Indian Journal of Dental Sciences.
June 2014
Issue:2, Vol.:6

www.ijds.in

Review Article

Indian Journal
of Dental Sciences

E ISSN NO. 2231-2293 P ISSN NO. 0976-4003

Tell Tale Shades Of Discolored Teeth - A Review

[1] Vishal Mehrotra
[2] Asheesh Sawhny
[3] Ira Gupta
[4] Rohit Gupta

[1] Reader, Dept. of Oral Medicine and Radiology,
[2] Professor, Dept of Conservative
[3] Reader, Dept. of Periodontics
[4] Reader, Dept. of Periodontics
Rama Dental College and Hospital, Lakhanpur

Address For Correspondence:
Dr. Vishal Mehrotra
Reader, Dept. of Oral Medicine and Radiology,
Rama Dental College and Hospital, Lakhanpur,
Email id: vishal4march@rediffmail.com
Mobile number: +919956575812

Submission : 17th April 2012
Accepted : 18th March 2014

Quick Response Code

Abstract
In today's society bright white teeth are highly regarded, and the consumers invest lots of money for their esthetic appraisal. Thus it is important for the consumers to avoid or limit exposure to materials that contribute to the discoloration of teeth. This article reviews and classifies the various causes for the intrinsic and extrinsic discoloration affecting both the primary or permanent dentition and also aims in creating awareness in the medical and dental field regarding the various medications, systemic diseases, genetic defects and hereditary causes resulting in the discoloration.

Key Words
Intrinsic, Extrinsic, Discoloration.

Introduction:

By this point in the 21st century, the treatment of tooth discoloration has evolved into an annual multibillion-dollar, highly sophisticated, scientific, and clinical discipline. However, the origins of the treatment date back thousands of years to ancient clinicians and beauticians who used rudimentary, yet innovative, natural materials to mask undesirable tooth discolorations.[1] Bright white teeth are highly regarded and sought after in today's society. Consumers invest a lot of money on cosmetic dentistry and in commercial products that promise an aesthetically pleasing smile. It follows that consumers should want to avoid or limit exposure to materials that contribute to the discoloration of teeth. [2]

Any structural changes involving the crown can affect the transmission and reflection of light by the tooth and result in a color change.[2],[3] Teeth gradually darken as a person ages. Over time, the gradual loss of the enamel, the laying down of secondary dentin and the accumulation of stains contribute to the light-transmitting properties of the teeth. [2],[4]

Tooth discoloration is usually described as extrinsic or intrinsic, based on the location of the stains. [2]

Intrinsic Tooth Discoloration:

Generalized intrinsic or permanent discoloration is indicative of a disruption of normal tooth development, whereas localized intrinsic staining of 1 or 2 teeth can occur before or after teeth have erupted.[5] Intrinsic discolorations occur following a change to the structural composition or thickness of the dental hard tissues. Localized discoloration may be a result of either preeruptive or posteruptive processes, whereas widespread involvement indicates a deviation in normal tooth formation. [5],[6] The various causes for intrinsic discoloration are mentioned as follows:

1) Dental Materials:

Dental restorations are one of the most common cause for intrinsic discoloration. Amalgam restorations can result in release of certain corrosion products (e.g. silver sulfide), there by resulting in a gray-black color in the tooth, especially in large cavity preparations with undermined enamel. Composites, pins, glass ionomer cement and acrylic restorations gradually can leave a gray hue in the tooth adjacent to the material. Other dental materials that cause intrinsic discoloration include formocresol, eugenol, root canal sealers, and polyantimicrobial pastes.[7]

2) Regressive Alteration of the Teeth and Dental Caries:

As the age advances the permanent dentition is subjected to the regressive alterations such as attrition, abrasion, and erosion, which may cause the dentition to progressively become more gray and yellow. This age-related phenomenon is due to a progressive loss in enamel from attrition or teeth wear that reveals the natural yellow color of underlying dentin.[7]

Incipient carious lesions are associated with plaque accumulation and manifest as chalky white areas of discoloration secondary to hypocalcification.[7] As caries progresses into the dentin, the overlying translucent enamel reveals the color of the underlying caries and appears yellowish brown. Extensive caries that involve destruction of both enamel and dentin produce a color that ranges from light brown, to dark brown or almost black .The brown color is attributed to the formation of Maillard pigments (reaction between proteins and small aldehydes produced by cariogenic bacteria), melanins, lipofuscins, and uptake of various food colors and bacterial pigments.[8]

3) Trauma:

Trauma to developing unerupted teeth can disturb the amelogenesis and may result in enamel hypoplasia, which appears as a localized opacity on the erupted tooth. Such teeth commonly are referred to as Turner's teeth.

Trauma that occurs to erupted teeth also causes discoloration. This frequently occurs in teeth that have fully formed roots and have sustained irreversible pulpal injury caused by avulsions,

intrusions, luxations and subluxations, or fractures involving the pulp chamber. Trauma results in intrapulpal hemorrhage and iron sulfide deposition along the dentinal tubules, producing a bluish black cast.

Occlusal trauma occurring over a protracted period of time (e.g. excessive orthodontic forces) may rarely lead to pulpal hemorrhage; however, it can produce a subtle grayish brown cast.[1]

4) Infections:

Since crown formation begins in utero; there is an inherent potential for extensive intrinsic discoloration of the primary dentition present throughout pregnancy. Periapical odontogenic infections of the primary teeth can disrupt normal amelogenesis of the underlying secondary (permanent) successors and involve a potential for localized enamel hypoplasia.[1]

Although rare, maternal rubella, cytomegalovirus infections and toxemia of pregnancy can lead to tooth discoloration, which generally manifests as a focal opaque band of enamel hypoplasia that is confined to the primary teeth forming enamel at the time of maternal infection.

Systemic postnatal infections (e.g., measles, chicken pox, streptococcal infections, scarlet fever) can also lead to enamel hypoplasia. This can be explained by the fact that the crown formation of the secondary dentition occurs until the child is aged approximately 8 years and may manifest as bandlike discolorations on the tooth surface.

5) Medication:

Fluorides:

Dental fluorosis is characterized by enamel discoloration resulting from subsurface hypomineralization due to the excessive ingestion of fluoride during the early maturation stage of enamel formation.[9] Discoloration or damage to tooth structure may occur when the total daily intake of the fluoride ion from sources such as water, toothpaste, prescribed drops, and tablets is high while the enamel is undergoing pre-eruptive formation and maturation.[10] The most common adverse effect of excess exposure to fluoride is dental fluorosis, which is characterized by enamel discoloration resulting from subsurface hypomineralization due to the excessive ingestion of fluoride during the early maturation stage of enamel formation. Fluorosis affects primary and secondary dentitions with a broad range of clinical findings. In its mildest form, fluorosis appears as faint white lines or streaks on the enamel. Moderate fluorosis has more obvious opaque regions referred to as enamel mottling, whereas severe fluorosis appears with extensive mottling that readily chips and stains and leads to pitting and brown discoloration.[11] Dental fluorosis is a dose-dependent condition, and the higher the level of exposure during tooth development, the more severe the fluorosis.[12] Grobler et al., described the fluoride intake during critical periods of tooth development and maturation, from approximately birth to 8 years of age, is in the range of 0.03 to 0.1 mg F/kg body weight per day.[13]

Tetracyclines:

Tetracyclines result in the discoloration of tooth enamel when prescribed during tooth development. Tetracycline diffuses through dentin to the enamel interface, chelating calcium ions and incorporating into hydroxyapatite as a stable orthophosphate complex, when administered during the calcification stage of tooth development.[14] Females exposed to tetracycline during the second or third trimester of pregnancy may give birth to a child who will have discolored teeth. Newly erupted teeth will usually present with a bright yellow band.[1] On exposure to sunlight, the tetracycline crystals in the teeth undergo a photochemical reaction and gradually darken to a grey or red-brown color.[1] Since the majority of mineralization of the permanent dentition is not complete until a child is eight years of age (excluding third molars), tetracyclines should also not be used by anyone under that age. Discoloration occurs with the greatest frequency in the developing dentition when total dosage administration is over 3 g, or treatment exceeds 10 days.[15] Tetracycline and oxytetracycline cause a yellow discoloration, whereas chlortetracycline produces a grey-brown discoloration.[16] It is also reported in the literature that tetracyclines (e.g., Ledermix - triamcinolone acetonide and demethylchlortetracycline) used within the tooth for endodontic therapy may also cause dark grey-brown discoloration.[17]

Minocycline:

Minocycline is a second-generation semi-synthetic tetracycline derivative. The use of minocycline in periodontal disease has seen a wide increase recently with the use of polymer beads as local carriers inserted directly into the periodontal pocket. Minocycline has been reported to cause generalized intrinsic green-grey or blue-grey tooth staining post-eruption.[18],[19] Staining of the adult dentition appears to occur in 3-6% of patients taking long-term minocycline at > 100 mg daily.[20] The onset of discoloration can occur at any time from 1 month to many years after the initiation of treatment. Poor oral hygiene and intense sunlight exposure can further make the staining more prominent.[19] Currently, there are four possible theories: first, the 'extrinsic theory',[20] where it is thought that minocycline attaches to the glycoproteins in acquired pellicles. The second is the 'intrinsic theory',[21] where the minocycline bound to plasma proteins is deposited in collagen-rich tissues, such as teeth. The third possibility is that hemosiderin, a breakdown product of iron, chelates with minocycline to form an insoluble complex.[22] The fourth suggestion is that minocycline could be deposited in dentin during dentinogenesis.[23]

Doxycycline:

Doxycycline has recently been reported to cause extrinsic staining of teeth, possibly by binding to glycoproteins in the dental pellicle in patients with poor oral hygiene in whom oxidation occurs (eg, sunlight exposure, bacterial) or via mechanisms similar to those for minocycline.[24]

Tigecycline:

Tigecycline, a glycylcycline tetracycline derivative, crosses the placenta and may cause permanent yellow, grey, or brown tooth discoloration if taken during the second and third trimesters.[25]

Ciprofloxacin:

A permanent greenish discoloration of new erupted teeth has been reported in infants who received intravenous ciprofloxacin 10 to 40mg/kg/day for Klebsiella infections.[6]

6) Nutritional Deficiencies and other Disorders:

Vitamins C and D, calcium, and phosphate are required for healthy tooth formation, thus there deficiencies can result in dose-related or exposure-related

enamel hypoplasia.

Number of diseases have the potential to cause hyperbilirubinemia which may result in intrinsic tooth discoloration, these include sickle cell anemia; thalassemia; hemolytic disease of the newborn (HDN) due to Rhesus factor, ABO, or other erythrocyte antigen incompatibility; biliary atresia;[26] and other rare pediatric diseases. These diseases have the ability to cause hyperbilirubinemia and the subsequent dose-dependent incorporation of biliverdin into developing teeth, producing jaundice like yellow-green tint on the tooth surfaces.[27]

7) Genetic defects and Hereditary Diseases:

Genetic defects in enamel or dentin formation include amelogenesis imperfecta, dentinogenesis imperfecta, and dentinal dysplasia. These are hereditary diseases with a propensity for intrinsic tooth discoloration.

Amelogenesis Imperfecta affects both primary and secondary dentitions and demonstrates numerous clinical manifestations that are classified into the following 4 types: [28]

Type 1 involves hypoplastic dentition. These teeth have rough or pitted enamel surfaces, thus are at a greater risk for extrinsic staining. The teeth typically have an abnormally thin enamel layer that reveals the yellow color of dentin beneath the enamel.

Type 2 involves hypomaturation. Teeth with hypomaturation have soft enamel with a mottled opaque white, yellow, or brown discoloration.

Type 3 involves hypocalcification. The enamel in the hypocalcified type is yellow to orange, soft, and lost soon after eruption. Therefore, hypocalcified teeth develop dark stains and are at high risk for dental caries.

Type 4 involves hypomaturation or hypoplastic dentition with taurodontism.

Dentogenesis Impefecta occurs in 3 types. One type is associated with osteogenesis imperfecta, and the other type affects the teeth alone. The primary and secondary teeth are affected, and they have a brown or blue appearance with a distinctive translucent quality. The enamel chips off easily, and the teeth are prone to occlusal wear and caries. The third type is the one found in bradywine population.[29],[30]

Dential Dysplasia occurs in 2 types. Teeth with Type 1-dentinal dysplasia have crowns with normal morphology and coloration, whereas teeth with Type 2-dentinal dysplasia have a blue, amber, or brown translucence. [29],[30]

Other hereditary diseases include erythropoietic (congenital) porphyria and epidermolysis bullosa (EB). Erythropoietic porphyria is a rare disease of porphyrin metabolism. The abnormally high levels of reddish brown or burgundy-red porphyrin pigments have an affinity for calcium phosphate and are incorporated into teeth during dental formation. The entire primary and secondary dentitions are most commonly pink or rarely described as reddish brown or purple.[29] Teeth fluoresce red under ultraviolet light. Patients with EB may have enamel hypoplasia and pitting, which produces a yellowish tint. These patients are at a high risk for dental caries.[29]

Table I: Extrinsic Tooth Discoloration

Color Of Tooth Discoloration	Causes
Brown Stain	Tobacco Products, Dental Plaque, Tea, Coffee, Wine, And Other Beverages, Metals, Iodine, Chlorhexidine/Cetylpyridinium Chloride Rinse, Stannous Fluoride, Doxycycline
Black Stain	Tobacco Products, Betel Nut, Dental Plaque, Chromogenic Bacteria, Tea, Coffee, Wine, And Other Beverages, Metals
Green Stain	Chromogenic Bacteria, Tea, Metals
Orange Stain	Chromogenic Bacteria, Metals, Doxycycline

Table II: Intrinsic Causes: - Localized Color Changes (In 1 Or 2 Adjacent Teeth)

Color Of Tooth Discoloration	Causes
White Opaque Stain	Mild Trauma To Permanent Teeth During Enamel Formation E.G. Turner Tooth, Periapical Infection Of Primary Tooth, Traumatic Injury To Primary Tooth Or Teeth, Incipient Caries.
Yellow Stain	Moderate Trauma To Permanent Teeth During Enamel Formation E.G. Turner Tooth ,Periapical Infection Of Primary Tooth, Traumatic Injury To Primary Tooth Or Teeth, Trauma Without Hemorrhage, Composites Or Glass Ionomer Or Acrylic Restoration, Dental Caries (Active), Focal Tooth Abrasion.
Brown Stain	Severe Trauma To Permanent Teeth During Enamel Formation, E.G. Turner Tooth, Periapical Infection Of Primary Tooth, Traumatic Injury To Primary Tooth Or Teeth, Composite, Glass Ionomer, Or Acrylic Restoration, Caries (Active Or Remineralized) Pulpal Trauma With Hemorrhage
Blue, Grey Or Black Stain	Amalgam Restoration, Glass Ionomer Or Acrylic Restoration, Metal Crown Margin Associated With Porcelain Fused To Metal Crown, Pulpal Trauma With Hemorrhage

Table III: Intrinsic Causes - Regional Color Changes

Color Of Tooth Discoloration	Causes
White Opaque Stain	Infection (Maternal Or Childhood) During Enamel Formation, Trauma To Multiple Teeth During Enamel Formation, Mild Fluorosis, Nutritional Deficiency
Yellow Stain	Infection (Maternal Or Childhood) During Enamel Formation, Moderate Fluorosis, Trauma To Multiple Teeth During Enamel Formation, Nutritional Deficiency, Epidermolysis Bullosa, Regional Tooth Abrasion Or Erosion, Diseases Causing Hyperbilirubinemia
Brown Stain	Infection (Maternal Or Childhood) During Enamel Formation, Severe Fluorosis, Trauma To Multiple Teeth During Enamel Formation
Blue, Grey Or Black Stain	Tetracycline Therapy (Short-term Exposure)
Green Stain	Diseases Causing Hyperbilirubinemia E.G. Biliary Atresia

Table IV: Intrinsic Causes - Generalized Changes (Involving Primary And/Or Permanent Dentitions)

Color Of Tooth Discoloration	Causes
White (Opaque) Stain	Mild Fluorosis, Amelogenesis Imperfecta
Yellow Stain	Moderate Fluorosis, Amelogenesis Imperfecta, Dentinogenesis Imperfect, Dentinal Dysplasia, Epidermolysis Bullosa, Diseases Causing Hyperbilirubinemia, Hemolytic Diseases, Generalized Tooth Attrition, Abrasion, Or Erosion
Brown Stain	Porphyria, Tetracycline Therapy (Long-term Exposure)
Blue, Grey Or Black Stain	Tetracycline Therapy (Long-term Exposure), Minocycline Therapy
Green Stain	Diseases Causing Hyperbilirubinemia E.G. Biliary Atresia

Extrinsic Tooth Discoloration:

Extrinsic stains are defined as stains located on the outer surface of the tooth structure and caused by topical or extrinsic agents. The Nathoo classification system of extrinsic dental stain describes 3 categories as follows:[31]

Nathoo type 1 (N1): N1-type colored material (chromogen) binds to the tooth surface. The color of the chromogen is similar to that of dental stains caused by tea, coffee, wine, chromogenic bacteria, and metals.

Nathoo type 2 (N2): N2-type colored material changes color after binding to the tooth. The stains actually are N1-type food stains that darken with time.

Nathoo type 3 (N3): N3-type colorless material or prechromogen binds to the tooth and undergoes a chemical reaction to cause a stain. N3-type stains are caused by carbohydrate-rich foods (eg, apples, potatoes), stannous fluoride, and

chlorhexidine.

There are number of important factors that predispose children and adults to extrinsic stains, these include enamel defects, salivary dysfunction, and poor oral hygiene.[31] Occlusal pits, fissures, and defects in the outer surface of the enamel are susceptible to the accumulation of stain-producing food, beverages, tobacco, and other topical agents. Since saliva plays a major role in the physical removal of food debris and dental plaque from the outer and interproximal tooth surfaces, diminished salivary output contributes to extrinsic stains.[7] The various causes for extrinsic tooth discoloration are mentioned as follows: **(Tables)**

1) Local Factors:

The most common cause of extrinsic stains is poor oral hygiene.[3] The inability to remove stain-producing materials and/or the use of dentifrices with inadequate cleaning and polishing actions cause discolorations.[7]

Accumulations of dental plaque, calculus and food particles cause brown or black stains. Deposition of tannins found in tea, coffee, and other beverages results in brown staining of the tooth surfaces. [7]

2) Tobacco/Betel Nut:

The smokeless and smoke producing forms of tobacco results in tenacious dark brown and black stains that cover the cervical one third to one half of the. [1],[7]

A combination of betel nut of the areca palm, betel leaf, and lime, also known as "pan" when chewed elicits a copious production of blood red saliva that results in a red-black stain on the teeth, gingiva, and oral mucosal surfaces. [1],[7]

3) Chromogenic Bacteria/Fungi:

Chromogenic bacteria cause stains, typically at the gingival margin of the tooth. The most common is a black stain caused by Actinomyces species. The stain is composed of ferric sulfide and is formed by the reaction between hydrogen sulfide produced by bacterial action and iron in the saliva and gingival exudates.[32]

Green stains are attributed to fluorescent bacteria and fungi such as Penicillium and Aspergillus species.[7] These organisms grow only in light and therefore cause staining on the maxillary surface of the anterior teeth. Chromogenic bacteria such as Serratia marcescens and Flavobacterium lutescens cause orange stains. [1]

4) Occupational Exposure:

Various metallic compounds can also cause dental discolorations, as a result of the interaction of the metals with dental plaque there by resulting in surface stains.[7] Industrial exposures to iron, manganese, and silver may stain the teeth black. Mercury and lead dust can cause a blue-green stain; copper and nickel, green–to–blue-green stain; chromic acid fumes, deep orange stain; and iodine solution, brown stain[1],[7]

5) Topical Medications:

Topical medications can also cause dental staining. Chlorhexidine rinse (0.12%) causes brown staining after several weeks of use, particularly on acrylic and porcelain restorations. Cetylpyridinium chloride is an ingredient in several mouthwashes can also cause dental staining.[33] Iron-containing oral solutions used for treatment of iron deficiency anemia cause black stains. Potassium permanganate mouthwash (violet-black stain), silver nitrate (black stain), and stannous fluoride (brown stain) also can induce dental discolorations.[7]

6) Systemic Medications:

Some systemic medications (eg, minocycline, doxycycline) can cause extrinsic staining. Long term use of doxycycline results in its binding to the gylycoproteins of the dental pellicle, and in patients with poor oral hygiene, undergoes oxidation when exposed to sunlight or bacteria, resulting in extrinsic tooth discoloration. [1],[24]

Linezolid, an oxazolidinone, was reported to cause a brown discoloration of the enamel on the lower front teeth of an 11-year old girl who received a dose of 600mg twice daily for 4 weeks.[34]

Antimicrobial agents chiefly cause pseudo-discolorations, possibly by chromogenic precipitates in the pellicle, or by overgrowth with chromogenic microorganisms.[5]

Conclusion:

A discolored dentition is one of the most common finding observed by the medical and dental practitioners in their routine clinical practice. In the present scenario, cosmetic dentistry is in its boom because of the increasing demand of the esthetic appraisal by the patients specially related to the stained dentition. This article summarizes the various etiologic factors related to the tooth discoloration, and also classifies it into intrinsic or extrinsic, resulting in localized or a generalized involvement of the primary/permanent dentition, further emphasizing on the various systemic, infectious, genetic, hereditary and medicinal causes related to the tooth discolorations, and thereby aims in helping the medical and dental practitioners to first identify the cause for the discoloration, before initiating the treatment.

Since tooth discoloration is one of the most common problems for compromising of the esthetics, prevention remains the key to minimize the physical and psychological trauma associated with it.

References:

1. Ross Kerr A, Ship JA. Tooth discoloration. eMedicine from WebMD. Available : www.emedicine.com/derm/ (accessed March 24, 2007).
2. Watts A, Addy M. Tooth discoloration and staining: a review of the literature. Br Dent J 2000; 190:309-16.
3. Wright JT. Anatomy and development of the teeth. UpToDate [electronic database]. Rose BD, editor. Waltham (MA); 2007.
4. Spraycar M, editor. Stedman's Medical Dictionary. 26th ed. Baltimore (MD): Williams and Wilkins; 1995.
5. Tredwin CJ,Scully C,Bagan-Sebastian JV. Drug- induced disorders of the teeth. J Dent Res 2005; 84(7): 596-602.
6. Kadam A, Ganachari M, Mahendra kumar B,Gurunath S. Drug Induced Tooth Discolouration. The Internet Journal of Dental Science. 2009; 7(2).
7. Hattab FN, Qudeimat MA, al-Rimawi HS. Dental discoloration: an overview. J Esthet Dent. 1999; 11(6): 291-310. .
8. Kleter GA. Discoloration of dental carious lesions (a review). Arch Oral Biol. Aug 1998; 43(8): 629-32.
9. DenBesten PK. Biological mechanisms of dental fluorosis relevant to the use of fluoride supplements. Community Dent Oral Epidemiol. Feb 1999; 27(1): 41-7.
10. DenBesten PK (1999). Mechanism and timing of fluoride effects on developing enamel. J Public Health Dent 59:247-251.
11. Pendrys DG. Risk of enamel fluorosis

in nonfluoridated and optimally fluoridated populations: considerations for the dental professional. J Am Dent Assoc. Jun 2000; 131(6): 746-55.
12. Fejerskov O, Stephen KW, Richards A, Speirs R (1987). Combined effect of systemic and topical fluoride treatments on human deciduous teeth—case studies. Caries Res 21:452-459.
13. Grobler SR, van Wyk CW, Kotze D (1986). Relationship between enamel fluoride levels, degree of fluorosis and caries experience in communities with a nearly optimal and a high fluoride level and in the drinking water. Caries Res 20:284-288.
14. van der Bijl P, Pitigoi-Aron G. Tetracyclines and calcified tissues. Ann Dent. Summer-Fall 1995; 54(1-2): 69-72.
15. www.continuingeducation.com / pharmacy / adverseoral / index.html (2004). Adverse Oral & Dental Effects of Medications.
16. Wallman IS, Hilton HB (1962). Teeth pigmentation by tetracycline. Lancet I: 827-829.
17. Kim ST, Abbott PV, McGinley P (2000). The effects of Ledermix paste on discolouration of immature teeth. Int Endod J 33:233-237.
18. Wolfe ID, Reichmeister J (1984). Minocycline hyperpigmentation: skin, tooth, nail, and bone involvement. Cutis 33:457-458.
19. Dodd MA, Dole EJ, Troutman WG, Bennahum DA (1998). Minocycline associated tooth staining. Ann Pharmacother 32:887-889.
20. Berger RS, Mandel EB, Hayes TJ, Grimwood RR (1989). Minocycline staining of the oral cavity. J Am Acad Dermatol 21:1300-1301.
21. Bowles WH, Bokmeyer TJ (1997). Staining of adult teeth by minocycline: binding of minocycline by specific proteins. J Esthet Dent 9:30-34.
22. Poliak SC, DiGiovanna JJ, Gross EG (1985). Minocycline-associated tooth discolouration in young adults J Am Med Assoc 254:2930-2932.
23. Good ML, Hussey DL (2003). Minocycline: stain devil. Br J Dermatol149: 237-239.
24. Ayaslioglu E, Erkek E, Oba AA, Cebecioglu E. Doxycycline-induced staining of permanent adult dentition. Aust Dent J. Dec 2005; 50(4): 273-5.
25. McEvoy GK,editor.AHFS Drug Information. Bethesda (MD): American Society of Health Syatem Pharmacists, Inc.; 2007.
26. Morisaki I, Abe K, Tong LS, Kato K, Sobue S. Dental findings of children with biliary atresia: report of seven cases. ASDC J Dent Child. May-Jun 1990; 57(3): 220-3.
27. Cullen CL. Erythroblastosis fetalis produced by Kell immunization: dental findings. Pediatr Dent. Nov-Dec 1990; 12(6): 393-6.
28. Neville BW, Damm DD, Allen CM, Bouquot JE. Abnormalities of the teeth. In: Neville BW, Damm DD, Allen CM, Bouquot JE, eds. Oral & Maxillofacial Pathology. ed. Philadelphia, Pa: WB Saunders; 1995.
29. Trodahl JN, Schwartz S, Gorlin RJ. The pigmentation of dental tissues in erythropoietic (congenital) porphyria. J Oral Pathol. 1972; 1(4): 159-71.
30. Shafer's Textbook of Oral Pathology.4thedition.Oral aspects of metabolic disease. In: W.B.Saunders, 1993:616-53.
31. Vogel RI. Intrinsic and extrinsic discoloration of the dentition (a literature review). J Oral Med. Oct-Dec 1975; 30(4): 99-104.
32. Nathoo SA. The chemistry and mechanisms of extrinsic and intrinsic discoloration. J Am Dent Assoc. Apr 1997; 128 Suppl: 6S-10S.
33. Eriksen HM, Jemtland B, Finckenhagen HJ, Gjermo P. Evaluation of extrinsic tooth discoloration. Acta Odontol Scand. 1979; 37(6): 371-5.
34. Matson KL, Miller SE (2003). Tooth discoloration after treatment with linezolid. Pharmacotherapy 23:682-685.

Source of Support : Nill, Conflict of Interest : None declared