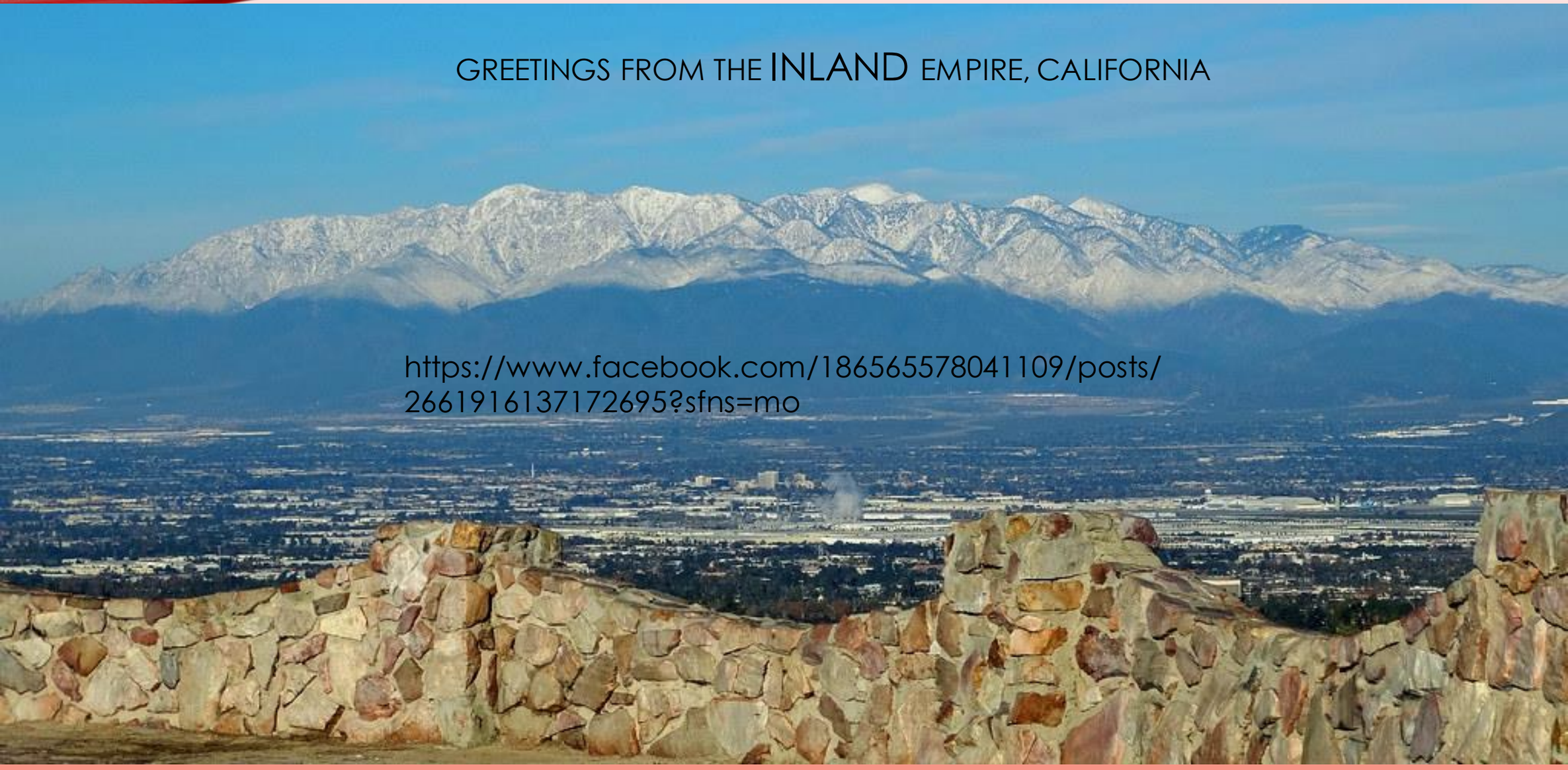
ORAL HEALTH AND SICKLE CELL DISEASE

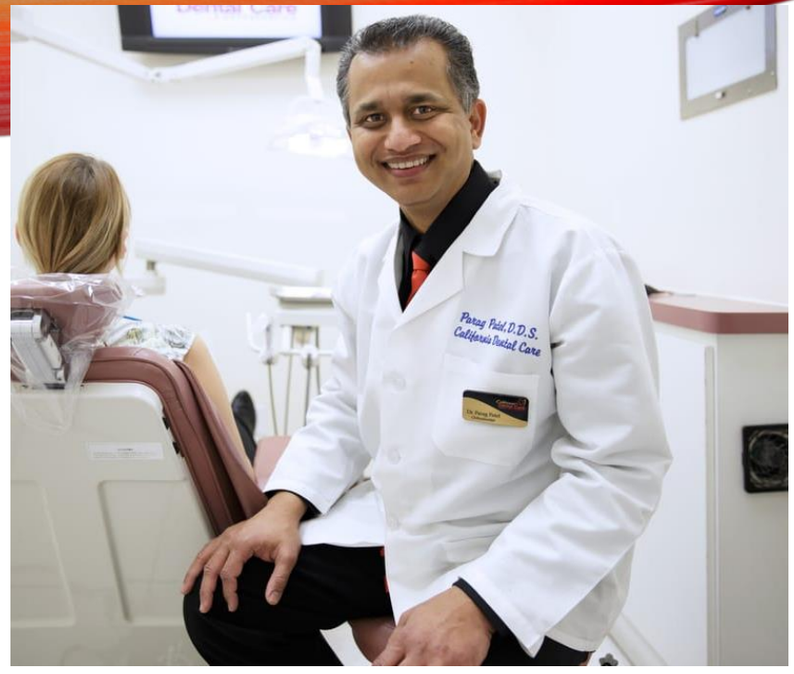
DENTAL CONSIDERATIONS IN TREATMENT OF THE SICKLE CELL WARRIOR.

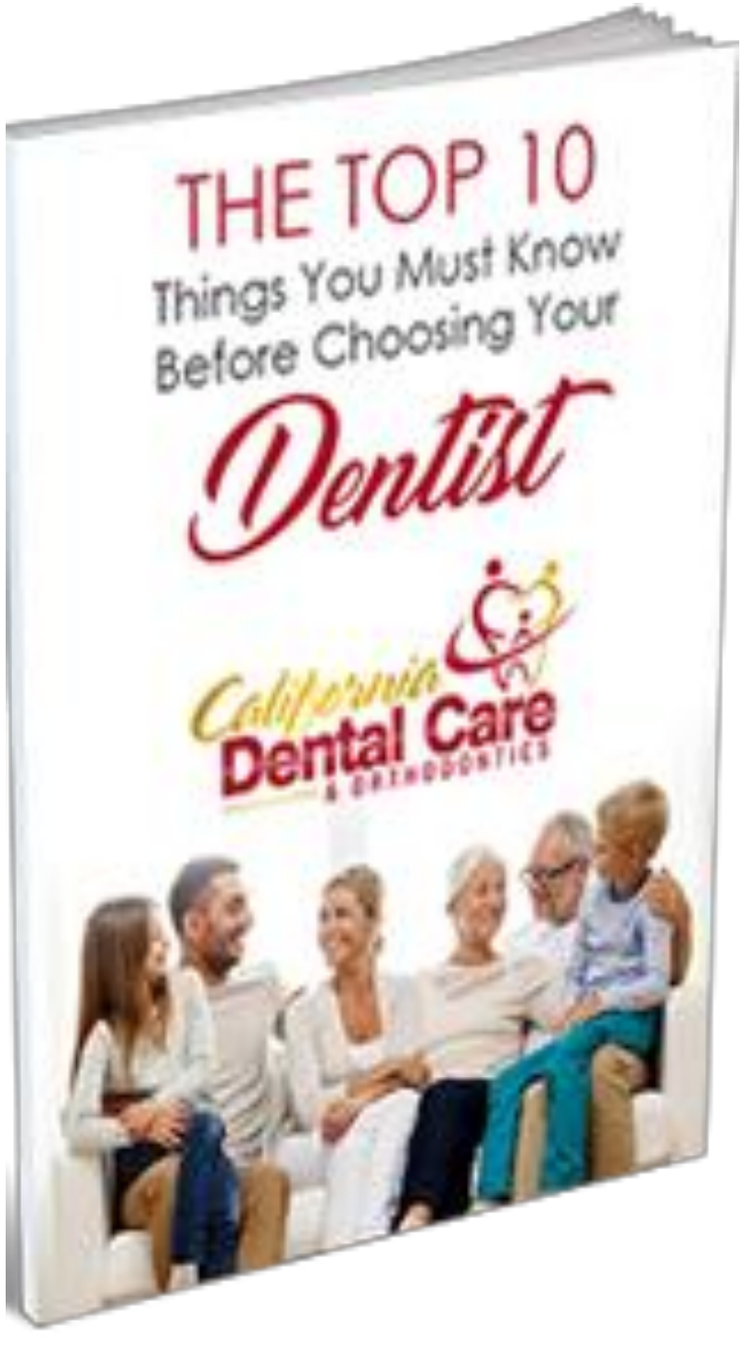
Disclosure Statement

I do not have a relevant financial relationships with any commercial interests in relation to this program/presentation.

GREETINGS FROM THE INLAND EMPIRE, CALIFORNIA






THE TOP 10
Things You Must Know
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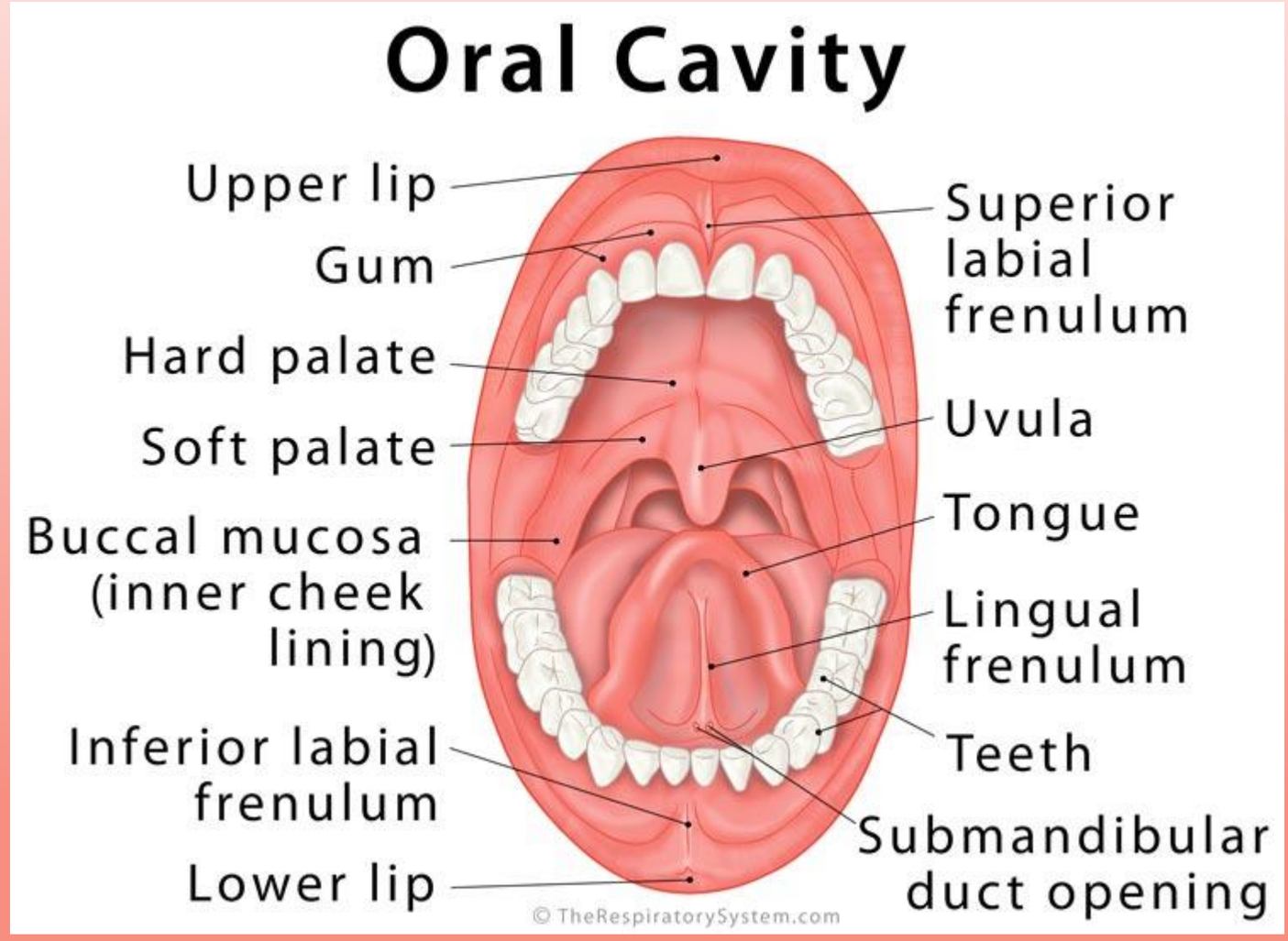
Oral Cavity
Upper lip
Gum
Hard palate
Soft palate
Buccal mucosa (inner cheek lining)
Inferior labial frenulum
Lower lip
Superior labial frenulum
Uvula
Tongue
Lingual frenulum
Teeth
Submandibular duct opening
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Reviewed by the Faculty of Columbia University College of Dental Medicine

What's in Your Mouth?

To understand what happens when your teeth decay, it's helpful to know what's in your mouth naturally. Here are a few of the elements:

•**Saliva** — Your mouth and teeth are constantly bathed in saliva. We never give much thought to our spit, but this fluid is remarkable for what it does to help protect our oral health. Saliva keeps teeth and other parts of your mouth moist and washes away bits of food. Saliva contains minerals that strengthen teeth. It includes buffering agents. They reduce the levels of acid that can decay teeth. Saliva also protects against some viruses and bacteria.

•**Plaque** — Plaque is a soft, gooey substance that sticks to the teeth a bit like jam sticks to a spoon. Like the slime that clings to the bottom of a swimming pool, plaque is a type of biofilm. It contains large numbers of closely packed bacteria, components taken from saliva, and bits of food. Also in the mix are bacterial byproducts and white blood cells. Plaque grows when bacteria attach to the tooth and begin to multiply. Plaque starts forming right after a tooth is cleaned. Within an hour, there's enough to measure. As time goes on, the plaque thickens. Within two to six hours, the plaque teems with bacteria that can cause cavities and periodontal (gum) disease.

Objectives

To determine the acquisition of *Streptococcus mutans* and dental caries experience in children with sickle cell anaemia (SCA) under various prophylactic therapies.

Methods

This study involved a total of 200 children who were divided into five groups comprising of 40 children each. Group SP included children who received prophylactic penicillin, Group SV who received pneumococcal vaccination, Group SPV who received both prophylactic penicillin and pneumococcal vaccination, Group SW who did not receive any prophylactic therapy and Group CC who were controls. Stimulated saliva samples from volunteers were collected and cultured in Mitis Salivarius Bacitracin (MSB) agar for estimation of *Streptococcus mutans*. Decayed, Missing, Filled (DMFT) index/decayed, exfoliated, and filled tooth (deft) index was used for assessment of dental caries.

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Article

Antibiotic Prophylaxis for Children With Sickle Cell Anemia

Sarah L. Reeves, Alison C. Tribble, Brian Madden, Gary L. Freed, Kevin J. Dombkowski

Abstract

BACKGROUND: Children with sickle cell anemia (SCA) are at increased risk for invasive pneumococcal disease; antibiotic prophylaxis significantly reduces this risk. We calculated the proportion of children with SCA who received ≥300 days of antibiotic prophylaxis and identified predictors of such receipt.

METHODS: Children aged 3 months to 5 years with SCA were identified by the presence of 3 or more Medicaid claims with a diagnosis of SCA within a calendar year (2005–2012) in Florida, Illinois, Louisiana, Michigan, South Carolina, and Texas. Receipt of antibiotics was identified through claims for filled prescriptions. The outcome, receipt of ≥300 days of antibiotics, was assessed annually by using varying classifications of antibiotics. By using logistic regression with generalized estimating equations, we estimated the odds of receiving ≥300 days of antibiotics, with potential predictors of age, sex, year, state, and health services use.

RESULTS: A total of 2821 children contributed 5014 person-years. Overall, only 18% of children received ≥300 days of antibiotics. Each additional sickle cell disease-related outpatient visit (odds ratio = 1.01, 95% confidence interval: 1.01–1.02) and well-child visit (odds ratio = 1.08, 95% confidence interval: 1.02–1.13) was associated with incrementally increased odds of receiving ≥300 days of antibiotics.

CONCLUSIONS: Despite national recommendations and proven lifesaving benefit, antibiotic prophylaxis rates are low among children with SCA. Numerous health care encounters may offer an opportunity for intervention; in addition, such interventions likely need to include social factors that may affect the ability for a child to receive and adhere to antibiotic prophylaxis.

We identified children with SCA using a case definition of the presence of at least 3 claims for a child within a calendar year that were SCA-related (282.61, 282.62). This case definition was previously demonstrated to have a high level of sensitivity (91.4%) and specificity (80%) as compared with the gold standard of newborn screening records.[17,18] Continuous enrollment in the Medicaid program for at least 1 calendar year within this time period was required. For each year a child was eligible for the study population, we restricted our analysis to children with no other forms of health insurance (ie, private insurance) during the study period to maximize the completeness of claims available. Children were eligible to contribute multiple nonsequential years to the study population (eg, 2005 and 2007). Children were <5 years old throughout each contributed person-year, consistent with NHLBI recommendations for penicillin prophylaxis.[11]

Definitions of Antibiotic Prophylaxis

Oral penicillin is recommended by the NHLBI for prophylaxis against IPD. However, the American Academy of Pediatrics recommends erythromycin for children with a suspected or proven penicillin allergy, and amoxicillin is sometimes prescribed for practical reasons and is equally effective against *S pneumoniae*. Therefore, we classified antibiotics by using the following 4 definitions:

1. oral penicillin;
2. oral penicillin or erythromycin;
3. oral penicillin, erythromycin, or amoxicillin; and
4. any antibiotic likely to protect against *S pneumoniae* (including penicillin, erythromycin, amoxicillin).[19–23]

Conclusions

Despite long-standing national recommendations, antibiotic prophylaxis against IPD remains low among children with SCA, and efforts aimed at increasing adherence are urgently needed. It is unknown which mechanisms will be the most effective; however, numerous health care encounters may offer an opportunity for intervention. In addition, such interventions likely need to include social factors that may affect the ability for a child to receive and adhere to antibiotic prophylaxis.

Footnotes

- Accepted December 12, 2017.

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Tex Dent J. 2013 Nov;130(11):1123-7.
What dentists should know about sickle cell disease.
Devine BP1.
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Treatment of the sickle cell patient should be a team approach between dentist, patient, and physician. Dental treatments should be conservative and stress free for the patient. Prevention of dental disease and infections are of the up most importance to the sickle cell patient. If your patient has sickle cell disease, know about it and talk to your patient about the disease. Maintaining excellent oral health to decrease the possibility of oral infections will ensure the best care for these patients. Key words: communication, sickle cell disease (SCD), sickle cell anemia (SCA), blood inherited disorder, sickle cell trait, crisis, African Americans, deoxygenation, hemoglobin, supporting dentist, prophylactic antibiotics, and infection.

The medical history should be a communication between the patient and the dentist. A good history will reveal a patient's medical problems, concerns, ideas, and expectations. Understanding medical conditions on a patient's medical history is of up most importance in providing the patient with the best possible standard of care.

ABSTRACT
This study sought to determine whether there was an association between sickle cell disease (SCD) and dental caries in African-American adults. A sample of 102 African-American adult patients with SCD from Washington, D.C., and Baltimore, Maryland, were matched to 103 African-American adult subjects, who did not have SCD. The match was by age, gender and recruitment location. Each subject underwent a standardized oral examination as well as an interview to ascertain risk factors for dental caries. For individuals with incomes of less than $15,000, subjects with SCD had more decayed (10.36 versus 1.58) and fewer filled (2.86 versus 8.45) surfaces compared to subjects without SCD with both differences being statistically significant ($p<0.05$) after adjusting for age and gender. The results suggest that low-income African Americans with SCD may be at increased risk for dental caries and are less likely to receive treatment with a restoration.

Dental health in sickle cell disease

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Abstract

Sickle cell disease (SCD) is one of the most common blood disorders typically inherited from one's parents. It is presented with a wide variety of clinical symptoms, and varied degrees of severity that can be determined based on the phase during which the disease is diagnosed, the age of the patient, number of hospitalizations in the past, requirement for continuous drug use and for blood transfusions, in addition to several other factors. It is highly critical that the physicians should be aware of the oral manifestations and physiopathology of the disease. Additionally, the dental surgeons should cautiously obtain the patient's clinical history and collect information about specific features so that they can build up a plan for any dental treatment that is in accordance to the patient's limitations and requirements. Maintaining a complete chart recording the general patient information along with periodically updating the medical history of the patient should be practice by all the physicians. The treatment strategy should focus on the achievement and maintenance of oral health and to decrease the risks of dental complications. The literature summarizes the treatment of dental complications in patients with SCD.

Key words

sickle cell disease, dental care, dental complications, caries, infection, malocclusion

Introduction

Sickle cell disease (SCD) is one of the most prevalent genetic disorders worldwide. Studies have reported a count of approximately 100,000 Americans being affected by this disease. It is also estimated that one in 500 US African American births is affected with sickle cell anaemia. The prevalence of the disease is highest in sub-Saharan Africa and is also widely spread through Middle East, Southeast Asia, and Mediterranean regions [1]. Around 5-7% of the population worldwide carries an abnormal haemoglobin gene with the frequency of SCD noted as one in four in every 50 West Africans and making itself the commonest genetic disease in UK and France [2,3]. SCD is the most prevailing form of hemoglobinopathy. The disease is characterized by morphologic changes in red blood cells (RBCs), triggered by abnormal haemoglobin (Hb) polymerization.

The sickle mutation is the consequence of single base change, GAG to GTG, in the sixth codon of exon 1 of the β-globin gene in charge for the synthesis of the β-globin polypeptide of the Hb molecule (α2β2). This change cause substitute of the normal glutamic acid with valine at position 6 of the β-globin chain and the development of sickle Hb (βsβs) [4-6]. Acute infections can activate sickle cell crises. Therefore, it is imperative that dental infections should be prevented but, if there is an occurrence of infection, then effective ways of dealing with it should be devised immediately. A clear understanding of the dental implications of SCA must be gained in order to successfully treat SCA patients. The treatment should always begin only after a thorough investigation on the patient's background has been performed.

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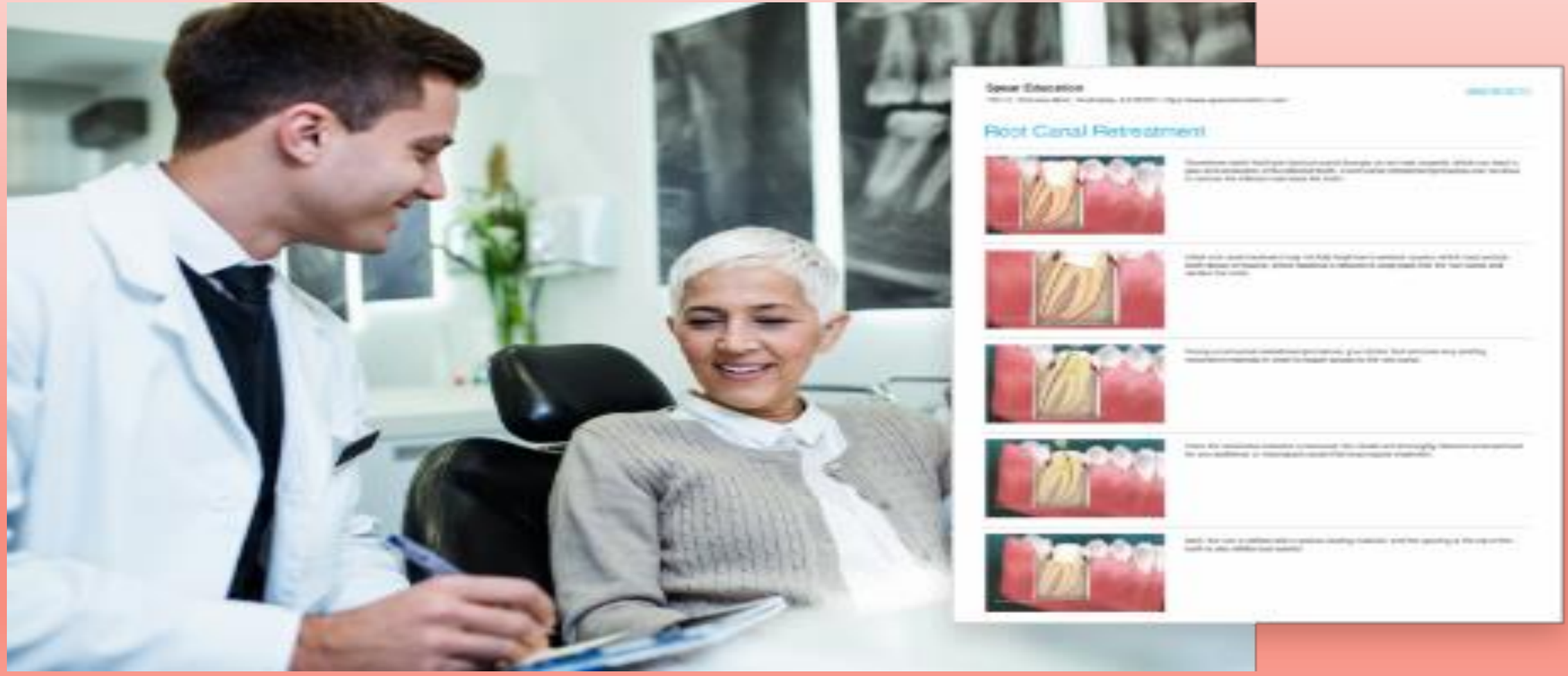
Automatically create handouts, including post-op instructions, that reflect your practice's branding and standards — giving patients info to carry home to learn how to protect their investment.



Clinical presentation of SCD

The signs and symptoms of SCD vary from one patient to the other and may change over time. A key aspect of the disease is vaso-occlusive crises of the microcirculation, which leads to inadequate blood supply to tissues and consequently results in tissue necrosis [7]. Pain is the main and the most chronic feature of this disease and dominates its clinical representation throughout the life of the patient. Patients frequently report pain in form of acute pain crises. It has a substantial impact on the quality of life of the patient and their families. Therefore, several medical and psychological treatment techniques are widely devised and used for pain management in patients affected with SCD [8,9].

In SCD, all body organs are affected or remain at the constant risk of being affected. As such, the most prevalent complications include diseases of nervous, cardiopulmonary, musculoskeletal, hepatobiliary, endocrine and genitourinary systems [10]. The most frequent oral manifestations of SCD greatly affect the oral mucosa, gingival tissue, mandible, osteonecrosis, facial swelling, increased risks for caries nerve supply, and tooth enamel and pulp [11-19] (Table 1). In addition, paleness of the oral mucosa, delayed tooth eruption, depapillation leading to atrophic alteration of the tongue, high degree of abnormality in the hypophosphatemic teeth, odontogenic infection, orofacial pain, craniofacial disorders such as protrusion of the midface area, maxillary expansion, mandibular retrusion, and maxillary protrusion.

Dentists play a significant role in avoiding these complications and improving the quality of life in SCD patients as the SCD patients are more vulnerable to infections and periodontal disease [20,21]. Furthermore, these patients are at a higher risk of developing dental caries leading to elevated occurrence of dental opacities arising due to the unremitting use of medication containing sucrose owing to the high incidence of complications and hospitalization required by the lack of proper oral cleanliness [22]. Managing dental complications is frequently ignored as SCD patients are more dedicated towards maintaining a standard general health because of the serious blood disorder. Ignoring minor dental health matters under these conditions not only worsen the problem but can also cause a painful sickle-cell crisis, leading to emergency hospital admission. Therefore, the management of oral complications in SCD patients require to be modified in accordance to their blood disorder, in order not to cause any additional deterioration to their overall health.

Dental Complications of SCD
Dental Caries
Infractions
Hypodontia
Dental Erosions
Malocclusions
Pulp Necrosis
Abnormal Trabecular Spacing
Infection

Dental complications in SCD

The dental complications in sickle cell anaemia must be understood at first to efficiently treat SCD patients. The management and treatment of the patients should begin with a thorough assessment of the patient's background. Studies have revealed that common oral findings associated with the disorder include mucosal pallor, dental hypoplasia, delayed eruption, and radiographic changes [23]. Furthermore, hypercementosis, which leads to excessive generation of normal calcified tissue on the roots of one or more teeth, has also been reported in SCD patients [24]. Malocclusion of the teeth, intrinsic opacity of enamel, dental caries, and diastemata are other dental remarks found in the disease in sicklers [25]. D

Dental caries

Dental caries are the most frequent dental complication worldwide. Patients with SCD are more susceptible to dental caries having more chances of tooth decay [26]. Caries is identified as an infectious disease of teeth causing progressive demineralization and destruction of the enamel, dentin, and cementum of the teeth [9]. The key source of caries is the acidification of the oral environment, which is caused by the fermentation of remaining food particles mainly sugars or carbohydrates on tooth surfaces [27]. Untreated caries can cause slow destruction and tooth fractures. It may also further lead to infection of the surrounding oral soft tissues and may also transfer to ear, neck and jaw [28] Sometimes, it may be as fatal as causing cavernous sinus thrombosis (CST) leading to the blood clot in the cavernous sinus, the reason being complication of an infection in teeth [29].

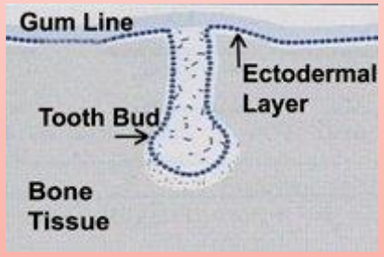
SCD patients appear to be more vulnerable to dental caries than healthy people. Also, the occurrence of caries in SCD children and adults are known to associated with the socioeconomic status of the affected families [22-30]. Low income most frequently affects the amount of value given on lifestyle, health and access to medical care and health information. Past results have shown that low-income individuals are more susceptible to decayed teeth problems as compared with individuals without the disease On the other hand, Passos et al. [31] reported that the disease itself does not particularly predispose to caries or periodontal disease. Risk the contributing factors including daily smoking, older age, and lack of daily dental flossing can attributed towards dental caries and periodontal diseases.

Hypodontia

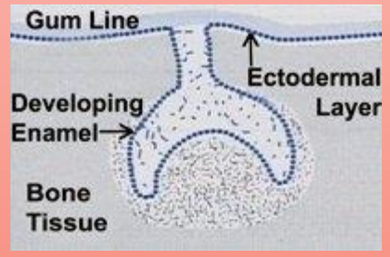
Hypodontia, also known as tooth agenesis, refers to missing teeth as a result of failure for them to develop. It has been described in one patient with HbSC disease [32]. These patients must be subjected to preventive care to lessen the chances of any functional complications.

Basic Tooth Structure and Tooth Root System

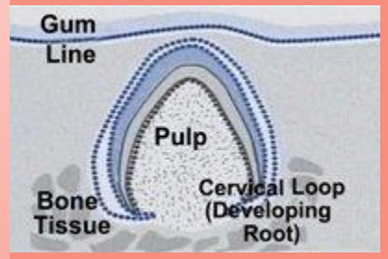
First, to help us understand the basic tooth structure, let's see how a tooth develops . . .



When a tooth is first formed, it is nothing more than "germ cells," cells from a specialized layer of developmental tissue that forms within the bone.

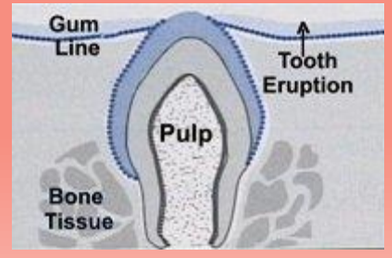


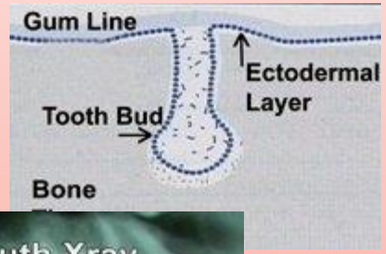
The first part of the tooth that forms in the bone is the outer shell of the enamel.

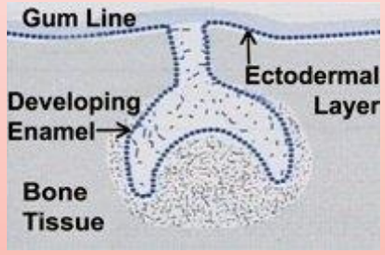


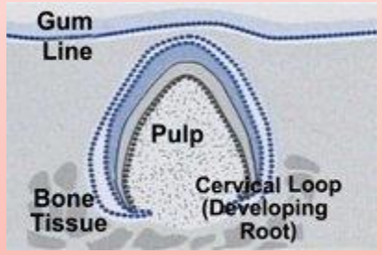
As the tooth fully develops within the bone, before it comes through the surface, it starts growing the tooth root. It does this because the ***blood*** flow that goes into the middle of the tooth starts laying down calcium, phosphorous, magnesium and other minerals that help the tooth to grow.

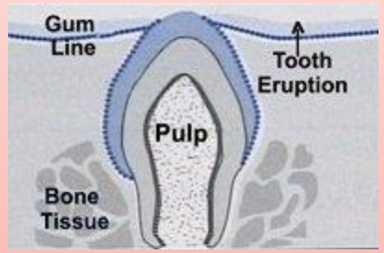
Finally, the developing tooth starts erupting into the mouth.

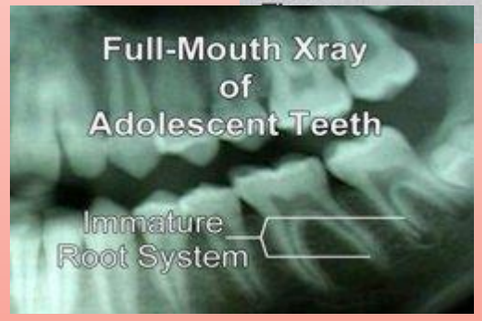












http://www.healthyrootcanal.org/basictoothstructure.html

completely developed until about the age of 15 to 20 years. At that point, the root starts changing, and begins to fully calcify itself.

Dental Patient Education Videos for On-Demand

Anatomy of a Tooth

https://viewmedica.com/ondemand-patient-education-videos/see-videos/dental/?_BwE#vm_A_44a01814gclid=EAIaIQobChMIg67n3av84wIVSR6tBh36VwJ0EAAYAi**Dental Patient Education Videos for On-Demand**AAEgLzHvD

Dental erosion

Dental Erosions, also known as acid erosions, result from acidity that is not caused by bacteria but by food particularly acidic fruit juices. Intrinsic causes of acidity comprise certain disorders such as gastroesophageal reflux where gastric acid is in contact with teeth. Erosions have not been described in SCD patients [33,34].

Malocclusion

Malocclusion, in dentistry, refers to the manner opposing teeth meet. The father of modern orthodontics, Edward Angle, introduced the term malocclusion which is defined as a misalignment of the teeth between the two corresponding dental arches (Figure 1A) [35]. He divided malocclusion into three classes in accordance to the severity and the relation between the teeth involved. In Class I, the occlusion in a SS patient is normal for the first maxillary molar but abnormal for the other teeth with crowding, rotation, and spacing and over or under eruption (Figure 1B). In Class II, inconsistent jaw growth is observed in a patient with HbSS whereas the lower jaw is deficient in forward growth causing an overjet>3 mm (Figure 1C). This increases the chances of dental trauma as compared to those with an overjet<3 mm In Class III, malocclusion in a patient with HbSS, it has been observed that the lower jaw extends too far forward ahead of the upper jaw causing an under-bite, which is also known as mandibular prognathism (Figure 1D) [36]. Reports have shown that Class II malocclusion was most frequent in SCD patients [9]. Furthermore, facial growth alterations which are frequent in SCD might also result in malocclusion because of protrusive maxilla and forward growth tendency of the mandible [37,38].

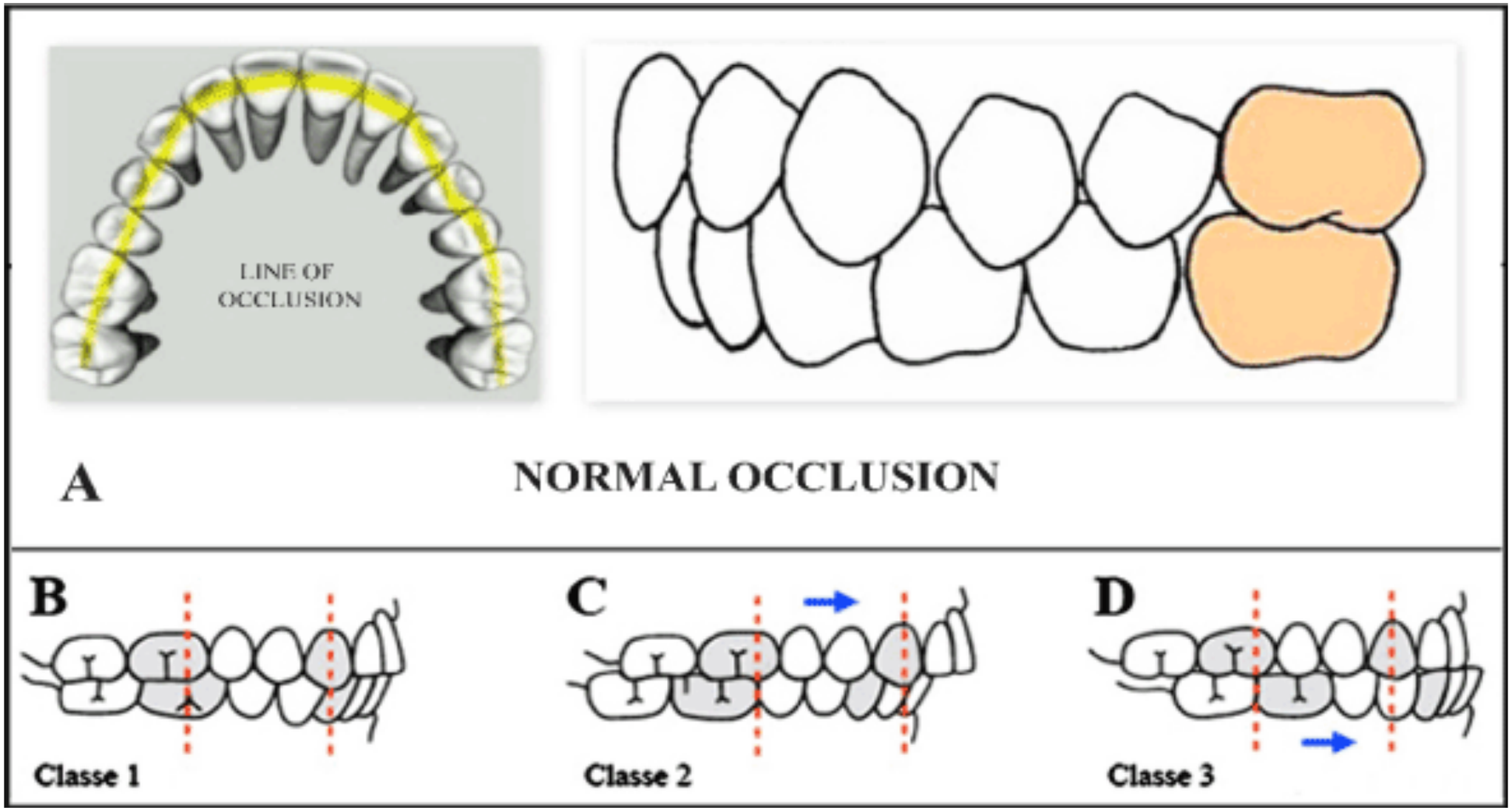

LINE OF
OCCLUSION
A
NORMAL OCCLUSION
B
Classe 1
C
Classe 2
D
Classe 3

Figure 1. Classification of dental malocclusion. (A) Normal occlusion: The cup of the maxillary first molar is aligned with the buccal groove of the mandibular first molar. There is alignment of the teeth, normal overbite and overjet and maxillary and mandibular midlines. (B) Class I malocclusion: A normal molar relationship exists but there is crowding, misalignment of the teeth, cross bites, etc. (3) Class II malocclusion: Has two divisions to describe the position of the anterior teeth; *division 1:* is when the maxillary anterior teeth are proclined and a large over jet is present; *division 2:* is where the maxillary anterior teeth are retroclined and a deep overbite exists. (D) Class III malocclusion: A malocclusion where the molar relationship shows the buccal groove of the mandibular first molar distally positioned when in occlusion with the mesiobuccal cusp of the maxillary first molar (Adapted from Dental Photographs, 2015) [39].

Pulp necrosis

Pulp necrosis, or dead pulp, is defined as necrotic dental pulp due to infection, trauma or chemical reaction characterized by no response to thermal stimulation. A necrotic dental pulp causes toothache, acute apical periodicities, discoloration of the tooth or dental abscess [43]. Previous reports suggest that HbSS is a potential risk factor for pulp necrosis in intact permanent teeth [44,45]. A reduced blood supply to teeth results in necrosis of the dental pulp in patients with SCD. Such abnormal blood flow to the dental pulp might result in increased toothaches in SCD patents. Javed et al. have also stated that there are also chances that a certain proportion of SCD patients may remain asymptomatic to pulp changes, which may make them unsuspecting of the ongoing dental pulp tissue damage.

Conclusion

This mini review was designed to present brief background information on SCD, and describing the course of oral complications in SCD patients. The dentist's aim should be to treat the SCD patient with a thorough understanding and knowledge of the disorder and the consequences of the disease must be considered carefully before dental treatment is started. One of the dentist's goals should be to instil a positive attitude in the SCD patient and their parents toward maintaining good dental health. In addition, it is always advisable that sickle-cell anaemia carriers should be encouraged to have their oral health under control by practicing preventive procedures as directed by the physicians.

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