionally in most cases.

The future of PDT lies in the (i)development of safe and effective agents that are rapidly cleared from the skin to minimize the need for protection of the patients from sunlight after therapy;(ii) use of agents that absorb light in the far red and near infrared to promote photodamage to pigmented and deep tumors;(iii) development of photosensitizers thah will enable superficial lesions to be treated without potential photodamage to the underlying normal structures.(iv)development of portable and economical light sources and light delivery systems that are simple and easy to operate.

PDT/photodynamic therapy is a new technique that will take a place with surgery, ionizing radiation, and chemotherapy for the treatment of tumors. It can be considered analogous to penicillin. Penicillin was only the beginning in the field of antimicrobials, like wise PDT may only be the beginning in the field of laser medicine and surgery for the treatment of neoplastic diseases.The beginning of an end?

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REVIEW ARTICLE

Role of Dental Surgeon in the Management of Hypertensive Patients

Sunila Thomas

ABSTRACT

A significant number of patients seeking dental treatment have undetected high blood pressure or uncontrolled hypertension. Patients with undetected high blood pressure or uncontrolled hypertension are at a high risk for developing cardiovascular disease and target organ disease. In this scenario, the Dental professionals should emphasize on screening their patients for hypertension and also make the general public aware of the significance of high blood pressure and its consequences.

KEYWORDS

Hypertension, Management, Dental Patients

Introduction

A significant number of patients seeking dental treatment have undetected high blood pressure or uncontrolled hypertension, should therefore be referred for medical evaluation. Dental Surgeons have played an important role in the detection of patients with hypertension.

Classification of Blood Pressure

In 2003, the National High Blood Pressure Education Program published their latest recommendations for hypertension. This was the seventh revision by the Joint National Committee on the Prevention, Detection, Evaluation and Treatment of High Blood Pressure and is known as the JNC-7 Report[1]. Their latest BP classification for adults is summarized in Table 1.

Estimation of Blood Pressure

Blood pressure reading should be taken on all new dental patients and at each recall appointments[2]. The patient should be allowed to rest quietly for at least 5 minutes before the procedure. The patient should not have smoked or had caffeine for at least about 30 minutes before the appointment. Seat the patient in an upright position and arms supported at heart level. The bladder of the cuff should cover about 80% of the arm, centered over the brachial artery. Two or more readings should be taken during the appointment, at least 5 minutes apart.

Table 1

Classification	Systolic BP	Diastolic BP
Normal	<120	<80
Prehypertension	120-139	80-89
Stage I hypertension	140-159	90-99
Stage II hypertension	>160	>100

Many patients have elevated blood pressure secondary to fear and anxiety. This is called "White coat" hypertension in which individuals present with persistent elevated BP in a clinical setting but present with non elevated BP in an ambulatory setting. Patients with labile white coat hypertension are diagnosed with BP readings taken over 24 hours which show a normal value. It is of interest to note that ambulatory readings taken after 24 hours sometimes reveal "masked" hypertension, in which patients who have a low BP in clinical setting may demonstrate an elevated BP over a 24 hour period[3].

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Faulty techniques will produce errors in BP readings[4]. A cuff too small or applied too loosely will give a falsely elevated reading. The width of the cuff should be about 40% of the diameter of the patient's arm and the bladder length should encircle about 80% of the arm. One should remember that, if the cuff is deflated too rapidly (>2-3 mm Hg per heart beat), the recorded systolic pressure will be too low and the diastolic pressure will be too high.

Risk factors for Hypertension

The major risk factors for hypertension are smoking, hyperlipidemia or Diabetes mellitus, being older than 60 years, being a man or a post menopausal woman and having a family history of cardiovascular disease for women under 65 years of age and for men under 55 years of age.

Target organ disease and clinical cardiovascular disease include left ventricular hypertrophy, angina, prior myocardial infarction, prior coronary revascularization, heart failure, stroke and transient ischemic attack, nephropathy, peripheral artery disease and retinopathy[5].

Recent studies stressed the importance of systolic hypertension and risk for cardiovascular disease[1,6,7,8]. According to JNC-7 report, systolic BP >140 in people over 50 years was an important risk factor than elevated diastolic pressure. Franklin[9], reported that subjects with isolated systolic hypertension are at greater risk for cardiovascular events than those with combined systolic / diastolic hypertension. Based on these studies it is evident that prolonged elevation of systolic pressure is more dangerous than that of elevated diastolic blood pressure.

Medical Management

Initial treatment of hypertension includes life style modifications like diet restriction, regular exercise, weight reduction and limiting the use of alcohol. The daily Sodium intake should be restricted to less than 2.4 gm. Even though sodium reduction causes a minimal decrease in blood pressure excessive sodium intake is definitely related to target organ damage such as left ventricular hypertrophy and renal disease[10].

The goal of medical management is to reduce the pressure up to at least the Prehypertension range (<140/90)[11]. It is advised to start the treatment with one drug at a low dose and then titrate the dose until the BP is controlled.

Diuretics are often the 1st drug given for the treatment of high BP. Diuretics (eg: Hydrochlorothiazide, Triameterene, Frusemide) reduce BP by decreasing vascular resistance and reducing blood volume.

Beta blockers (eg. Propranolol, Sotolol) decrease BP by reducing the rate and force of contractions. They are used in patients with history of angina and myocardial infarctions.

ACE inhibitors (eg: Captopril, Enalapril) produce vasodilatation by inhibiting the conversion of Angiotensin 1 into Angiotensin 2, thus lowering the BP.

Calcium channel blockers (eg: Amlodipine, Nifedipine, Diltiazem) decrease total peripheral resistance, slow the heart rate and decrease the force of contraction.

Alpha blocking agents (eg: Prazosin, Terazosin) inhibit the binding of norepinephrine to receptors in the arterioles leading to vasodilatation.

Directly acting vasodilators (eg: Nitroglycerin, Minoxidil) work independent of the ANS to relax vascular smooth muscles.

Methyl Dopa and Clonidine act in the CNS to decrease sympathetic nervous system output.

Angiotensin 2 receptor blockers (eg: Losartin, Telmisartan) are class of drugs which prevent vasoconstrictor from binding on smooth muscle sites in the arterials, promoting vasodilatation.

Combination of two drugs from different classes provide increased efficacy and minimize adverse effects of each drug. Common drug combinations are beta blockers and diuretics, ACE inhibitors and diuretics, Angiotensin 2 receptor blockers and diuretics, Calcium antagonist and ACE inhibitors[5].

Dental Management

Management of dental patient with hypertension include reduction of stress and anxiety, pain control and judicious use of vasoconstrictors in local anaesthetics. The Dentist should be aware of the systemic and oral side

effects of antihypertensives and the common drug interactions.

Stress and anxiety can raise the blood pressure[2]. The Dentist should maintain a good rapport with the patient. Very anxious patients can be given Diazepam 5 mg or short acting Benzodiazepines such as Oxazepam 30mg the night before and 1hour before the dental appointment. For more anxious patients Nitrous oxide can be used during the procedure.

Effective control of pain during and after the dental procedure is a very important aspect in the management of hypertensives. Previous studies clearly indicate that operative pain can increase the patients' blood pressure [12, 13]. Chronic use of NSAIDS decrease the effectiveness of some antihypertensive drugs[14]

Patients who are controlled hypertensives can be safely given up to 2 cartridges of 2% Lignocaine with 1:100000 epinephrine (0.036 mg epinephrine)[15]. If the resulting anaesthesia is inadequate, the dentist can use his judgment regarding the use of additional anaesthetics. Patients release endogenous epinephrine in response to pain during a dental procedure. A stressed patient can release upto 40 times the base line catecholamine level [16]. JNC-7 supported the fact that vasoconstrictors are not contraindicated in hypertensives, especially during a painful procedure.

Most of the antihypertensive medications have systemic and oral side effects and the Dentist should be well versed in the management of these side effects.[17,18,19] (Table 2 and Table 3).

Orthostatic Hypotension is one of the most common systemic side effects of antihypertensive drugs. To avoid this, the chair position should be changed slowly and patient should sit on the dental chair in an upright position for about 30 to 60 seconds before standing.

Calcium channel blockers, particularly Nifedipine, can cause gingival hyperplasia. In such cases the Dentist can consult with the patient's physician to consider an alternative antihypertensive agent.

Xerostomia is another adverse effect with most of the antihypertensive medications. The primary aim of treating these patients is to provide symptomatic relief and minimize the complications of xerostomia. Patients may be encouraged to take frequent sips of water and chew sugarless gum. Topical fluoride application and systemic medications like Pilocarpine and Cevimeline should be included in the treatment.

Lichenoid reaction, a condition clinically similar to lichen planus may occur in patients using Diuretics and Beta blockers. Patients should be referred to their Physicians to change to a new drug and reviewed to see if the lesion subsides. Symptomatic lesions may be managed with high potency steroid Clobetasol or antimetabolite Cyclosporine[20].

Table 2

SYSTEMIC SIDE EFFECTS OF ANTIHYPERTENSIVE DRUGS

Drug	Systemic side effect
Diuretic	Orthostatic hypotension, blood dyscrasia
Beta blockers	Orthostatic hypotension, blood dyscrasia
ACE Inhibitors	Orthostatic hypotension, Renal failure, Neutropenia
Calcium channel blockers	Orthostatic hypotension, renal failure
Alpha blockers	Orthostatic hypotension
Direct acting vasodilators	Orthostatic hypotension, blood dyscrasia
Central acting agents	Rebound hypertension if agent stopped abruptly, dizziness
Angiotensin 2 receptor blocker	Cough, muscle cramping, orthostatic hypotension

Conclusion

Patients with undetected high blood pressure or uncontrolled hypertension are at a high risk for developing cardiovascular disease and target organ disease. In this scenario, the Dental professionals should emphasize on screening their patients for hypertension and also make the general public aware of the significance of high blood pressure and its consequences.

Table 3

Oral side effects of antihypertensive drugs

Drug	Oral side effects
Diuretic	Dry mouth, Lichenoid reaction
Beta blockers	Dry mouth, taste changes, lichenoid reaction
ACE Inhibitors	Loss of taste, dry mouth, ulceration, angioedema
Calcium channel blockers	Gingival enlargement, dry mouth, altered taste
Alpha blockers	Dry mouth
Direct acting vasodilator	Facial flushing, increased risk of gingival bleeding and infection
Central acting agents	Dry mouth, taste changes, parotid pain
Angiotensin 2 antagonists	Dry mouth, angioedema, sinusitis, taste loss

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REVIEW ARTICLE

Antibiotic prophylaxis in the prevention of infective endocarditis - A review

Mathew Jose

ABSTRACT

Patients with certain cardiovascular disorders are recognized to be at risk of developing infective endocarditis (IE) following an invasive dental procedure. Whenever a clinically significant bacteremia is anticipated in susceptible patients antibiotic prophylaxis is advocated. However it has been observed that daily routine procedures like brushing and flossing also create significant amount of bacteremia which is much higher compared to that with a single tooth extraction. As a very few cases of IE are correlated with a preceding dental event, the role of antibiotic prophylaxis is questionable. It is recommended that more stress has to be given on maintenance of good oral health and hygiene in the prevention of IE. Knowledge about IE prevention among dentists is found to be inadequate. This article attempts to give an insight into the bacteremia and pathogenesis of endocarditis. Utility value of amoxicillin as a baseline antibiotic in chemoprophylaxis is evaluated. Furthermore, recommendations about prevention of IE with regard to patient motivation; dentist's awareness and cardiologist's role are elaborated.

KEY WORDS

Antibiotic prophylaxis, Infective endocarditis, Dentistry, Practise guidelines.

Endocarditis is defined as an inflammation of the endocardium, the lining membrane of the heart. It may involve only the membrane overlying the valves (valvular) or the general lining of the heart (mural). When the endocardium becomes infected with microorganisms that have entered the circulation, there will be progressive destruction leading to anemia, toxemia and ultimately cardiac failure, which is the most common cause of death. There is no absolute evidence about whether antibiotic prophylaxis is effective or ineffective against IE in people at risk associated with an invasive dental procedure[1].American Heart Association (AHA) has revised the recommendations periodically and now need for antibiotic prophylaxis is limited to a very few cardiac conditions. Guidelines in many countries recommend antibiotic administration prior to any invasive dental procedures for people at high risk of endocarditis. However updates from national Institute of health and clinical Excellence (NICE) in England and Wales recommend that antibiotics are not required[1]. Active debate is going on whether to go ahead with chemoprophylaxis or not. Cunha et al [2] reported a case where patients with Mitral valve prolapse who has taken chemoprophylaxis throughout her life for dental procedures and never developed IE. Because of changes in AHA guidelines in2007 she did not receive prophylaxis for a dental procedure and developed endocarditis 3 months later. Agent was found to be Streptococcus intermedius. Bacteraemia occurs with routine daily activities like brushing and flossing but considering the frequency of IE in high risk patients its significance requires further clarification.

Bacteraemia during invasive dental procedures result in dissemination of an undisputed etiologic

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agent in endocarditis, which after entering the blood stream might lodge inside the heart. A normal heart with a smooth lining will make it difficult for bacteria to adhere, while congenital heart diseases will produce roughening of the surface of endocardium where bacteria can localize and multiply. It is reported recently that the knowledge and practice regarding prevention of endocarditis among dentists were found grossly inadequate, with about 70% ignorant about current AHA guidelines[3]. This might result in over use of antibiotics as well as gross neglect in critical cases.

Bacteraemia from oral cavity and pathogenesis of endocarditis

Over 700 bacterial species have been identified in the human oral cavity and approximately 30% of gingival crevice flora is streptococci with the viridians group predominating[4,5,6]. More than 100 species of oral bacteria have been recovered from blood cultures following dental procedures. It has been observed that the blood stream is protected from the oral flora by a thin layer of mucous epithelium, any compromise to it could introduce the microbes into the vascular and the lymphatic system[7].Everett and Hirschman found that most episodes of bacteremia related to dental procedures will last for less than 10 minutes[8]. Roberts et al observed that the highest frequency of positive cultures occur 30 seconds after tooth extraction [9, 10], and bacterial seeding occurs within minutes of bacteria being introduced into the blood stream[11].

Whether bacteremia is due to dental or any other procedures involving the aero digestive system, the common factor in the development of IE is the adhesion of bacteria to sterile fibrin platelet vegetation known as nonbacterial thrombotic endocarditis (NBTE). NBTE is most often caused by the turbulent flow of blood across the cardiac tissue. This causes trauma to the tissue and subsequent deposition of platelets and fibrin. This area is ideal for bacterial adhesion and proliferation .Once the NBTE is infected with bacteria IE develops.

The Virulence of bacteria causing IE is related to the surface components that allow bacteria to adhere to NBTE. Streptococci, staphylococci and enterococci have several surface components that in animal studies have been shown to function as adhesins. Some viridians group of streptococci has a surface protein named Type 1 Fimbrial subunit or FimA which is a major adhesin to the fibrin platelet aggregates in NBTE[4, 12, 13]. Numerous bacterial molecules like FimA and staphylococcal adhesins function as mediators of adhesion to molecules in mammalian cells such as fibronectin, fibrin, fibrin platelet aggregates and other extracellular protein matrices[14]. Briefly, the pathogenesis of bacterial endocarditis can be summarized as;

- Damage to the endocardial surface, leading to adherence of platelets and fibrin. This adherence results in the development of non-bacterial thrombotic vegetation (NBTV)
- Discharge of bacteria into the blood from a local site, leading to a transient bacteremia
- Adherence of bacteria to NBTV with further deposition of fibrin and platelets
- Multiplication of bacteria within the vegetation
- Development of local and systemic consequences of bacterial endocarditis

Infective endocarditis most commonly affects the mitral valve followed by aortic valve and rarely found on the pulmonary valve. More than 90% of microorganisms in left or right sided valvular vegetation are inactive metabolically which severely limits the bactericidal effectiveness of antibiotics[15,16,17].

Relevance of antibiotic prophylaxis

Even though it is obvious that bacteremia occurs with many dental procedures, bacteremia from normal everyday activities such as brushing teeth twice daily for an year is about 5-6 million times higher than that occurring from a single tooth extraction[18]. If we consider bacteremia associated with daily activities such as flossing tooth brushing (20-68%),use of water irrigation devices(7-50%) use of wooden tooth picks(20-40%) and chewing of food (7-51%)the frequency of bacteremia due to dental procedure is insignificant (19).Colony forming units (CFU) of bacteria/ml introduced into the blood stream in a basic dental procedure is found to be less than 104 CFU which is quite comparable to that occurs after routine daily activities like brushing or eating.

Vander Meer observed that various dental procedures were correlated with a small number of IE cases and so prophylaxis could prevent this small number of cases if at all the therapy was effective[20]. Absolute risk for IE from a single dental procedure is 1/100 000 to 1/1 000 000 in patients with valvulopathy. In the general population the absolute risk is 1/14 000 000and so it is not justifiable to pre-medicate 100 000 to 1000 000 individuals when there is no concrete evidence that it is effective in preventing IE[7].

Another factor of serious concern is that risk of adverse events related to antibiotic use far exceeds any benefit that could be gained from antibiotic prophylaxis in certain patients. It has been reported that approximately 3% of adverse reactions are due to amoxicillin which is the preliminary antibiotic administered in those who are not allergic to penicillin for IE prophylaxis. Skin reaction accounted for 82% occurrences ,gastrointestinal effects (7%), hematologic complications and liver reactions accounted for 1 percent [21]. Prophylaxis with amoxicillin in a large unselected population carries a risk of death from anaphylaxis that is five times greater than the risk of developing IE. Indiscriminate use of antibiotics may contribute to the emergence of resistant strains especially Methicillin resistant staphylococcus aureus (MRSA)(12,22). An invitro evaluation of microbial flora of orofacial infection in 80 patients has revealed that amoxicillin was resistant in 92.85% and Ofloxacin as the most sensitive agent. Lack of response of bacteria to amoxicillin is correlated with it's over use[23].

Recommendations for minimizing the chance for infective endocarditis

It has been observed that maintaining good oral health and hygiene may present a more direct method of preventing and reducing bacteremia than IE prophylaxis prior to dental procedures. AHA also upholds the same concept[24]. Antibiotic prophylaxis carry a lot of limitation and its efficiency in preventing IE is questionable. AHA recommendations should be followed in high risk patients (Table 1, Table 2), but it is obvious that this will give coverage for bacteremia occurring during that procedure alone. Hence all attempts aimed at prevention of IE should be a team effort by the patient, the cardiologist and the dental surgeon.

Patient awareness.

Those who are vulnerable for IE should be well informed about their cardiac status and the consequences they might face if they get the disease. They should understand the need for maintaining good oral heath. Poor oral hygiene and gingival disease were significantly associated with IE related bacteremia during brushing and presence of generalized bleeding after tooth brushing was associated with almost eight fold increase[26]. They should inform their dentist regarding their cardiac status prior to the procedure. They should respond for dentists interests in oral prophylaxis, oral health care advices and should accept prompt dental preventive treatments.

Role of a Cardiologist.

Cardiologists are the best people to educate the patients who are vulnerable for IE. They should assure that the patient is taking regular dental check up and they carry a certificate of dental fitness from their dentists once in every six months. Cardiologists can dictate appropriate antibiotics suitable for the patient.

Role of a dental specialist

Dental specialist should have a thorough knowledge about the current recommendations of AHA regarding the management of cardiac patients who are vulnerable for IE. Selection of antibiotics is dependent on clinical experience of the dentist. Emergence of resistant strains should be identified by the dentist by serial sampling and culture and sensitivity tests which will help him to develop an efficient antibiotic preference list. The appropriate management of dental patient with cardiac disease is contingent on appropriate assessment and evaluation. A good medical history, medical evaluation, and definitive assessment of vital signs are essential for the safe delivery of dental care. In identified groups a regular check up schedule should be designed. This should include thorough oral and dental evaluation, radiographic survey, and oral health maintenance and dietary advices.

Use of local antiseptics prior to the procedure will considerably reduce the number of oral microorganisms and highly beneficial in minimizing

Table 1

Previously/No longer recommended	Currently recommended
Mitral valve prolapse	Patients with history of previous IE
Rheumatic heart disease	Patients with a prosthetic cardiac valve
Bicuspid valve disease	Recipients of cardiac transplantation who develop cardiac valvulopathy
Congenital heart conditions (eg. atrial septal defect, ventricular septal defect, hypertrophic cardiomyopathy)	Patients suffering from the following forms of congenital heart disease (CHD).
	Completely repaired CHD with prosthetic device or material during the first six months following the procedure where placement is either by surgery or catheter.
Calcified aortic stenosis	Unrepaired cyanotic CHD, which includes palliative shunts and conduits.
	Repaired CHD with residual defects at or adjacent to the site of a prosthetic device or patch (inhibiting endothelialization).
	Antibiotic therapy is no longer recommended for any other form of CHD other than those named above.

(Adapted from Wilson W, Taubert KA, Gewitz M, et al. Prevention of bacterial endocarditis: Recommendations by the American Heart Association. Circulation, Apr 2007)

bacteremia. Even after meticulous aseptic care and chemoprophylaxis IE has been expressed especially in the first 30 days[25]. Patient should be warned of this risk and asked to report back in case of any unexplained illness like fever fatigue and weakness are observed.

Briefly, the three management tenets proposed for patients at risk of bacterial endocarditis are

a. Communication with the patient's physician to establish the risk for development of endocarditis
b. Aggressive pre and intra treatment oral hygiene maintenance to minimize gingival inflammation
c. Antibiotic prophylaxis following latest guidelines by the AHA

Recommendations

It is recommended that while planning treatment for these patients, only achievable goals should be formulated.

- Overambitious treatment plans should be eliminated.
- The communication with the patient's physician is considered the most reliable method for identifying the risk involved. The medical history completed by the patients tend to exaggerate the risk, and out of 56 patients who reported a condition associated with bacterial endocarditis, only 14 were found to have risks requiring antibiotic prophylaxis[27].
- Adherence to strict oral hygiene measures is an effective method to prevent bacterial endocarditis. Poor oral hygiene results in plaque accumulation, as well as an increase in bacterial counts. Subsequent development of gingivitis results in bleeding from broken capillaries and may lead to transient bacteremia.
- In order to maintain high standards of oral hygiene, plaque assessment should be made a routine procedure during every dental appointment, along with counseling for patient motivation.
- Electric toothbrushes, which are prone to induce bacteremia, should be avoided, as the AHA does not include it under regular oral hygiene aids.

Table 2

Current recommended regimens for dental procedures

Patient	Antibiotic	One dose 30–60 min before procedure	
		Adults	Children
Able to take oral medication	Amoxicillin	2 gm	50 mg/kg
Not able to take oral medications	Ampicillin	2 g IM or IV	50 mg/kg IM or IV
	Cefazolin Ceftriaxone	1 g IM or IV	50 mg/kg IM or IV
Allergy to Penicillins or ampicillin	Cephalexin*	2 g	50 mg/kg
Oral	Clindamycin	600 mg	20 mg/kg
	Azithromycin Clarithromycin	500 mg	15 mg/kg
Allergy to penicillins or ampicillin and not able to take oral medications	Cefazolin	1 g IM or IV	50 mg/kg IM or IV
	Ceftriaxone Clindamycin	600 mg IM or IV	20 mg/kg IM or IV

*Do not use cephalosporins in patients with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin.

(Adapted from Wilson W, Taubert KA, Gewitz M, et al. Prevention of bacterial endocarditis: Recommendations by the American Heart Association. Circulation, Apr 2007)

- Antimicrobial mouth rinses with 0.2% w/v chlorhexidine as the main ingredient, might be helpful in reducing oral bacterial numbers. Repeated use of these agents does not reduce its sensitivity compared to long-term use of antibiotics.

Conclusion

Infective endocarditis is an established condition where oral bacteremia plays a major role and chemoprophylaxis was considered to be the most effective way of preventing IE. In the world of evidence based medicine and practice this concept lacks support. More stress is now given for regular maintenance of oral health and hygiene, early detection of dental problems and its management. It appears prudent, at the time a heart lesion is diagnosed, to institute a full dental comprehensive examination and later at frequent and regular intervals, as this ensures early diagnosis and treatment of dental lesions. Clearly much remains to be done to enhance the co operation between the cardiologist and the dental surgeon for the efficient management of the susceptible patients.

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REVIEW ARTICLE

Intensity Modulated Radiotherapy – A revolution in the treatment of head and neck cancer

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ABSTRACT

Advances in computers have paralleled advances in imaging technologies. This complexity has also been incorporated into radiotherapy treatment planning systems by the use of complex computer algorithms. Treatment planning of head and neck cancers with promising results of an unimpaired quality of life has been in the main attention of the clinician. This has ushered in an era of Intensity Modulated Radio Therapy which is a computer driven technology of optimized treatment plan-ning and a computer-controlled treatment delivery system. This article highlights the main aspects of IMRT and how this newer modality has changed the way most head and neck cancers can be treated.

KEYWORDS
Radiotherapy, Cancer.

Head and neck (H&N) cancers are usually treated with radiotherapy, surgery alone or a combination of surgery and pre- or postoperative radiation therapy. The greatest challenge of radiation therapy or cancer therapy is to attain the highest probability of cure with the least morbidity. In practice, the challenge lies in identifying the cancer cells and targeting them with radiation.

As distant metastases are rare, H&N cancers represent malignancies where loco-regional control is the most important factor determining survival. Local recurrence rates increase with increasing stage from around 0% for stage I, (American Joint Committee for Cancer[1]), up to 50% for stage IV tumours[2]. Besides the risk of a loco-regional recurrence, there is also a risk of treatment related side effects such as dry mucus membranes and radionecrosis while surgery can result in mutilation with cosmetic and functional loss.

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Advances in computer and Linear Accelerator technology in radiotherapy have significantly impacted treatment of head and neck cancers by improving the ability to maximize tumour dose while minimizing the dose to adjacent normal critical structures. Technical improvements in the application of X-rays, Computed Tomography scans, magnetic resonance imaging, ultrasound, positron emission tomography scans has greatly improved the ability to accurately target and localise the tumour. Image-based treatment planning has been widely implemented, facilitating both the planning and delivery of three-dimensional conformal radiation therapy (3DCRT).

Due to the complex anatomy of the H&N region there is an increased severity of radiation associated toxicity. It has always been difficult to deliver a strictly tumoricidal dose, sparing the vital structures in the near vicinity of the tumour owing to the geometrical complexity.

Radiation induce toxicities of H&N cancer can be acute toxicities which occur during the treatment period and are self limiting and delayed toxicities seen months to years after treatment and

can be permanent. Acute toxicities encompass manifestations like hair loss, reddening, itchiness and soreness of skin, mucositis, odynophagia, salivary changes and salivary viscosity. Late toxicities include xerostomia, sensorineural hearing loss as well as vision loss and most importantly osteoradionecrosis of the jaws. Radiotherapy can permanently damage the function of any salivary glands that are in the treatment area. Xerostomia bears negative impact on the quality of life by affecting functions of speech, swallowing.

IMRT was first conceptualised in the 1960s. However, the Intensity Modulated Radiation Therapy Collaborative Working Group made the treatment planning algorithm commercially available in 2001. Intensity modulated radiation permits modulation of the intensity of each radiation beam, so that each field may have one or more areas of high intensity radiation and any number of lower intensity areas within the same field, thus allowing for greater control of the dose distribution with the target. In this method, the clinician defines the accurate treatment volume and demarcates it from the normal tissue volume by means of computer algorithms to co-ordinate the beam. The computer optimises the shape of the radiation beam parameters and plans the delivery of the radiation dose. Thus, it is possible to specify dose restriction to the neighbouring normal tissue and vital structures close to the tumor. A particular form of IMRT is called serial tomotherapy, as 'slices' could be treated by a continually rotating gantry; With serial and helical tomotherapy, the intensity modulation is achieved whereby radiation is either delivered or not.

Clinical implications and application of IMRT in head and neck cancer

In the head and neck region, the distance between the gross tumour volume (GTV) and the areas at risk for microscopic disease (clinical target volume) and critical neighbouring structures such as the salivary gland is no more than a few millimetres. Therefore, head and neck is an ideal site for IMRT due to the complex geometry of this area and the greater severity of radiation-associated toxicity.

The advantages of using IMRT over conventional radiotherapy are multitude. Firstly, there is excellent maintenance of current tumour control rates while decreasing toxicity profiles. IMRT in H&N cancer strives to maintain conventional dose to primary tumour and at-risk nodal regions, while diminishing dose to adjacent normal tissue structures such as salivary glands and spinal cord. Secondly, this method strives to achieve an escalated target dose while maintaining acceptable levels of toxicity. The high dose volume conforms to the 3D configuration of the tumour and the sparing of the nearby normal tissue allow for achieving higher tumour dose thus achieving an increased therapeutic ratio[3]. This is thus a mode of "in field tumour boosting".

IMRT can prevent xerostomia by preventing undue dose to the parotid glands in about 70-80% of the patients. It is the single most important indication for H&N cancers if more than two thirds of the parotid gland is likely to enter the radiation field.

However, IMRT culminates certain issues which cannot be overlooked. As compared to conventional radiotherapy this method results in a greater target dose inhomogeneites. It can be a challenge to minimize unwanted "hot spots" within the GTV and even within the normal tissues. These "hot spots" can lead to a higher like-lihood of post treatment complications, depending on their location[4]. IMRT has a very sharp dose fall-off gradient between the gross tumour target and surrounding normal tissue. Therefore, accurate and optimal target volume delineation becomes essential.

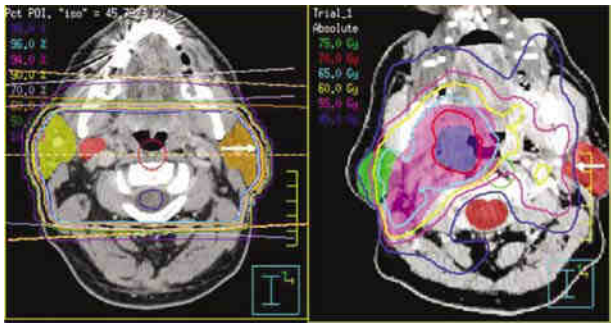

Fig: Isodose distributions of H&N treatment plans in conventional (left) and IMRT (right) methods. Significant reduction of dose to the left parotid gland is achieved with the IMRT plan.

(Courtesy: TS Hong, MA Ritter, WA Tome and PM Harari. Intensity-modulated radiation therapy: Emerging cancer treatment technology. British Journal of Cancer (2005) 92, 1819 – 1824)

GTV delineation is done in a multidisciplinary fashion, including a team consisting of a radiation oncologist, a neuro-radiologist, and, whenever necessary, a head and neck surgeon. Diagnostic information from different imaging modalities such as CT, MRI and PET are integrated to assist a radiation oncologist[5].

In a study of head and neck cancer patients by Kuppersmith et al[6] with respect to the normal tissue doses, the parotid gland received less than 30 Gy for midline tumours. Their incidence of acute toxicity was much lower than with conventional radiotherapy. Following clinical trials and assessment of results, capabilities of IMRT were stated as: (a) decreased normal tissue doses during re-irradiation of previously treated patients; (b) ability to accurately trace the cranial nerves while minimizing the dose to the parotid glands and other surrounding structures; (c) ability to administer varying doses to the primary site as opposed to the cranial nerves; (d) multiple targets could be treated simultaneously with an accelerated course. This technique was referred to as Simultaneous Modulated Accelerated Radiation Therapy (SMART).

Treating recurrent head and neck tumours with IMRT

The management of recurrent Head and Neck cancer is challenging especially when the patient has already received radical radiation therapy. If the patient is incapable of tolerating surgery, re irradiation can be considered. In such cases meticulous planning and radiation technique are required. Conservative margins around the tumour of no more than 1 cm are appropriate for re irradiation which can be meticulously achieved using IMRT. Improved target dose uniformity and lower doses to the vital structures can be thus achieved.

In conclusion it can be said that IMRT is a promising advancement in the field of radiotherapy which is expected to supersede other techniques including 3D conformational radiotherapy. This imaging and treatment modality has shown acceptable toxicity and encouraging efficacy thus bringing about clear opportunity to improve the therapeutic ratio for cancer patient outcome. It is believed that IMRT shall soon become the standard method of treatment for head and neck cancers.

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REVIEW ARTICLE

Phytochemicals - The natural fighters against oral cancer

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ABSTRACT

Oral cancer is one of the most common cancers worldwide. Genetic predisposition, tobacco smoking, alcohol consumption, betel quid chewing, infections (syphilis, oral candidiasis, viral infections to HPV), improper dietary intake like increased red meat consumption, lower fiber intake etc. are the various factors that increase the risk of developing cancer. It is estimated that roughly one-third of all cancers including oral cancer may be related to the type of diet and can be prevented by lifestyle and dietary measures to a certain extent. Fruits and vegetables are naturally low in fat and high in fiber and are important sources of chemo preventative elements called 'phytochemicals'. Phytochemicals act synergistically to protect the body, fight against diseases and boosting our immunity. Our aim is to discuss the anticancer properties of phytochemicals present in plant foods (fruits and vegetables).

KEYWORDS
Phytochemicals, Phytonutrients, Antioxidants, Anticancer foods, Cruciferous vegetables.

Introduction

Oral cancer ranks sixth among the different kinds of cancers worldwide. Along with the other accepted causative factors like tobbaco usage, excess alcohol consumption, areca nut usage, poor oral hygiene, UV light, genetic predisposition etc., dietary role has also been implicated as an important predisposing factor in the etiology of oral cancer. It has been found that diet with low levels of vitamins (namely A, C, E B_{12} etc.), inadequate consumption of vegetables (especially allium and cruciferous vegetables), nutrient sparse foods like concentrated sugars, refined flour products, low fiber intake, consumption of red meat etc. all contribute to the excess risk of developing oral cancer.

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It has been estimated by the American Institute for Cancer Research and the World Cancer Research Fund that 30-40 percent of all cancers can be prevented by appropriate diets incorporating a lots of fruits and vegetables, physical activity in form of exercise and maintenance of appropriate body weight.[1, 2]

A rapidly growing number of published studies show that a certain groups of food components are seen to help prevent cancer and sometimes even assist the body in fighting the disease if one already has cancer. These components called 'chemo protective' agents that include a group commonly known as 'antioxidants', appear to protect the body from the effects of carcinogenic elements[3].

Phytochemicals are biologically active chemical compounds in plants. The prefix "*Phyto*" is from a Greek word meaning '*plant*'. In plants, phytochemicals act as a natural defense system for host plants and provide colour, aroma and flavour. To date up to 4000 phytochemicals in plant foods have been identified with still many that are unknown. Any one serving of vegetables could provide as many as 100 different phytochemicals.

The best-known phytochemicals are Carotenoids, Flavonoids and Isoflavones. *Carotenoids* include yellow, orange and red pigments in fruits and vegetables. Dark, green leafy vegetables are rich in Carotenoids especially Beta Carotene but the usual yellow colour is masked by Chlorophyll, the green pigment in the vegetables. *Flavonoids* are reddish pigment, found in red grapes skins and citrus fruits. *Isoflavones* are found in peanut, lentils, soy and other legumes.

The most important action of these chemicals with respect to human beings is that they function as antioxidants that react with the free oxygen molecules or free radicals in our bodies. Free radicals can damage the cells of our bodies and must be removed.[4]

Review of Literature

Block et al reviewed about 200 studies of cancer and fruit and vegetable intake. A statistically significant protective effect of fruits and vegetables was found in 128 of 156 studies that gave relative risks. For most cancers, people in the lower quartile (1/4 of the population) who ate the least amount of fruits and vegetables had about twice the risk of cancer compared to those in the upper quartile who ate the most fruits and vegetables.[1, 5]

In a population based prospective study of Japanese individuals conducted by N. Kurahashi et al, 2009 showed that consumption of vegetables, green-yellow and green leafy vegetable had an inverse association with the development of cancer.[6]

Carcinogens can enter the body from all kinds of sources: tobacco smoke, pollution, pesticides, chemicals in food etc. Phytochemicals recognize them and block their action before they can induce cancer into a normal cell, for e.g. Garlic has immune enhancing allium compounds (diallyl sulfides) that increase the activity of immune cells that fight cancer and indirectly help break down cancer causing substances i.e. carcinogens. These compounds also helps block carcinogens from entering cells and slows down tumour development or even otherwise helps in rendering the carcinogens inactive.[7] Cruciferous vegetables are a potent source of the phytochemical, 'sulforaphane' which is an isothiocyanate that has an important action in modulating carcinogen metabolism thereby protecting against cancer. [8]

The anticarcinogenic effect of phytochemicals is mediated by certain mechanisms mainly:

- Detoxification and enhanced excretion of carcinogens
- Suppression of inflammatory processes such as cyclo-oxygenase-2 expression
- Inhibition of mitosis and induction of apoptosis at various stages in the progression and promotion of cancer. [9]

For example Genistein, an isoflavone present in soy products is seen to be associated with induction of tumour cell apoptosis and inhibition of tumour angiogenesis in vivo.[10]

Phytochemicals from edible plants have been reported to interfere with a specific stage of the carcinogenic process with recent attention been focused on intracellular-signalling cascades as common molecular targets for various chemopreventive phytochemicals.[11]

According to a study in the European Journal of Cancer Prevention (2009), both in-vitro and animal studies demonstrate that several phytochemicals, including curcumin, resveratrol, green tea catechins, oltipraz and silbinin possess important chemo-preventive and chemotherapeutic properties against cancer.[12] This kind of phyto-protective mechanism explains why cultures whose diets are rich in plant foods have the lowest rates of cancer. For example, the Mediterranean diet, emphasizes garlic, tomatoes, onions, fruits, whole grain and olive oil, all of which contain cancer fighting phytochemicals.

Normal cells utilize oxygen for generating energy and during this process molecules called free radicals are released. These free radicals have a net negative charge due to presence of an extra electron making them highly reactive, thus making them react with other cells. On doing so they damage the DNA and other molecular substances in cells. Damage to the structure of DNA is a precursor to cell transforming from normal to malignant. Phytochemicals in the form of antioxidant molecules (e.g. Carotenoids, Flavinoids) have a positive charge, which helps in neutralizing the negatively charged free radicals preventing them from damaging cells. As we get older the body's ability to repair itself diminishes with age and so our body requires such antioxidant molecules.

Antioxidants also help prevent damage by carcinogens such as UV radiation, tobacco smoke, environmental pollution etc.

Diet containing antioxidants are even more important for those undergoing treatment for cancer. Radiation and chemotherapy causes significant collateral damage to the body overall while they are attacking the cancer itself. As the treatment take a severe toll, a proper diet rich in antioxidant phytochemicals is needed to rebuild cells and fortify the body during treatment.

Studies have suggested that oral administration of green tea, which contains EGCG (Gallacatechin – 3 – gallate), an important polyphenolic constituent, has enhanced tumour-inhibitory effects of doxorubicin (an anti cancer drug). It was shown that intake of green tea seemed to increase the concentration of doxorubicin in tumour but not in normal tissue.[13] EGCG also inhibit the action of metalloproteinases, which are effectors of inflammation and extracellular matrix breakdown.[1]

Antioxidant phytochemicals mobilize the body's immune cells, called Natural Killer cells (N-K cells) and Helper T cells. They act as a protective barrier inhibiting enzymes that suppress immune response and stimulate cancer growth. Polyphenols like Proanthocyanidin present in grapes have strong antioxidant activity in the following ways: [14]

- free radical scavenging activity
- chelation of transition metals
- inhibition of cancer promoting enzymes

Phytochemicals operate under the "*Biochemical Principle Of Synergy*" (1+1=3). For e.g. Flavonoids and Carotenoids have more health promoting properties when they are eaten together in the same food rather than being taken separately in the form of a supplement. When considering the cancer fighting potential of a healthy diet, it is important to remember that many different foods contain different kinds of beneficial phytochemicals.

Evidence based research on potential synergy of phytochemicals suggests that the additive and synergistic effects of the phytochemicals in fruits and vegetables are responsible for the potent antioxidant and anti cancer activities and that the benefit of a diet rich in fruits and vegetables is attributed to a complex mixture of phytochemicals present in whole foods. This explains why, a single antioxidant cannot replace the combination of natural phytochemicals in fruits and vegetables to achieve the required health benefits. The evidence suggests that antioxidants or bioactive compounds are best acquired through whole-food consumption, not from expensive dietary supplements.[15]

When one phytochemical may bind to a carcinogen and prevent it from entering the normal cell, another phytochemical may remove carcinogens out of the cell, while still another may neutralize free radicals before they circulate around the body, and an other phytochemical may stimulate the body's' own enzymes to break up potential cancer causing chemicals. Certainly a multi vegetable salad is more cancer protective than an apple. Better still a salad for lunch and fruits as dessert would be most beneficial. It is recommended that about 5 to 10 servings of a wide variety of fruits and vegetables should be taken daily to reduce the risk of chronic illnesses including cancer.[15]

Food preparation also affects the state of the phytochemicals. Usually raw vegetables have more nutrients than cooked ones but sometimes this is not true. Cooking brocolli, releases the enzyme indole that fights cancer. Cooked tomatoes have more concentration of the phytochemical 'lycopene' than in a raw state.[1] Site-specific case control studies suggest a protective effect of high intake of raw and/or cooked garlic against cancer.[16] Crushing or chopping of garlic releases the enzyme allinase, to produce the active phytochemical, allicin. A substance called 'falcarinol' found in carrots has been found to reduce the risk of cancer according to researchers at Danish Institute of Agricultural Sciences (DIAS). This substance is a polyacetylen, however, so it is important not to cook the carrots.[7]

Apple contains a variety of phytochemicals including quercetin, catechin, floridzin and cholorogenic acid all of which are strong antioxidants. The phytochemical composition of apples varies greatly and there are also small changes in them during maturation and ripening procedures. Storage has little or no effect on phytochemicals, but processing seems to affect their bioavailability and antioxidant behaviour.[17]

The most popular phytochemicals that are known for their powerful antioxidant, anticancer, and heart disease protective properties are: [1, 7]

1. CAROTENOIDS :(alpha carotene, beta carotene, leutin, zeaxanthin): Yellow-orange fruits and vegetables, asparagus, carrots, cantaloupe, papaya, pumpkin, squash, sweet potatoes, broccoli, dried apricots, asparagus, kale, green leafy vegetables, and peppers
2. FLAVONOIDS: Soy, green tea, tomatoes, sweet potatoes, cruciferous vegetables, citrus fruits, red wine, red grapes, berries, onions, tapioca
3. ISOFLAVONES: Legumes (beans, peas, lentils), soy products
4. INDOLES: Cruciferous vegetables
5. SULFORAPHANE: Cruciferous vegetables
6. ALLICIN: Garlic, onions, scallions, leeks, chives
7. LYCOPENE: Tomatoes, tomato paste, tomato juice, guava, pink grapefruit, watermelon
8. BETA CRYPTOZANTHIN: Tangerines, papaya, oranges, peaches, mangoes, nectarines
9. GENISTEIN: Soy products (e.g., tofu)
10. POLYPHENOLS: Green tea
11. ANTHOCYANINS: Wild blueberries, strawberries, bilberries, black berries, grapefruits, citrus fruits
12. LIMONOIDS: Citrus fruits e.g. oranges, lemons
13. STEROLS: Cruciferous vegetables, cucumbers, squash, sweet potatoes, soy foods, eggplant, whole grains, tomatoes, tapioca
14. CAPSAICIN: Chili peppers, spices, jalapenos
15. ELEGIAC ACID: Strawberries, Blueberries, blackberries
16. LIGNANS: Nuts and seeds, flax seeds
17. POLYPHENOLS: Redwine, Green tea, Black tea

NOTE: Cruciferous vegetables include broccoli, cabbage, brussels sprouts, mustard greens, kale, turnips and cauliflower.

While nearly all plant foods contain health-promoting phytochemicals, the following are the most phyto-dense food sources: [18]

1. Soy
2. Tomato
3. Broccoli
4. Garlic
5. Flax seeds
6. Citrus fruits
7. Melons: cantaloupe, watermelon
8. Pink grapefruit
9. Blueberries
10. Sweet potatoes
11. Chili peppers
12. Legumes: beans, and lentils

Honorable mention: Green tea, red grapes, papaya, carrots, kale, nuts and seeds, eggplant, artichoke, cabbage, brussel sprouts, onions, apples, cauliflower, dried apricots, pumpkin, squash, spinach, mangos, and shiitake mushrooms.

Discussion

It is well understood that the dietary factor plays a key role in preventing oral cancer. Recent evidence has proved that phytochemicals present in fruits and vegetables play a major role in fighting cancer.

It is difficult to say that one phytochemical is more superior to other. Most of the phytochemicals have different anticarcinogenic activities with which it protects the body. All the anticancer actions of each phytochemical and their respective sources have been discussed in detail. The mechanism by which the phytochemical act, are also different. The most common anticancer action among the phytochemicals is the 'antioxidant action' by neutralizing the circulating free radicals, enhancing immunity and promoting cell differentiation.

Certain plant foods are considered to be more 'phytodense' (i.e. rich in phytochemicals) than others e.g. Soy, Tomato, Broccoli, Garlic, Flax seeds, Citrus Fruits, Melons, Grapefruit, Blueberries, Sweet potatoes etc.

Recent studies on ocean-based plants including marine phytoplankton too, has shown to provide unique antioxidant elements similar to that found in plant foods. Besides trace minerals, they contain literally hundreds of potent phytochemicals having similar anticancer properties as well.

Evidence suggests that regular consumption of a healthy diet rich in fruits and vegetables decreases the risk of chronic illnesses including cardiovascular

Table 1 - Table of Phyto Chemicals in Food materials and their anti cancer effects.

slno	food	Phyto chemical	Anticancer effect
1	Apples	Carotenoids- *Beta carotene*	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
		Flavonoids – *Quercetin, Floridzin*	Slows down cell division Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Improve lipid blood profile Remove toxin form tissue cells
		Polyphenols – *Catechin*	Act as antioxidants Reduce damaging effects of nitrosamines Kill human cancer cells
		Flavonolignans- *Silbinin*	Weak nonsteroidal compounds that block estrogen and androgen hormones from binding to their receptor which helps in retarding cellular proliferation
		Chlorogenic acid	Antioxidant action Block effects of free radicals; inhibit formation of nitrosamine (carcinogen)
2	Artichoke	Polyphenols	Act as antioxidants Reduce damaging effects of nitrosamines Kill human cancer cells
3	Avocados	Carotenoids – *Beta Carotene* obtained from Glutathione	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
4	Beans and legumes	Isoflavones	Act as antioxidants Stimulate enzymes that detoxify carcinogens Inhibit enzymes causing cancer
5	Berries (Strawberries, raspberries, blueberries, cranberries, bilberries)	Ellagic acid (Richest in strawberries and raspberries)	Neutralizes carcinogens in the liver Antioxidant Inhibits cancer cell division
		Polyphenols- *Anthocyanosides* (richest in blueberries)	Act as antioxidants Reduce damaging effects of nitrosamines Kill human cancer cells
		Flavonoids	Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation
6	Carrots	Carotenoids – *Beta Carotene*	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
		Sterols - *Falcarinol*	Prevent cells from becoming cancerous Lower fat levels in body
7	Cruciferous Vegetables (Broccoli, cabbage, Brussels's sprouts, Mustard greens, kale, cauliflower, turnip)	Carotenoids – *Lutein, Zeaxanthin*	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
		Indoles- *Indole 3-carbinol*	Convert cancer promoting hormones (e.g. estrogen) to an inactive state Stimulate enzymes that deactivate free radicals and detoxify carcinogens
		Sterols	Prevent cells from becoming cancerous Lower fat levels in body

diseases and cancer. Fruits and vegetables, which are a rich source of power packed nutrients called phytochemicals, are amazing health protectors, detoxifiers, and provide a 'nutrition power house' for the body. Molecular science is finally confirming that these phytonutrients that give fruits and vegetables their many colours, also provide a lot of 'Mother Natures's medicine'. The additive and synergistic effect of a complex mixture of phytochemicals obtained from a varied diet daily is responsible for the anticancer and antioxidant activities to protect the body's tissues, detoxify the blood and remove toxins, enhance oxygenation and circulation and reverse abnormal cell division that can lead to cancer. So its highly recommended that one should adopt a healthy diet incorporating a

Table 1 - continued....

		Flavonoids	Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Remove toxin form tissue cells Improves blood lipid profile
		Isothiocyanates – *Sulforaphane* (present mainly in broccoli, kale)	Antioxidant, enhances detoxifying effect of liver's enzymes to remove carcinogens
8	Chillipeppers, spices, jalapenos	Capsaicin	Prevents carcinogens from binding to DNA
9	Dark green leafy vegetables (spinach, mustard greens, romaine, chicory, swiss chard, parsley, lettuce)	Carotenoids	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
		Folic acid	Integral role in DNA methylation and DNA synthesis
10	Figs	Benzaldehyde	Effective in shrinking tumours
11	Flax seeds	Omega 3 fatty acids *(Alpha linolenic acid)*	Antioxidant action Blocks and suppresses cancerous changes Regulates production of prostaglandin the cell
		Lignans- *Phytoestrogens* (Enterodiol, Enterolactone)	Weak nonsteroidal compounds that block estrogen and androgen hormones from binding to their receptor which helps in retarding cellular proliferation
12	Garlic (including scallions, onions, leek and chives	Allium compounds- *Diallyl sulfides, S-allyl cysteine*	Increases liver enzymes to detoxify carcinogens Increase activity of immune cells Blocks carcinogens from entering into cell Retards tumour growth
13	Grape fruits (including citrus fruits)	Monoterpenes- *Limolene* (present mainly in oranges and lemons)	Decreases cell prolilferation by decreasing activity of ornithine decarboxylase (ODC), an enzyme that helps trigger the cancer-causing process Stimulate production of cancer killing immune cells Prevents expression of cancer-causing genes (oncogenes) Stimulates enzymes that detoxify carcinogens
		Polyphenols – *Proanthocyanidin*	Act as antioxidants Reduce damaging effects of nitrosamines Kill human cancer cells
		Flavonoids	Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Improve lipid blood profile Remove toxin form tissue cells
		Folic acid	Integral role in DNA methylation and DNA synthesis
		Ascorbic acid	Antioxidant action Reduce absorption of cancer causing nitrosamines
14	Grapes and Red wine	Flavonoids	Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Improve lipid blood profile Remove toxin form tissue cells

Table 1 - continued....

		Resveratrol	Block enzymes necessary for cancer growth and those that suppress immune response Prevent cell damage Anti inflammatory agent
		Ellagic acid	Neutralizes carcinogens in the liver Antioxidant action
		Polyphenols – *Proanthocyanidin*	Act as antioxidants Reduce damaging effects of nitrosamines Kill human cancer cells
15	Licorice root	Glycyrrhzin	Prevent growth of cancer
16	Mushroom	Lentinan	Polysaccharide that helps in building immunity Source of beta glucan
		Lectin	Attacks tumour cells and prevents them from multiplying
		Thioproline	Stimulates production of interferon
17	Nuts and seeds	Flavonoids – *Quercetin, Campferol*	Slows down cell division Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Improve lipid blood profile Remove toxin form tissue cells
		lignans	Weak nonsteroidal compounds that block estrogen and androgen hormones from binding to their receptor which helps in retarding cellular proliferation
		Selenium (component of glutathione peroxidase)	Decrease tumour growth Enhance the immune system Induces P450 enzymes leading to detoxification of carcinogenic molecules Active site of many enzymes including thioredoxin reductase which catalyse oxidation-reduction reactions that encourages cancerous cells to undergo apoptosis
18	Papaya	Ascorbic acid	Antioxidant action Reduce absorption of cancer causing nitrosamines
		Folic acid	Integral role in DNA methylation and DNA synthesis
19	Pumpkin	Carotenoids	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
20	Rosemary	Polyphenols – *Carnosol*	Act as antioxidants Reduce damaging effects of nitrosamines Kill human cancer cells
21	Sea weeds/ vegetables	Carotenoids –*Beta carotene*	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
		Chlorophyll	React with carcinogens to form complexes which are removed from the body
		Chlorophyllone	Important type of fatty acid that fights against cancer
		Vitamin B 12	Important for genetic stability, DNA repair, and for cancer therapy
22	Soy products (e.g. tofu)	Isoflavones- *Genistein*	Act as antioxidants Stimulate enzymes that detoxify carcinogens Inhibit enzymes causing cancer

Table 1 - continued....

		Lignans-*Phytoestrogens*	Weak nonsteroidal compounds that block estrogen and androgen hormones from binding to their receptor which helps in retarding cellular proliferation
		Sterols	Prevent cells from becoming cancerous Lower fat levels in body
23	Sweet potato	Carotenoids – *Beta carotene*	Act as antioxidants,neutralize free radicals, enhance immunity; promote cell differentiation
		Flavonoids	Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Improve lipid blood profile Remove toxin form tissue cells
		Sterols	Prevent cells from becoming cancerous Lower fat levels in body
24	Tapioca	Flavonoids	Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Improve lipid blood profile Remove toxin form tissue cells
		Sterols	Prevent cells from becoming cancerous Lower fat levels in body
25	Tomato	Lycopene	Protects from cell damage
		Flavonoids	Prevent attachment of cancer-causing hormones to cells by blocking receptor sites Decreases biological chemicals that influence cell growth and proliferation Improve lipid blood profile Remove toxin form tissue cells
		Sterols	Prevent cells from becoming cancerous Lower fat levels in body
26	Turmeric	Polyphenols -*Curcumin*	Assists the liver in detoxifying carcinogens; arrests cancer cells

wide variety of fruits and vegetables (mainly allium and cruciferous vegetables) daily, to meet the nutrient requirements for optimum health and to protect our body from cancer.

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ABSTRACTS FROM JOUNRALS

Variation in Periodontal Diagnosis and Treatment Planning Among Clinical Instructors

Sharon K. Lanning, D.D.S.; Scott D. Pelok, D.D.S.; Brent C. Williams, M.D., M.P.H.; Philip S. Richards, D.D.S., M.S.; David P. Sarment, D.D.S., M.S.; Tae-Ju Oh, D.D.S., M.S.; Laurie K. McCauley, D.D.S., Ph.D.

J Dent Educ. 69(3): 325-337, 2005

ABSTRACT

Consistency in clinical decision making may be necessary for reliable assessment of student performance and teaching effectiveness, yet little has been done to examine variation in periodontal diagnosis and treatment planning among dental school faculty. The purpose of this investigation was to examine variation among faculty in diagnosis and management of common periodontal diseases. Twenty-seven clinical instructors (periodontists, general dentists, dental hygienists, and first- and second-year periodontal graduate students) reviewed three web-based cases and answered a brief questionnaire focusing on radiographic interpretation, periodontal diagnosis, and treatment planning. Response rates for the three cases ranged from 62 percent to 70 percent. Clinical instructors' rating of percent bone loss in the majority of cases varied between three descriptive categories for the same tooth. Greater consistency in periodontal diagnosis was noted within the graduate student group as compared to periodontal and dental hygiene faculty groups. Diagnoses offered for one of the three patients varied between gingivitis and chronic and aggressive periodontitis. Six to nineteen different treatment plans (many with subtle differences) were submitted for each of the three cases. Inter-rater variation was qualitatively more prevalent than intra-rater variation. To our knowledge, this is the first study to document substantial variation among instructors in radiographic interpretation, diagnosis, and treatment planning for common periodontal diseases. Qualitative judgments speculating on the impact of variability among dental school faculty on student performance and patient care can be made but as yet remain unknown. Consistent use of accepted practice guidelines and greater consensus-building opportunities may decrease variation among faculty and enhance dental education.

A pilot study using remote broadcasting equipment to provide instruction in pedodontics

PK Domoto, P Weinstein, and T Getz
J Dent Educ. 43(11): 599-601, 1979
ABSTRACT

This study investigates the value of remote broadcasting equipment in instruction in pedodontics. Twenty students, between their junior and senior years, were randomly chosen and videotaped during an operative dentistry appointment with a child. Ten students reviewed and critiqued their own tapes immediately after the clinic session. Ten students received instruction using remote broadcasting equipment in an attempt to provide cues and immediate feedback. Results indicate the usefulness of remote broadcasting equipment as a new pedagogic technique in pedodontics.

Use of telemedicine for pre-implant dental assessment – a comparative study

Hans-Joachim Nickenig [*], Manfred Wichmann [*], Andreas Schlegel [*] and Stephan Eitner [*]
J Telemed Telecare 2008 ; 14:93-97
ABSTRACT

We evaluated real-time telemedicine for exchanging expert opinions in the area of pre-implant dental assessment. From 2003 to 2005, every tenth patient at the armed forces' dental clinic in Cologne-Wahn seeking implant counselling was discussed via videoconference (intervention group, $n = 85$). Indications, prosthodontic options, the required number of implants and implant positions were determined. The mean time required for the videoconferences was 3.5 min (range 1.0–9.5). In the control group ($n = 772$), the implant consultation was performed based on existing records, without using telemedicine. In three cases (3%), a basic change in the prosthodontic concept was required as compared to the telemedicine plan; in the control group, the concept changed in 7% of cases. The changes in the number and position of implants during therapy were also similar in the two groups. The results showed that telemedicine permitted satisfactory preoperative evaluation of the implantation operation.

Attitude and tendency of cheating behaviours amongst undergraduate students in a Dental Institution of India

M. Monica, A.V.Ankola, B. R. Ashokkumar and I. Hebbal

European journal of dental education; volume4; issue2; p79-83.

ABSTRACT

Honesty and integrity are key characteristics expected of a doctor, although academic misconduct amongst medical students is not new. Academic integrity provides the foundation upon, which a flourishing academic life rests. The aim of this study was to investigate the attitude of undergraduate dental students about the seriousness of cheating behaviours and to determine the rate of malpractice amongst these students. A self designed closed ended questionnaire was distributed to 300 undergraduate students in a Dental Institution in India, to rate the seriousness of six cheating behaviours and to assess the rate of malpractice. The response rate was 100%. Two of the six cheating behaviours were considered by at least 61% of the students as very serious cheating behaviours. Almost 70% of the students agreed that they have involved in malpractice in examinations at least once. The majority also felt that cheating in examinations will not have any significant effect on their future. This study has revealed that cheating is an important issue, which needs to be addressed for the benefit of the society at large.

Patient Risk Related to Common Dental Radiographic Examinations

The Impact of 2007 International Commission on Radiological Protection Recommendations Regarding Dose Calculation

John B. Ludlow, DDS, MS, Laura E. Davies-Ludlow, BS and Stuart C. White, DDS, PhD

J Am Dent Assoc, Vol 139, No 9, 1237-1243.

ABSTRACT

Background. In 2007, the International Commission on Radiological Protection (ICRP) revised estimates of the radiosensitivity of tissues including those in the maxillofacial region. The authors conducted a study to reassess patients' risk related to common dental radiographic exposures using the 2007 ICRP recommendations.

Methods. The authors used a tissue-equivalent head phantom to measure dose. They calculated effective doses by using both 1990 and revised 2007 ICRP recommendations. Effective dose is a calculation that takes into consideration the different sensitivities of organs to long-term effects from ionizing radiation. It is the preferred method for comparing doses between different types of exposures.

Results. Effective doses (per the 2007 ICRP) in microsieverts were as follows: full-mouth radiographs (FMX) with photo-stimulable phosphor (PSP) storage or F-speed film with rectangular collimation, 34.9 μSv; four-image posterior bitewings with PSP or F-speed film with rectangular collimation, 5.0 μSv; FMX using PSP or F-speed film with round collimation, 170.7 μSv; FMX with D-speed film and round collimation, 388 μSv; panoramic Orthophos XG (Sirona Group, Bensheim, Germany) with charge-coupled device (CCD), 14.2 μSv; panoramic ProMax (Planmeca, Helsinki, Finland) with CCD, 24.3 μSv; posteroanterior cephalogram with PSP, 5.1 μSv; and lateral cephalogram with PSP, 5.6 μSv. These values are 32 to 422 percent higher than those determined according to the 1990 ICRP guidelines.

Conclusions. Although radiographs are an indispensable diagnostic tool, the increased effective doses of common intraoral and extraoral imaging techniques are high enough to warrant reconsideration of means to reduce patients' exposure.

Clinical Implications. Clinicians can reduce patients' dose substantively by using digital receptors or F-speed film instead of D-speed film, rectangular collimation instead of round collimation and radiographic selection criteria.

ABOUT THE JOURNAL

The Trivandrum Dental Journal, the official publication of the Indian Dental Association , Trivandrum Branch, is intended to be a research periodical that aims to inform its readers of ideas, opinions, developments and key issues in dentistry - clinical, practical and scientific - stimulating interest, debate and discussion and an opportunity for life long learning ,amongst dentists of all disciplines. All papers published in the TDJ are subject to rigorous peer review by our excellent review board. We have tried to design the journal in such a way that the readers can find the relevant information fast and easily.

The journal is intended for dentists, dental undergraduates, members of the dental team, hospital, community, academic and general practitioners.

To start with, we have

Review articles: scientific peer-reviewed papers with a focus on clinical research to enable researchers and scientists to communicate their findings to the rest of the community.

Case reports: articles, and papers on the latest developments and information relevant for those in dental practice. This section contains essentially case reports and general articles about clinical matters.

Practice section:. to include clinical guide , how-to-do-it papers, dental business articles ,and the latest developments and information relevant for those in dental practice.

Abstracts: a selection of abstracts from dental journals.

We plan to include

Opinion section: intended to keep the readers aware of what people are thinking in dentistry today, and introduce differing views for debate by including letters and articles expressing the views and opinions of people that are open to debate and discussion.

Education section: any type of paper, article or report that is relevant to the vital subject of dental education, whether it is undergraduate, postgraduate, specialist or lifelong learning

Summaries: this section acts as a bridge between the practice and research sections, providing a summary of the research papers in this issue. Besides the abstract and 'in brief' box, in this page, we plan to includes a comment on each paper by a specialist in the field, emphasizing the relevance of the paper , to ensure that the information from the research is easily available to both practitioners and researchers.

The cover page design

The shanku or the conch was considered as one of the common emblems of majority of Kerala feudal kingdoms of the past, including Travancore. The official Kerala state emblem also symbolises two elephants guarding the imperial conch and its imperial crest. The graphical representation of the conch (' shanku') is adapted to be the design on the cover page of the TRIVANDRUM DENTAL JOURNAL.

The Cover Photograph : Cytokeratins, a group comprising at least 29 different proteins, are characteristic of epithelial and trichocytic cells. Monoclonal anti cytokeratins are specific markers of epithelial cell differentiation and have been widely used as tools in tumor identification and classification. Monoclonal Anti Pan Cytokeratin (mixture) is a broadly reactive reagent, which recognizes epitopes present in most human epithelial tissues. It facilitates typing of normal, metaplastic and neoplastic cells. Synergy between the various components results in staining amplification. This enables identification of cells, which would otherwise be stained only marginally. The mixture may aid in the discrimination of carcinomas and nonepithelial tumors such as sarcomas, lymphomas and neural tumors. It is also useful in detecting micrometastases in lymph nodes, bone marrow and other tissues and for determining the origin of poorly differentiated tumors. The cover photograph shows photomicrograph of central odontogenic fibroma, showing an island of odontogenic epithelium stained positively with Pan Cytokeratin.