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ORIGINAL RESEARCH ARTICLE

KNOWLEDGE, ATTITUDE AND PRACTICES ABOUT APRON HYGIENE AMONG CLINICAL DENTAL STUDENTS IN A DENTAL COLLEGE IN KERALA - A CROSS SECTIONAL SURVEY

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ABSTRACT

Background: Healthcare-associated infections (HAIs), also known as nosocomial infections, constitute a significant hazard for patients and their families visiting a healthcare facility. In a dental setup, white coatsare known to be potentially contaminated with pathogenic bacteria and there has been always a concern about the risk of transmitting pathogenic bacteria in clinical settings.Thus apron hygiene is a very important aspect of protective clothing. This study was undertaken with the objective of assessing the knowledge, attitude and practices regarding apron hygiene among clinical dental students and house surgeons in a dental college in Kerala.

Methodology: The study was a cross-sectional questionnaire based survey. The target population were the dental students and house surgeons. The questionnaire contained 20 questions to assess the knowledge, attitude and practice about apron hygiene. Results were expressed as a number and percentage of respondents for each question and were analyzed using Chi-square test.

Results: All the 106 respondents believed there was a necessity to wear aprons in clinic. About 89% opined that apron in worn for personal protection. About 45% are using the present apron since less than a year. About 83% of the respondents have 2 aprons or more. 78% prefer separate aprons for clinic and laboratories. 88% wore apron outside the clinic and laboratory premises among whom 95% wear it in canteen and over 50% wear it outside the college premises.

Conclusion: Although the knowledge and attitude regarding apron hygiene is good, the practice seems poor. A more serious approach towards inclusion and practice of apron hygiene in dental curriculum needs to be done.

Key Words: Apron hygiene, dental students, universal precaution, cross infection.

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Introduction

Healthcare-associated infections (HAIs), also known as nosocomial infections, constitute a significant hazard for patients and their families visiting a healthcare facility. The World Health Organization (WHO) defines such an infection as an infection occurring in a patient in a Health care facility in whom the infection was not present or was incubating at the time of admission. This includes infections acquired in the hospital but appearing after discharge, and also occupational infections among staff of the facility. [1]

A white coat, apron or laboratory coat (abbreviated lab coat) is a knee-length overcoat or smock worn by professionals in the medical field or by those involved in laboratory work to protect their street clothes. The garment is made from white cotton or linen to allow it to be washed at high temperature and make it easy to see if it is clean. [2]

In a dental setup, the environment in the working area is contaminated due to the aerosols produced by high-speed hand-pieces and ultrasound scalers. Hence, these white coats are known to be potentially contaminated with pathogenic bacteria and there has been always a concern about the risk of transmitting pathogenic bacteria in clinical settings.

Thus apron hygiene is a very important aspect of protective clothing to avoid cross contamination and transmission of infection between patients, dentists and auxiliaries. However literature reveals less satisfactory results pertaining to knowledge, attitude and practice regarding apron hygiene among dental students.

Thus, this study was undertaken with the objective of assessing the knowledge, attitude and practices regarding apron hygiene among clinical dental students and house surgeons in a dental college in Kerala.

Methodology

The study was a cross-sectional questionnaire based survey. The target population was the dental students and house surgeons of a dental college in Kothamangalam, Kerala. The study was conducted in the month of June 2015. A prefabricated validity tested questionnaire that was administered to the target population. The ethical approval was obtained from the Ethical Review Committee of the Institution. The questionnaire was divided into two parts. The first part consisted of questions on personal and professional data including age, gender, and year of study. The second part contained 20 questions on assessment of knowledge, attitude and practice regarding apron hygiene. All questions in the questionnaire were close-ended. The questionnaires were distributed by the House surgeons posted in the Public Health Dentistry Department. The respondents were asked to return the questionnaire immediately. All returned questionnaires were coded and analyzed. Results were expressed as a number and percentage of respondents for each question and were analyzed using the SPSS Version 17 software. Chi-square test was performed and the level of significance was set at $p < 0.05$.

Results

Table 1 shows the profile of the respondents

	N	PERCENTAGE
GENDER		
MALES	12	11.33%
FEMALES	94	88.67%
YEAR OF STUDY		
III YEAR BDS	19	17.92%
FINAL YEAR BDS	51	48.12%
HOUSE SURGEONS	36	33.96%

Table 2 shows the response of the study subjects to questions assessing the knowledge, attitude and practices regarding apron hygiene. All the 106 respondents believed there was a necessity to wear aprons in clinic. About 89% opined that apron in worn for personal protection. Majority of the respondents preferred full sleeve aprons over half sleeve. Majority of interns preferred full sleeve over half sleeve. About 45% are using the present apron since less than a year. The third year students wore newer aprons compared to interns. About 83% of the respondents have 2 aprons or more. The observation is significantly different among males and females (p=0.001) with almost 50% of the males owning just one apron. Almost 97% believe that contaminated aprons can act as a source of infection. The response showed a significant difference in relation to gender. (p=0.002). About 56% believe doctors are at risk. Only 6.6% wash their aprons daily. Almost 80% of the participants clean their aprons with detergent and majority of them dry it under direct sunlight. About two thirds of the respondents believe lab procedures soil the apron the most and about 78% prefer separate aprons for clinic and laboratories. About 9% agreed that they had a habit of exchanging apron with others. Almost all the study participants had the habit of keeping things in the apron pockets and about 88% wore apron outside the clinic and laboratory premises among whom 95% wear it in canteen and over 50% wear it outside the college premises.More than half the number of respondents carries their apron back to hostel/home either wearing them. Very few carry it in their bags or along with instruments. About 54% were interested in gaining more knowledge about apron hygiene.

Discussion

Wearing of aprons in dental colleges is mandatory as a universal precaution. Apron itself brings dignity to the profession. It helps for easy identification and made doctors look more professional. However, white coats have been shown to act as fomites and harbor potential contaminants.[3]

There has always been concern about the risk of transmitting the same in hospitals. The conclusion of many studies stated that white coats of doctors', nurses' uniform and other hospital garments, may play a part in the transmission of pathogenic bacteria in dental hospital settings. In case of dental healthcare professionals, the white coats are contaminated with splashes of blood, saliva and aerosols while providing the dental care which may be the important risk factor for infection with various organisms. There have also been several debates over whether doctors should be allowed or not to wear white coats in areas such as canteens, and libraries.[4] Hence, the knowledge, attitude and practices regarding proper apron hygiene is quintessential in serving its purpose. Hence this study was undertaken with the same objective.

All the 106 respondents believed there was a necessity to wear aprons in clinic. The institution rules mentioning the compulsory wearing of aprons as the protocol is reflected in this observation. In spite of this, it is noteworthy that less that 5% of the respondents only wore it out of compulsion.

An important finding is that about 16% managed with only one apron. This is in contrast to s study reported by Pydiet. al. in Andhra Pradesh[3] where the corresponding value was 8%. Because of the high frequency of the patient contact in a busy college environment, it is reasonable to expect the white coats to become colonized with potentially pathogenic bacteria. It has been also seen that the coats become contaminated quickly once worn thereby demanding the use of more number of aprons. The significant difference between males and females in this regard could be due to the skewed gender distribution of the study subjects.

About 45% are using the present apron since less than a year. The third year students wore newer aprons compared to interns. This showed that most of the respondents were using the same aprons from the beginning of their clinical posting in third year till internship.

It's a noteworthy observation that almost 97% believe that contaminated aprons can act as a source of infection. Males had a significantly lower knowledge in this regard compared to females. The knowledge is greater in this regard in comparison to a study conducted among medical students in Shivamogga in Karnataka[1] where only 84% believed it could act as a source of infection. This observation may be due to the fact that the Shivamogga study was mainly done among 2nd year medical students.

It was observed that about 96% washed their apron in a frequency of one week or less. This is in similar to the results of a study conducted in Manipal by Priya et al (2), Andhra Pradesh by Pydi et. al.(3) and

Sl. No.	QUESTION	RESPONSE	RESPONSE n (%)	p value (assessed by chi square test)
1	Do you think it is necessary to wear aprons in clinic?	Yes	106 (100%)	
		No	0	
2	What do you think is the relevance of wearing aprons in clinic?	Personal protection	95 (89.62%)	Gender: p= 0.76 (NS) Year: p= 0.278 (NS)
		Uniformity	13 (12.24%)	
		Sign of profession	39 (36.79%)	
		Compulsory	06 (5.66%)	
3	What do you prefer wearing?	Half sleeve cotton	11 (10.37%)	Gender: p= 0.77 (NS) Year: p=0.002
		Full sleeve cotton	34 (32.07%)	
		Half sleeve polyester	28 (26.41%)	
		Full sleeve polyester	32 (30.20%)	
4	Since how many years are you using the present apron?	< 1 year	45 (42.5%)	Gender: p=0.06 (NS) Year: p= 0.001
		1-2 year	31 (29.3%)	
		2-4 years	17 (16.1%)	
		>4 years	13 (12.3%)	
5	How many aprons do you own?	1	16 (15.1%)	Gender: p= 0.001 Year: p= 0.056 (NS)
		2	73 (68.9%)	
		3	13 (12.3%)	
		4 or more	04 (3.7%)	
6	Do you think contaminated aprons can act as a source of infection?	Yes	103 (97.2%)	Gender: p= 0.002 Year: p= 0.679 (NS)
		No	03 (2.8%)	
7	Whom do you think are most affected by contaminated apron?	Doctors	60 (56.6%)	Gender: p=0.707 (NS) Year: p= 0.062 (NS)
		Patients	41 (38.7%)	
		Chair side Asst	45 (42.1%)	
		Others	35 (33.2%)	
8	How often do you wash your aprons?	Daily	07 (6.6%)	Gender: p=0.010 Year: p= 0.475 (NS)
		Twice a week	58 (54.7%)	
		Weekly	36 (34%)	
		When it is soiled	04 (3.8%)	
		More than a week	00	
9	How do you clean your aprons	Detergent	84 (79.2%)	Gender: p= 0.001 Year: p=0.797 (NS)
		Drycleaning	22 (20.8%)	
		Disinfectant	00	
		Others	00	
11	How do you dry your aprons after washing	Direct sunlight	76 (71.7%)	Gender: p=0.01 Year: p=0.306 (NS)
		Indirect sunlight	16 (15.1%)	
		Under fan	13 (12.3%)	
		Using iron box	01 (0.9%)	
12	What makes your apron soiled the most?	Lab procedures	77 (72.7%)	Gender: p=0.964 (NS) Year: p= 0.057 (NS)
		Treatment procedures	40 (37.8%)	
		Assistance	37 (35%)	
		Handling of instruments	29 (27.4%)	
13	Do you think it is necessary to wear separate aprons for clinics and laboratories?	Yes	82 (77.4%)	Gender: p=0.641 (NS) Year: p=0.078 (NS)
		No	24 (22.6%)	
14	Do you exchange your apron with others?	Yes	9 (8.5%)	Gender: p=0.001 Year: p= 0.851
		No	97 (91.5%)	
15	Do you have the habit of keeping things in apron pockets?	Yes	104 (98.1%)	Gender: p=0.610(NS) Year: p= 0.394(NS)
		No	02 (1.9%)	
16	Do you wear aprons outside the clinic/lab	Yes	93 (87.8%)	Gender: p=0.153 (NS) Year: p=0.001
		No	13 (12.2%)	
17.	If yes where?	Canteen	89 (95.7%)	Gender: p=0.219(NS) Year: p= 0.670(NS)
		Outside campus	48 (51.6%)	
		Lectures	93 (100%)	
		Hostel	25 (26.9%)	
		Bathrooms	33 (35.5%)	
		Library	63 (67.8%)	
18	How do you carry your aprons back to home/hostel	In hands/wearing	56 (52.9%)	Gender: p=0.410 (NS) Year: p=0.767 (NS)
		With books in bag	18 (17%)	
		With instruments	7 (6.6%)	
		Leave in the department	24 (22.7%)	
19	Do you think it is necessary to change colour of apron?	Yes	13 (12.2%)	Gender: p=0.001 Year: p=0.237 (NS)
		No	93 (87.8%)	
20	Do you require any more knowledge about apron hygiene?	Yes	57 (53.4%)	Gender: p=0.919 (NS) Year: p=0.001
		No	49 (46.6%)	

Table 2: Response to questions assessing the knowledge, attitude and practices regarding apron hygiene

Pune by Saxena R. K et al (5),where the corresponding figure was 94%, 95% and 99% respectively.

Almost 80% of the participants clean their aprons with detergent and majority of them dry it under direct sunlight. However 20% dry it under indirect sunlight or fan, methods, not indicated for drying the aprons.

About two thirds of the respondents believe lab procedures soil the apron the most and about 78% prefer separate aprons for clinic and laboratories. As dental students, there is a significant amount of laboratory work in various departments. Although the amount of microbial contamination is lesser in laboratories compared to clinical environment, visible soiling and dirtying of aprons occur most commonly in laboratory settings. Hence separate lab coats for laboratories and aprons for clinical environments can be recommended owing to different nature of work and also to prevent cross contamination About 9% agreed that they had a habit of exchanging apron with others. The observation is in contrast with the Manipal study[2] where there was no exchange of aprons reported among under graduates. In the Andhra Pradesh study[3], however it was reported at 28% and in the Pune study 7% [5].

Yet another significant observation was that 88% wore apron outside the clinic and laboratory premises. This in in contrast with the Andhra Pradesh Study[3], where majority (80%) of the study participants wore their white coat only before entering the respective department or laboratory. Among them about 50% wore aprons outside college premises. This practice is more detrimental in maintenance of proper apron hygiene. In the study conducted at Shivamogga only 8% wore it outside college premises. However in this study, about 95% wore it to canteen and 35% to bathrooms. In the study conducted in Pune, 71% reported to wearing aprons while eating food [5]. All these practices can potentially be responsible for cross contamination and infections - a matter of concern. About 54% were interested in gaining more knowledge about apron hygiene.

Although significant difference in knowledge attitude and practice was observed in the study among males and females, a discussion in this regard might not be valid as the distribution of the study subjects in relation to gender is highly skewed.

Conclusion

The study portrays the knowledge, attitude and practices towards apron hygiene among clinical dental students and house surgeons in a dental college in Kerala. Although the knowledge and attitude regarding apron hygiene is good, the practice seems poor. A more serious approach towards inclusion and practice of apron hygiene in dental curriculum needs to be done. Hence to reduce bacterial contamination carried by dental healthcare professionals' aprons, there should be a ban on wearing of aprons in nonclinical areas such as canteen, classroom and library. It is recommended that guidelines should be there for handling and washing procedures of aprons. Further microbial studies on nature of organisms colonising the aprons are recommended.

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PSYCHIATRIC CHALLENGES IN ORTHODONTICS - A REVIEW

ABSTRACT

Many diagnosable psychiatric disorders are noticed right from childhood years. However psychiatric disturbances are more common in adolescent patients as it's a period of physical and mental transformations. This makes the role of orthodontists all the more important as this is the age when majority of patients seek orthodontic treatment. Also, treatment duration of 12-24 months and frequent appointments puts the orthodontist in a better position than clinicians of other disciplines in noticing such disorders in patients and making appropriate referrals. It is a well known fact that many of the psychiatric disorders that develop during adolescence show high suicidal tendency. This article reviews the etiology, diagnosis and management of various psychiatric disorders that an orthodontist is likely to encounter in his clinical practice.

Key words: orthodontics, psychiatry, adolescents.

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Introduction:

The psychological aspect of management is often ignored by orthodontists in their clinical practice. Considering the enormous physical and psychosocial changes that adolescents undergo, and the number of patients that belong to this age group seeking orthodontic treatment, the relevance of this aspect should be well understood by an orthodontist.

Cassidy1 reported that 14-20% of American children and adolescents develop diagnosable psychiatric disorders. Overall prevalence of mental disorders in India was analysed to be 65.4 per 1000 population in a study conducted by Madhav.[2] Overall rates of child and adolescent mental disorders in India range from 6-15%.3-5 Most of the psychiatric disorders have an insidious onset that can be traced back to the patient's childhood or adolescence.[6] Considering their frequent appointments with the patients and the long duration of treatment, orthodontists are at an advantage in noticing psychiatric changes in their patients. An intimate and combined working relationship between the orthodontist, patient and parents further helps the cause.

Teenagers are extremely susceptible to depression and illicit drug abuse secondary to peer pressure and other psychological compulsions at this tender age. The role of abusive (illicit) drugs as a cause suicidal tendencies and depression has been reported by Perkins[7] and Parker[8] respectively. Since orthodontic treatment has often been affected by suicidal tendencies, the orthodontist should be aware of the behavioral patterns and risk factors associated with this undesirable outcome.[9]

This article aims to review the etiology, noticeable behavioral patterns and suggested orthodontic management of such adolescent patients with psychiatric disorders.

Mood disorders

Major Depressive Disorder (MDD) and Bipolar Disorder (BD) are among the two most common mood disorders seen in adolescent children. Though episodes of depression are common to both, BD is better characterized by maniac episodes and marked mood swings.[10] These disorders are further characterized by their tendency to relapse and are associated with peer pressure.[11,12]

Mood disorders often result in gloominess, hopelessness, depression and suicidal behavior. Patients also report with sleep difficulties, loss of appetite and guilt feeling. Patients show a general lack of compliance and lack of interest in treatment. Oral hygiene will be typically poor and patient often blames self for treatment failure.[10]

MDD is a strong predecessor of suicides in adolescents and the risk is directly proportional to the length of depressive episodes.[13] Risk factors include substance abuse, personality disorders, anxiety disorders and disruptive disorders.[14] The symptoms include severe depression, gloom, lack of interest to communicate, hopelessness and above all suicidal thoughts and tendencies.

The prevalence of depression in Indian population is estimated to be 31.2 per 1000 population.2 Also, Indian females are found to be more commonly affected than males.[15,16]

The orthodontic implication and relevance of such adolescents include- missed appointments, lack of compliance, poor oral hygiene, lack of interest in treatment and self-blame for the same.

On the other hand, BD is not commonly seen in children and adolescents. The symptoms include manic episode, grandiose thoughts, decreased sleep, pressured speech, racing thoughts, distractibility, irritability, tantrums, appetite changes, psychotic symptoms etc. Attention-deficit hyperactivity disorder (ADHD) and conduct disorders are the sequel to this disorder.[17] The prognosis of BD is poor and the regimen used for adults is not effective for adolescents.[18]

Management of patients with mood disorders can be quite challenging for the orthodontist. Xerostomia is a common side effect of drugs like tricyclic antidepressants, anticonvulsants and selective serotonin re-uptake inhibitors which form the core of regimen used in such disorders. Moreover, patients with mood disorders often report with rampant caries and periodontal problems.[19] Suicidal tendency and lack of compliance further multiply the difficulties faced by the orthodontist.

Ideally, good control of these disorders should be attained before the start of treatment. Unfortunately, mid- orthodontic treatment diagnosis is bound to

occur, and the orthodontist should then decide whether to continue treatment until better disease control is attained.[20]

Eating disorders

Anorexia nervosa and bulimia nervosa are the two common eating disorders seen in adolescents, especially girls. The mortality is higher for Anorexia nervosa (10%) within ten years of diagnosis as compared to bulimia which is (1%) within 10 years.[21]

Clinical symptoms include lack of interest towards food, depression, lack of sleep etc. Oral manifestations of eating disorders include dental caries, dental erosion, dentinal hypersensitivity, periodontal disease, cheilosis, salivary gland hypertrophy, extrusion of amalgam restorations and xerostomia.[22,23]

Questions asked about satisfaction with eating patterns such as eating in secrecy often help to detect an eating disorder.[22] It would not be atypical for the orthodontist or the dentist to be the first person to notice an adolescent's eating disorder, because of the close working relationship.[20]

Personality disorders

Personality disorders exist when the way of thinking and habitual behaviors of individuals are rigid (inflexible) and maladaptive resulting in social, work, or school problems.[10] Like many of the other psychiatric disorders, personality disorders are believed to have their roots developed during the adolescent period,[10] Estimated prevalence of personality disorders is in the range of 4.4% to 13.0% .[24] Risk factors associated with the development of personality disorders in childhood or adolescence include poor family support, physical or sexual abuse, family disruption or criminality, and peer influences.[24] About 55-65% of patients with suicidal behavior are found to be suffering from personality disorders.[25]

Diagnosis and treatment of these disorders are difficult and a pretreatment questionnaire is of no use usually. The orthodontist should consider personality disorder as a possibility, if the patient's behavior is much stronger than normal- either negative or positive.[26] The orthodontist might find it difficult in trusting these patients because patients with personality disorders, particularly BPD, actively attempt to manipulate and provoke the clinician.[26,27]

Searight has suggested various strategies to handle such difficult patients or those with suspected personality disorder.[27]

Attention-deficit hyperactivity disorder (ADHD)

ADHD is a common disorder in children and young adults and is characterized by inappropriate hyperactivity, forgetfulness, impulsivity, and inattention.[10] Studies have shown that this is a lifelong disorder manifesting as greater activity (hyperactive behavior) than seen in peers.[28] A study conducted in Chandigadh, India 2 out of 186 followed up cases were diagnosed with ADHD.[16] Another study found the prevalence of ADHD in 6-12 yr old Indian children to be 4.67%.[29]

The disorder has a strong genetic inheritance with around 10% to 35% risk to immediate family members.[30] These children often have oppositional, overactive, defiant and/or conduct disorders.[47-49]

Children with ADHD are ten times more likely to develop antisocial personality disorder(APD) than those without it.[50] Adults with ADHD are prone to exhibit personality disorders, drug or alcohol abuse, mood disorders, and anxiety disorders.[51-54] In India, ADHD was found to be more common in males than in females.[16] The risk of suicide has been shown to be greater in males than in females in cases diagnosed as ADHD.[55] Although stimulants are the mainstay drugs used in the treatment of ADHD, antidepressants and adrenergic agonists are often used in refractory patients.[31] It is important for the orthodontist to note that Elia[31] has suggested that there can be growth disturbances in children taking methylphenidate. They also reported that discontinuation of the drug during the summer alleviated the chances of growth disturbances in control groups.[31] On the contrary, Klien[32] reported no deleterious growth effects in children taking stimulants. It is interesting to note that Spencer detected evidence of growth disturbances in children associated with ADHD.[33]

Orthodontic management of children and adolescents with ADHD is challenging because the patients often exhibit non-compliance with the home-care instructions and their improper behavioral patterns during office visits.[20]

More frequent appointments and involvement of the

parents is a must for placement of elastics and activation of appliances. Shorter appointments and frequent oral hygiene maintenance counseling during treatment can ensure better patient behavior.[20]

Schizophrenia

Schizophrenia is characterized by illogical thinking, delusions and hallucinations. However, it should not be mistaken with common childish behavior of that age. The onset of the disorder usually occurs during adolescence, but prevalence steadily increases with age unlike what happens with normal children.[34]The prevalence of this disorder in an Indian population was estimated to be 2.3 per 1000 population.[2]

The classical symptoms of schizophrenia include both positive and negative symptoms. The positive symptoms like delusions, hallucinations and agitation and negative symptoms like inability to pay attention, loss of will and social withdrawal are classical in most cases.[10] It is often the negative symptoms that pose a challenge for the psychiatrist and the orthodontist. The patient compliance is usually very low.

Overall prognosis is poor in adolescent-onset schizophrenia, with only 25% of patients achieving partial remission.[35] Treatment of schizophrenia involves anti-psychotic drugs which may cause xerostomia. They also cause side effects like dystonia and tardive dyskinesia which questions the usage of intraoral appliances. Risk of soft tissue laceration outweighs the potential benefits with continued treatment and as such are seldom recommended.[20]

Patients typically ignore oral hygiene instructions and show laxity in wearing of elastics.[36] Patients with florid schizophrenia almost always are not candidates for orthodontic therapy.[20] Even patients under control with medication should be approached cautiously. The treatment should always be rendered only with psychiatrist's consent.[20]

Substance abuse

Substance abuse has been linked to many cases of depression[7] and suicidal behavior[8]. It is a significant problem seen in adolescents and adults. A study done in America revealed that a significant number of patients receiving orthodontic treatment use at least one illicit drug during adoloscence.[37] Tobacco, alcohol, opiates, amphetamines, marijuana and steroids are some of the commonly used addictive drugs. Treatment in these types of cases warrants a combination of counseling and pharmacotherapy.[38]

In patients with substance-abuse problems, changes in the orthodontic treatment regimen will be necessary.

Wendell et. Al have outlined the modifications required for managing a patient with known drug abuse.[38]

1. It may be prudent to remove fixed appliances and place the patient in a temporary retention phase until therapy can be safely resumed.
2. Smooth-surface caries can be rampant in those with substance-abuse problems even without orthodontic appliances. A fixed appliance might accelerate or worsen the existing decalcification in such patients.
3. Treatment time could increase significantly if compliance is affected by a substance-abuse problem.
4. Although it is not within an orthodontist's capacity to treat substance- abuse disorders, it is important to help such patients by appropriate referral.

Because of frequent visits and long duration of treatment, orthodontists have the chance to build a good rapport with the patient. Knowing an adolescent patient for such a long duration helps the orthodontist detect changes secondary to substance abuse readily.

Discussion

The possible relationship between psychiatric disorders and orthodontic treatment has not been studied much. However, the research on older age groups though available, is beyond the scope of orthodontic therapy.[39-42] Studies have suggested that pre-surgical psychological assessment should not be used to predict the changes in mental state during postoperative orthodontic treatment as children transform rapidly in forms of health.[43,44] Hence, for the same reason, compliance with orthodontic treatment cannot be predicted by evaluating pretreatment personality traits of the patient.[45,46] However, the pretreatment evaluation should be performed, with appropriate

screening questions asked either directly or as a questionnaire. Mental status questions concerning prior suicide attempts, psychiatric therapy, counseling, school or legal trouble, substance-use patterns, and abuse situations should be on the evaluation form or asked verbally to the parents.[38]

The common feature seen in all the disorders discussed in this review is the suicidal tendency. Orthodontist should pay attention to changes seen in patient behavior throughout the course of treatment and also be prudent enough to assess what is normal and what isn't. A decrease in patient compliance or worsening of oral hygiene should alert the orthodontist about a possible change in psychological status of the patient.

A psychiatric referral should be preceded by a discussion with the parent/s. It should always be remembered that psychiatric diseases are viewed upon negatively by the society and the orthodontist should display utmost sensitivity when the issue is discussed with the parents.

Diagnosis given by the psychiatrist should not be discussed with the patient or parent directly and requires skill to explain and put it across with compassion. Working with institutes related to special care for children such as spastic societies , physically challenged children, blind homes etc. teaches the art of handling both the parents and patients with compassion and confidence.

Conclusion

The prevalence of psychiatric disorders world over is not negligible. Children and adolescents undergo a number of mental changes rapidly. Orthodontist may be the first to encounter psychiatric problems in adolescent patients. Familiarity with psychiatric disorders will inspire the orthodontist to offer better care for such patients, as he understands the delicate dimension of this seemingly diversified treatment need.

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SUPPORTIVE PERIODONTAL THERAPY

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ABSTRACT

Supportive periodontal therapy is performed at certain intervalsso as to reduce the disease progressionand to assist the patient in maintaining oral health.A thorough evaluation of the periodontal condition is necessary inorder to remove the persisting subgingival deposits. Patients with mild gingivitis are being recalled twice in a year where as periodontitis patients are recalled within three months after completing scaling and rootplaning.This treatment evolved from traditional dental prophylaxis and now emphasizes treatment of areas of previous attachment loss and areas where clinical signs of inflammation are found.

Keywords: Maintenance Therapy, Periodontitis, Supportive Periodontal Therapy.

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Introduction

Periodontitis is a multifactorial disease and can be treated effectively using nonsurgical and surgical periodontal therapy.[1]However in some situations subgingival plaque is left behind and it regrows within the pocket and causes inflammation. Inorder to establish conditions that are conducive to future optimal plaque control and to prevent subgingival bacterial growth a regular program of clinical revaluation is essential.[2] The maintenance of periodontal health requires considerable effort from both the patient and dental team. Thus, following the completion of active therapy, most patients need professional assistance at regular intervals for maintaining a proper oral hygiene. It involves improved motivation and instruction for oral hygiene, elimination of calculus and other plaque retentive factors and thorough professional cleaning of teeth. Such professional assistance has been termed as maintenance therapy, maintenance care, periodontal maintenance, periodontal recall, maintenance care or supportive periodontal therapy(SPT)[3]. These procedures are performed at selected intervals to assist the periodontal patient in maintaining oral health. The maintenance phase of periodontal treatment starts immediately after the completion of Phase I therapy.

Objectives and Therapeutic Goals

The main objective of supportive periodontal therapy (SPT) is to support the results of the initial therapy through a periodic professional recall system and maintenance of optimal plaque control, supragingivally and subgingivally, as well as to find out and remove irritants that were not eliminated during the treatment and healing phase.

The therapeutic objectives of supportive periodontal therapy are:

- To arrest the progression and recurrence of periodontal disease in patients who have previously been treated for gingivitis and periodontitis.
- To prevent the loss of dental implants after clinical stability has been achieved.
- To diminish tooth loss my monitoring the dentition and any prosthetic replacements of the natural teeth; and
- To identify and manage, in a timely manner, other diseases or conditions found within or related to the oral cavity.[4,5.]

Types of SPT

Preventive SPT -designed to prevent the inception of disease in individuals without periodontal pathosis.

Trial SPT-designed to maintain border line periodontal conditions over a period to assess the need for corrective therapy for problems-inadequate gingiva, gingival architectural defects, furcation defects, while maintaining periodontal health.

Compromise SPT- designed to slow the progression of disease in patients for whom periodontal corrective therapy is indicated, but cannot be implemented for reasons of health, economics, inadequate oral hygiene, or other considerations

Post treatment SPT- designed to prevent the recurrence of disease and maintain the periodontal health achieved during therapy.[6]

Rationale of SPT

Occasionally lesions may recur due to inadequate plaque control, failure of the patient to return for periodical checkups, presence of any uncontrolled systemic disease that may affect host resistance and inadequate restorations. Properly performed SPT can evaluate and correct all these factors thereby preventing further periodontal breakdown. So patients with a history of periodontitis usually require periodic SPT since personal supragingival oral hygiene alone has not been shown to control attachment loss in them.[2]

Clinical Diagnosis of SPT

The patient's risk assessment for recurrence of periodontitis may be measured on the basis of a number of clinical conditions in which both the risk factors and risk indicators are also evaluated. It includes bleeding on probing, residual pockets

greater than 4mm,bone loss, furcation involvement, crowding, malposition of tooth, iatrogenic factors, systemic diseases, genetic conditions and environmental factors. A risk assessment at subject, tooth and site level may be useful in evaluating the prognosis of periodontal disease activity and determining periodontal stability and may indicate the need for specific therapeutic measures during SPT visits[7]. A high risk patient has got increased chance of recurrence of disease[8].

Frequency of SPT

Numerous studies have shown that less attachment loss occurs, and fewer teeth are lost when patients maintain regular SPT. Patients with suboptimal plaque control and/or concomitant high prevalence of bleeding sites should be recalled more frequently than patients exhibiting excellent plaque control and healthy gingival tissues. Patients with healthy gingival conditions, but with a severely reduced height of periodontal support, should also be recalled with short time intervals (not exceeding 3-4 months) in order to exclude or at least reduce the risk of additional tooth loss.[9] It seems reasonable to commence post-therapeutic maintenance with recall visits once every 3-4 months and then shorten or prolong these intervals in accordance with the other aspects.[5]

Recall Intervals for various classes of recall patient has been included in Table 1.

Compliance with Supportive Periodontal Schedule

If patients fail to practice effective plaque removal and keep recall appointments, recurrence and progression of disease will occur despite the best efforts of periodontal therapists.[10] Both prospective and retrospective studies have proved that patients who comply in maintenance therapy are able to maintain periodontal health status, including reduced probing depth, less bleeding on probing, and reduced plaque index. Several patients do not comply due to fear of dental treatment, economic factors, health beliefs and stressful events in their lives.[11]

Effectiveness of SPT

Several studies have documented that periodic professional prophylactic visits in conjunction with reinforcement of personal oral hygiene all effective in controlling gingivitis. Patients presenting with mild to moderate periodontitis, have shown that less attachment loss occurs and fewer teeth are lost when they maintain regular SPT intervals. However, patients seeking SPT less than once per year over 8 year lost further periodontal attachment during the period of observation. Adjunct use of local drug delivery agents have demonstrated significantly more gain of attachment and decrease in mean pocket probing depth among periodontal patients. Patients with implants are susceptible for periimplantitis and are more prone to plaque induced inflammation with bone loss than those with natural teeth. So patients with oral implants require continuous supervision, evaluation of the soft tissues and interceptive prophylactic measures in the same way as patients with a natural dentition who are susceptible to periodontal disease. After uncovering the implants, patients must use ultrasoft brushes, chemotherapeutic rinses, tartar controlled pastes, irrigation devices and yarn like material to keep implants and natural tooth clean. Only plastic instruments should be used for calculus removal.[12] It is directly clear that some patients should be referred to a specialist, whereas most patients clearly have problems that can be treated by a general dentist. The decision to have the general practitioner treat a patient's periodontal problem should be guided by a consideration of the degree of risk that the patient will lose a tooth or teeth for periodontally related reasons. [13]

Conclusion

All types of Periodontal and Implant Therapy require continous recall visits and periodontal maintenance care because of the constant microbial challenge, and this response must be effective to prevent further tissue damage. Successful periodontal therapy with regular SPT can promote periodontal health and reduce tooth loss. Following active periodontal therapy, an interval is established for

periodic ongoing care. Active periodontal therapy consists of non-surgical and/or surgical treatment. An interval of three months between appointments appears to be an effective treatment schedule, but this can vary depending upon the clinical judgment of the dentist and the disease status of the patient.

Merins Classification 1996	***Characteristics***	***Recall internal***
First year	First year patient routine therapy and uneventful healing or First year patients difficult case with complicated prosthesis, furcation involvement, poor crown to root ratio, or questionable patient co-operation	3 months 1 to 2 month
Class A	Excellent results well maintained for 1 year or more patients displays good oral hygiene, minimum calculus, no occlusal problems, no complicated prosthesis, no remaining pockets, and no teeth with **less than 50% of alveolar bone remaining**	6 months to 1 year
Class B	Generally good results maintained reasonably well for 1 year or more, but patient displays some of the following factors 1. In consistent or poor oral hygiene 2. Heavy calculus formation 3. Systemic disease that predisposes to periodontal breakdown 4. Some remaining pockets. 5. Occlusal problems 6. Complicated prosthesis	3 to 4 months

	7. Ongoing orthodontic treatment 8. Recurrent dental caries 9. Some teeth with less than 50% of alveolar bone support 10. Smoking 11. Positive genetic test.	
Class C	Generally poor results following periodontal therapy and / or several negative factors from the following list : 1. Inconsistent or poor oral hygiene 2. Heavy calculus formation 3. Systemic disease that pre disease to periodontal breakdown 4. Remaining pockets 5. Occlusal problems 6. Complicated prosthesis 7. Recurrent dental caries. 8. Many teeth with less than 50% of alveolar bone support 9. Smoking 10. Positive genetic test 11. Periodontal surgery indicated but not performed for medical, psychologic or financial reasons 12. Conditions too for advanced to be improved by periodontal surgery. 13. More than 20% of pockets bleed on probing	1to 3 months

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TRANSMISSIBLE SPONGIFORM ENCEPHALITIS(TSE) OR PRION DISEASES; A NEED TO BE AWARE!

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ABSTRACT

Prion diseases are an interesting but puzzling group of diseases affecting both animals and human, that transmits their infectivity by a misfolded protein to the host. It then recruits other normal proteins to form a β pleated amyloid sheet which affects the neurons in an as yet undefined manner causing the various symptoms. The principal site of pathology is in the brain. Many diseases have been found to be caused by prions over the last century or so and new ones are being discovered. The infectious agent is unique as it does not have any DNA or RNA and it has a long incubation period. However they evoke no immune response leading to anoninflammatory pathologic process limited to the CNS. This article provides a short review of the several diseases caused by prions, and the agent causing infection. The precautions that need to be taken by healthcare workers are also discussed.

Key words: prion diseases; transmissible spongiform encephalopathy (TSE); prion protein; kuru; CJD, scrapie.

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Introduction

Transmissible spongiform encephalitis (TSE) or prion diseases are an enigmatic group of disease that has gathered tremendous interest since the last century. The prion diseases are a closely related group of rare incurable neuro degenerative diseases affecting both humans and animals. However, they elicit no immune response, resulting in a non inflammatory pathologic process confined to the central nervous system, usually have an incubation period stretching many years, and are invariably fatal within 1 year after diagnosis[1-2].

The classical triad of spongiform vacuolation, astrocytic proliferation and neuronal loss, and accompanied by the deposition of amyloid plaques usually, was proposed by Beck and Daniel in 1987 and is a common finding in both animal and human prion diseases[3]. The agent that produced the infection was at first thought to be a slow virus; but a virus was not isolated in this disease which led to further investigations that revealed the agent to be an indigenous protein particle which was later named as prions by Prusiner[4]. It can be infectious, inherited or develop sporadically. Several types of diseases are now out in the scientific domain starting with the discovery of scrapie that was initially mentioned in the scientific literature about 1732.

Animal prion diseases

I) Scrapie

ii) Bovine spongiform encephalopathy

iii) Transmissible mink encephalopathy

iv) Chronic wasting disease

v) Feline spongiform encephalopathy

Human prion diseases

I) Kuru

ii) Creutzfeld -Jakob disease

iii) Gerstmann-Straussler-Scheinker disease

iv) Fatal familial insomnia

v) Variably protease-sensitive prionopathy (VPSPr)

Scrapie

Scrapie has been demonstrated since 18th century, initially in the reports by shepherds. Fearing economic ruin they would keep it a secret from the veterinarians resulting it being unknown to the general public[5].

The afflicted sheep showed clinical signs lasting from early as two weeks to six months or more showing peculiar social behaviour and hyper excitability to human exposure. The condition of sheep gradually deteriorates, and there might be a change in colour of the fleece which may be the first change noticed by the shepherd. The sheep shows a tendency to rub against fences apparently due to pruritis and usually bites the affected area leading to denudation. Ataxia is usually common. In the final stages of the disease, although the appetite may appear normal, the animals lose the ability to feed themselves and the condition degenerates. The reproductive ability of the sheep is not affected even though affected by scrapie, but later muscle atrophy can interfere with the ability to move. Lambs can, therefore, be born successfully to mothers in the clinical phase of the disease and rams remain fertile and active even when affected by ataxic signs[6].

Scrapie was earlier thought to be a hereditary, infectious, or sexually transmissible disease. After the demonstration of scrapie to be a transmissible disease in 1936, it took many more years until the infectious agent - the prion - could be identified.[7-8]

Bovine spongiform encephalopathy

BSE is a prion disease affecting cattle first observed in the United Kingdom in 1986, starting as an epidemic that has since come down since 1993. It was surmised that consumption of cattle feed contaminated with meat and bone meal (MBM) initiated the disease. Why the United Kingdom was affected most was suggested to be because of several factors, including a high ratio of sheep to cattle; a relatively high rate of endemic scrapie; the heavy feeding of MBM to dairy cattle; and changes in the meat processing operation used to prepare MBM[9].

Production of clinically and pathologically similar

CJD in macaques by intracerebral injection of brain material from affected cows[10] and observing the biochemical similarities between human and BSE cases[11] suggest that BSE is transmissible to man. Infection from cattle to people was theorised as to the development of a variant of Creutzfeldt-Jakob disease (vCJD) [12].

Animals like deer, elk and cat apart from sheep and cow are also susceptible to prion diseases; symptoms usually are similar to that seen in cattle and sheep.

Kuru

A strange disease among the Fore tribe in Papua New Guinea was investigated by Gajdusek and Zigas in 1957, where a section of the tribe had tremors and loss of coordination. Ritual cannibalism was found to be the cause due to the thorough research by these scientists and others. The first case, as reported by their oral history dated back to 1920s. The disease primarily affected women and children. This was later found to be due to the fact that women and children used to consume dead relatives, the act which was thought to free up the spirits of the dead[13].

Kuru is a cerebellar syndrome with a characteristic and relentless progression through defined clinical stages, and is invariably fatal. The illness might last for one year or less. The patient becomes aware of the disease after noticing altered gait preceded by headaches and limb pains. Truncal instability, ataxia and tremor of the head and extremities aggravated by cold soon follow. Slurred speech, dysphagia, convergent strabismus, involuntary movements, emotional lability and mild dementia appear in most cases. Morphologic changes are seen in only the central nervous system[14].

William Hadlow noticed the similarity of the pathology in kuru and scrapie, which led scientists to the discovery of more similar diseases[15]. The cause of the diseases however still eluded scientists. Genetic, endocrine, hormonal, and toxic causes were explored and an infectious cause also proposed due to spread from man to man by way of cannibalism.

Creutzfeld-Jakob disease

It was first described in the early 1920s by two German neurologists[16-17]. The many different types included sporadic, familial and iatrogenic forms of the disease. Creutzfeldt-Jakob disease (CJD) is a rare, neuro degenerative, invariably fatal brain disorder. It affects about one person in every one million people per year worldwide; in India the cases are usually underreported[18]. CJD usually appears in middle age and runs a rapid course. Typically, symptoms start to be seen about age 60, and about 90 percent of individuals die within 1 year of contracting the disease. In the early stages of disease, people may have failing memory, behavioural changes, lack of coordination and visual disturbances. As the illness progresses, mental faculty deteriorates, involuntary movements, blindness, weakness of extremities and coma may occur.

There are three major types of CJD:

- In sporadic CJD, the disease appears even though the person has no known risk factors for the disease. It is thought that the normal form of the prion protein is changed into the infectious type of the protein and consequently changes all the normal proteins in a cascading pattern. This is by far the most common type of CJD and accounts for at least 85 percent of cases.

- In hereditary CJD, the person has a family history of the disease and/or tests positive for a genetic mutation associated with CJD. About 5 to 10 percent of cases of CJD in the United States are hereditary.

- In acquired CJD, the disease is transmitted by exposure to brain or nervous system tissue, usually through certain medical procedures. There is no evidence that CJD is contagious through casual contact with a CJD patient. Since CJD was first described in 1920, fewer than 1 percent of cases have been acquired CJD[19].

Variant CJD (vCJD) is another type, in which the disease is thought to be transmitted by the consumption of cattle afflicted by the agent of Bovine spongiform

encephalopathy also known as classical BSE, first reported in United Kingdom in 1986. As opposed to the other CJDs, the affected patient is often younger.

Gerstmann-Straussler-Scheinker disease

The main feature of GSS is a progressive degeneration of the cerebellum, as well as different degrees of dementia. The usual signs and symptoms develop by age 35-50 and can include weakness in the legs, poor reflexes, abnormal sensations, progressive ataxia, cognitive dysfunction, slurred speech and spasticity. After diagnosis the patients die after approximately 60 month (2-10 years), on an average[20-21].

Fatal familial insomnia

This is an inherited prion disease affecting mainly the thalamus. It is thought that loss of neurons in the thalamus, as well as other mechanisms not understood, cause the symptoms of FFI. The first symptoms of FFI usually start in middle age and may include progressive insomnia, weight loss, lack of appetite, and rapidly progressive dementia. Almost all cases of FFI are caused by certain mutations in the PRNP gene and are inherited in an autosomal dominant manner.[22] There are a very small number of reported sporadic cases of FFI.[23] There is currently no effective treatment for FFI, but research for a treatment and cure is ongoing.[24] Death usually occurs within 12-18 months of the first symptoms.[25]

Variable Protease-sensitive prionopathy

VPSPr resembles Gerstmann-Sträussler-Scheinker disease (GSS) in terms of the characteristics of the abnormal prion protein (PrPSc). However, unlike in GSS, no mutations in the prion protein gene have been identified.

Clinical manifestations differ from those of Creutzfeldt-Jakob disease, and the PrPSc is less resistant to digestion by proteases; some variants are more sensitive to proteases than others, hence the name: variably protease-sensitive.

Patients present with psychiatric symptoms, speech deficiencies, and cognitive impairment. Ataxia and Parkinsonism can develop. Average age at onset is 70 yrs., and duration of survival is 24 months. About 40% of patients have a family history of dementia.[26]

The Agent

Researchers believed for quite a long time that a "slow virus" or similar organism causes all the above mentioned diseases. However, they were never able to isolate a virus or other organism in people with the disease. The agent had some peculiarities that were unusual for known organisms such as viruses and bacteria. It was difficult to eliminate the agent and surprisingly it did not appear to contain any genetic information in the form of nucleic acids (DNA or RNA), and had an unusually long incubation period before symptoms appeared. In some cases, the incubation period may be as long as 50 years. The molecular nature of the infectious agent lay largely untested for some time until Stanley Prusiner and co-workers achieved the biochemical enrichment of infectious activity and showed its association with a specific protein. At the present time it is believed that the agent causing TSE is caused by a type of protein named asprion; short for proteinaceous infectious particle. Stanley Prusinerwas awarded the Nobel Prize in Physiology/Medicine in 1997 for prion research[27-28].

The prion proteins exist in a normal form as a harmless protein in the body's cells, and also as an infectious form, which causes disease. Both the harmless and infectious forms of the prion protein have the same sequence of amino acids but the infectious form of the protein takes on a different β pleated shape than the normal protein. The transmissible agent, or prion, seems to consist principally of an abnormal isoform of the prion protein (PrP); designated PrPSc. PrPSc is known to be derived from the cellular isoform, PrPC, by a post-translational mechanism. While PrPC is fully sensitive to proteolysis, PrPSc, which accumulates in the brain during disease, is partially protease resistant. Sporadic CJD may develop because some of a person's normal prions spontaneously change into the infectious form of the protein and then alter the prions in other cells in a chain reaction[29].

Once they appear, abnormal prion proteins can aggregate, or clump together as amyloid plaques.

Investigators think these protein aggregates may lead to the neuron loss and other brain damage seen in CJD. However, the exact mechanism is still debatable.

Medical precautions

The principal target of prion pathology is the brain, yet most TSEs also display prion replication at extra-cerebral locations, including secondary lymphoid organs and sites of chronic inflammation. It has been found that the brain (including dura mater), spinal cord, posterior eye and pituitary tissue have the highest rate of infectivity. Other tissues have a low to no risk of infectivity. The proper handling of such tissues by the health care personnel, cannot be overemphasized .

The following clinical practices are recommended when handling suspected cases of prion disease[30-32]:

1. Use disposable instruments whenever possible;
2. Destruction of all used instruments, protective clothing, tissues, and body fluids by incineration;
3. Surface protection with disposable, waterproof drapes;
4. Limitation of people and items in the operating room;
5. Use of barrier protective apparel (double gloves, etc.);
6. Use of manual saws to reduce aerosol formation;
7. Washing surfaces with 1 N NaOH and leaving as a wet film for 1 hour at room temperature.

For instruments and equipment which cannot be destroyed, the following are recommended:

1. Wipe thoroughly before contaminated surface dries;
2. Gravity displacement autoclaving at 132°C for 1 hour, 1 M sodium hydroxide for 1 hour;
3. Porous load autoclaving at 134–138°C for 18 minutes, sodium hypochlorite (20,000 mg/L chlorine for 1 hour)

Dental precautions

As dental instruments have the potential to come into contact with a range of oral tissues that may carry prion infectivity, particularly the peripheral nerves and lymphoid tissue, it would be prudent on a precautionary basis to use the most effective instrument cleaning and sterilisation approaches to control this risk, especially as relatively high levels of infectivity may be present in tissues early in the disease incubation period, before clinical symptoms are observed[33].

But many case-control studies have still not found any evidence that dental procedures increase the risk of iatrogenic transmission of TSEs among humans. In these studies, CJD transmission was not associated with dental procedures (e.g. Root canals or extractions), and there was no convincing evidence of any prion detection in human blood, saliva, or oral tissues, nor have there been reports of any dental health personnel becoming occupationally infected with CJD. In 2000, prions were not found in the dental pulps of eight patients who were confirmed CJD patients, by using electrophoresis and a Western blot technique[34].

Prions exhibit unusual resistance to conventional chemical and physical decontamination procedures. Though scientific data is lacking regarding the risk of transmission of prion diseases, special precautions in addition to standard precautions in case of dental treatment might be indicated when treating known prion disease patients; the following list of precautions is recommended by CDC[35].

1. Use single-use disposable items and equipment whenever possible.
2. Items difficult to clean (e.g., endodontic files, broaches, and carbide and diamond burs) may be considered as single-use disposables and discard after one use.
3. Minimize drying of tissues and body fluids on a device; keep the instrument moist until cleaned and decontaminated.
4. Clean instruments thoroughly and steam-

autoclave at 134ºC for 18 minutes[36].

5. Do not use flash sterilization for processing instruments or devices.

Though the transmission of prion disease to the dentist is slim, nevertheless standard prevention protocols apply.

Conclusion

TSE as a group of diseases still remain a riddle among the investigators as to the nature and pathophysiology of the infections. Newer technologies and ideas have contributed to demystifying the disease to some extent. However prevention of the diseases is critical to stop the spread of the disease in the general population as the diseases have no known cure at present. Proper sterilisation techniques should be followed in hospitals and health care settings. Looking at the history thus far; the possibility of discovering newer prion diseases is a distinct possibility. The next prion disease must be still out there; waiting to be discovered and health care personnel should be aware and take necessary optimum steps to minimise its damage among the general population.

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UNICYSTIC AMELOBLASTOMA IN A 7 YEAR OLD MALE : A RARE ENTITY

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ABSTRACT

Ameloblastoma is the most common aggressive benign odontogenic tumors of the jaws the tumor is often asymptomatic , presenting as a slow growing facial swelling or an incidental finding on a radiograph. Ameloblastoma is a locally destructive tumor with a propensity for recurrence if not entirely excised. A few cases of malignant changes with distant metastasis have been reported in the literature. Ameloblastoma is more commonly seen in 3rd and 4th decades of life and is considered as a rarity in the younger age groups.1 The treatment of ameloblastoma is still controversial as it explains some special problems in the growth and development of jaws in children. Incidence behavior and prognosis of tumor in children make surgical consideration different from adults.

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Introduction

Many benign lesions cause mandibular swellings, and these can be divided into those of odontogenic and non odontogenic origin lesions include ameloblastoma, radicular cyst, dentigerous cyst, keratocystic odontogenic tumor, central giant cell granuloma, fibro-osseous lesions and osteomas.[2] The most common tumor of odontogenic origin is ameloblastoma which develops from epithelial cellular elements and dental tissues in various phases of development. Relative frequency of Unicystic Ameloblastoma (UA) has been reported between 5% and 22% of all types of ameloblastomas.[1] The concept of Unicystic Ameloblastoma (UA) was first described by Robinson and Martinez[1]. More than 90% of Unicystic ameloblastoma occurs in mandible usually in posterior region. The present case has a significant importance because of its early age of occurrence and absence of impacted teeth.

Case Report

A 7 year old male patient presented with a swelling on the left side of the mouth for 1 month. There was no associated pain, difficulty in mouth opening and chewing or articulating. There is no relevant dental or medical history. On physical examination there was a diffuse swelling seen intraorally measuring 2x1 cm from the mesial aspect of 74 to distal aspect of 75, there was no signs of erythema , ulcerations or sinus tract. On palpation of the lesion swelling was found to be firm to bony hard and non tender. Temperature over the swelling was no raised. No neck nodes were palpable OPG of the lesion showed circumscribed unilocular radiolucency in relation to 74, 75 region. Based on the history, clinical and radiographic examination a provisional diagnosis of odontogenic cyst was made. After parental consent an excisional biopsy was performed. Histopathology revealed an odontogenic cyst lining epithelium exhibiting a basal layer with hyperchromatic nuclei arranged in palisading manner, in many foci basal layer exhibits cytoplasmic vacoulisation. Supra basilar layer exhibits stellate reticulum like cells. The histopathologic features correspond to Unicystic Ameloblastoma - Luminal type.

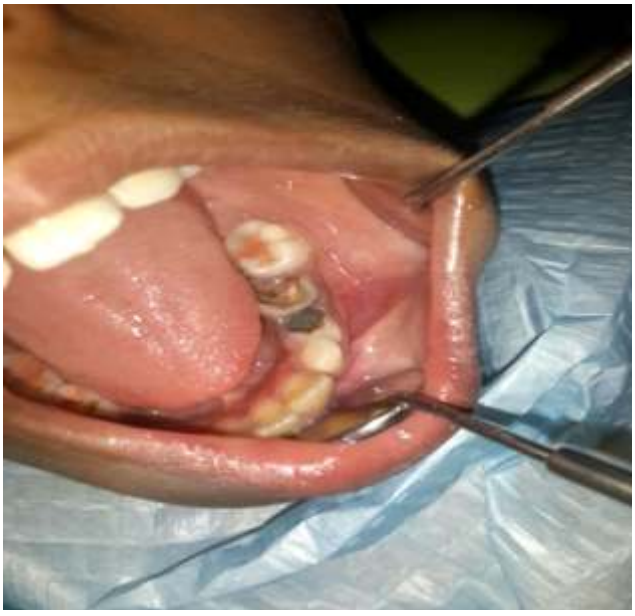
Discussion

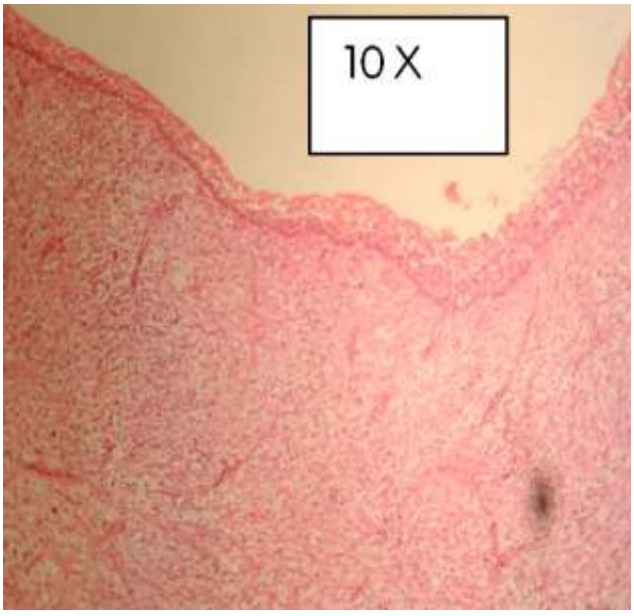
Ameloblastomas are benign tumors whose importance lies in their potential to grow to enormous size with resulting bone deformity and a higher rate of recurrence following incomplete excision. Ameloblastoma is rare before the age of 10 years.[2,3,4] The radiographic stages of ameloblastoma are not a characteristic- a local area of bone destruction of cyst like often unilocular appearance.[4] This is not surprising as it is generally recognized that ameloblastoma amay arise in the wall of a non neoplastic cyst as a result of neoplastic change[1]. Due to the strong likelihood of recurrence curettage or mass excision without a safety margin is not recommended for the treatment of ameloblastoma.[5] When a diagnoss of ameloblastoma is obtained the treatment must be aggressive and radical.[6]

UA shares clinical and radiographical features with other odontogenic lesions and hence the diagnosis can not be made on clinical and radiographic features alone[7,8]. Thus a histopathological evaluation is mandatory for the confirmation of diagnosis. According to Robert and Diane, UA may arise from reduced enamel epithelium or may occur as transformation of dentigerous cyst into UA or due to cystic degeneration of solid ameloblastoma. Ackerman classified entity into three histological groups namely, Luminal, intra luminal and mural, Ackermans classification was modified by Philipsen and Reichart, reclassified into 4 subtypes namely subtype 1 luminal UA , subtype 1.2 luminal and intraluminal UA, 1.2.3 Luminal, intraluminal and intramural UA, 1.3 luminal and intra mural UA. The recurrence depends on histological variant and treatment type. Mural UA has highest recurrence rate among all UAs . [1]

Conclusion

Though considered as benign ameloblastoma is locally invasive odontogenic tumor with a high rate of recurrence.[9] UA is characterized by specific clinical imaging and histological features. For proper understanding of such cases more in depth analysis and long term follow up is mandatory and also utmost importance to correlate histopathologic findings with clinical and radiographic features to

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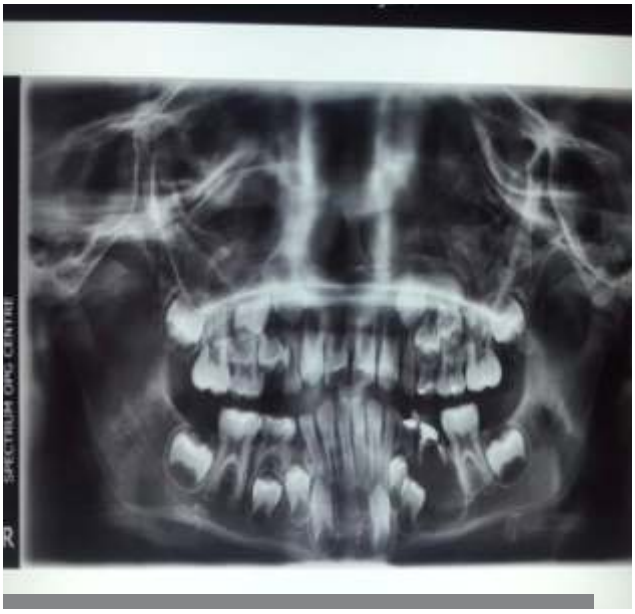

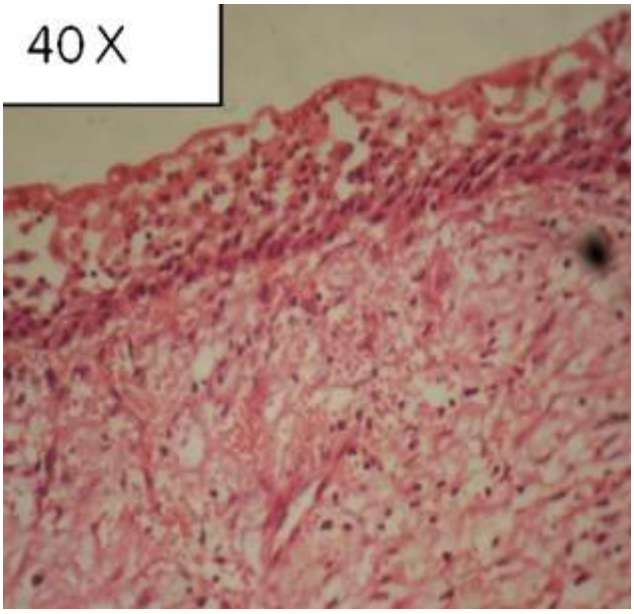
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achieve at a correct definitive diagnosis as all such lesions may have prognostically different biologic behaviors and the final diagnosis may alter the therapeutic decision significantly.[10]

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