

Etiology of tooth discoloration- a review

*Manuel ST, Abhishek P, Kundabala M

Department of Conservative Dentistry and Endodontics,
Manipal College of Dental Sciences, Manipal University,
Mangalore, Karnataka, India.

*Correspondence: Manuel ST

Email: manuel2dr@gmail.com

Abstract

Discoloration of the tooth can erode the sparkle from a smile. In the management of patients with discolored tooth, knowledge of the mechanisms behind tooth discoloration is of relevance as it can influence the treatment plan. In certain instances it may even have an effect on the outcome of the treatment. A grasp of the pathological process involved in tooth staining will also allow the dental practitioner to explain to the patient the exact nature of the condition. An overview of the etiology and the clinical appearance of tooth discoloration are discussed in this review.

Key words: Etiology, extrinsic discoloration, intrinsic discoloration

Introduction

Discoloration of the tooth is one of the most frequent reasons why a patient seeks dental care. Tooth discoloration is usually esthetically displeasing and psychologically traumatizing. An understanding of the etiology of tooth discoloration is important to a dentist in order to make the correct diagnosis. The knowledge of the cause of discoloration will also help the dental practitioner to explain the exact nature of the condition to the patient. In some instances, the mechanism of staining may have an effect on the outcome of treatment and influence the treatment options offered by the dentist to the patients⁽¹⁾.

Classification

The causes for tooth discoloration can be classified according to the location of the stains, either as extrinsic or intrinsic^(2, 3, 4). Extrinsic discoloration lies on the tooth surface or in the acquired pellicle. The intrinsic discoloration occurs when the chromogens are deposited within the bulk of the tooth, which maybe of local or systemic origin⁽⁵⁾.

Extrinsic discoloration

Extrinsic discoloration is defined as discoloration located on the outer surface of the tooth structure and is caused by topical or extrinsic agents⁽⁶⁾. This can be divided into two groups; direct and indirect. Direct staining is caused by compounds incorporated into the pellicle layer and the stain is a result of the basic color of the chromogen. Direct staining has multi-factorial etiology with the chromogens derived either from the diet or substances habitually placed in the mouth. Indirect staining on the other hand is caused by a chemical interaction at the tooth surface. It is usually associated with cationic antiseptics and metal salts. These agents are without color or a different color from the stain produced on the tooth surface⁽⁷⁾. Traditionally, extrinsic tooth discoloration can also be classified according to its origin, as metallic or non-metallic⁽⁸⁾.

Factors responsible for extrinsic discoloration (Table 1)

Diet: Brown stains on the surface of the teeth could be due to the deposition of tannins found in tea, coffee and other beverages⁽²⁾.

Oral hygiene: Accumulations of dental plaque, calculus and food particles cause brown or black stains (**Figure 1**). Chromogenic bacteria have also been suggested as an etiological factor in the production of stains typically at the gingival margin of the tooth⁽⁹⁾.

Habits: Tobacco from cigarettes, cigars, pipes, and chewing tobacco causes tenacious dark brown and black stains that cover the cervical one third to midway on the tooth⁽¹⁰⁾. Chewing of pan results in the production of blood red saliva that results in a red-black stain on the teeth, gingiva and oral mucosal surfaces^(11,12) (**Figure 2**).

Medication factors: Cationic antiseptics such as chlorhexidine, cetylpyridinium chloride and other mouth washes can cause staining after prolonged use^(3, 13). Chlorhexidine, for example, produces brown to black discoloration. Most evidence indicates that the likely cause of staining is the precipitation of anionic dietary chromogens onto the adsorbed cations^(14,15).

Some systemic medications (e.g. minocycline⁽¹⁶⁻¹⁸⁾, doxycycline^(19, 20), co-amoxiclav⁽²¹⁾, linezolid⁽²²⁾) are also shown to cause extrinsic staining. Metallic compounds are also implicated in dental discoloration (e.g. Iron containing oral solutions^(23, 24), mouth rinses containing metal salts^(25, 26)).

Occupation and environmental factors: Industrial exposure 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 and chromic acid fumes may cause deep orange stain⁽⁴⁾. There is a positive correlation between dental extrinsic stains and the concentration of trace elements, especially iron in the water sources⁽²⁷⁾ (**Figure 3**).

Intrinsic Discoloration:

There are several causes of intrinsic tooth discoloration which have either an endogenous or exogenous origin. These changes may occur during or after odontogenesis.

Table 1: Extrinsic causes of tooth discoloration

Classification	Factors responsible	Examples	Color	
Non-Metallic stains	Direct stains	Diet	Tea, coffee & other foods	Brown to black
		Oral hygiene	Dental plaque, calculus & Food particles	Yellow/brown
			Chromogenic bacteria	Brown/black/green Orange
	Indirect stains	Habits	Tobacco smoking/chewing	Dark brown/black
			Pan chewing	Red-black
		Medications	Cationic antiseptics e.g.: chlorhexidine	Yellow brown
Metallic stains	Medications	Essential oils/phenolic mouthrinse	Yellow	
		Sysgtemic antibiotics e.g Minocycline	Green-gray	
		Iron containing oral solutions	Black	
			Copper salt in mouth rinse	Green
	Potassium permanganate in mouth rinse		Violet to black	
	Occupation and environment	Stannous fluoride	Golden brown	
Silver nitrate		Gray		
Exposure to iron, manganese, silver		Black		
Exposure to mercury and lead dust		Blue green		
Copper & nickel		Green		
Chromic acid fumes	Deep orange			

During odontogenesis, teeth may become discolored from the changes in the quality or quantity of enamel or dentin, or from the incorporation of discoloring agent into the hard tissues. Post-eruption discoloration occur when the discoloring agent enters the hard tissues. They may originate from the pulp or the tooth surface⁽²⁸⁾ (**Tables 2 and 3**).

Pre-eruptive causes for intrinsic discoloration (**Table 2**)

Metabolic: The diseases that have the potential to cause neonatal hyperbilirubinemia may cause the incorporation of bilirubin into developing teeth, producing jaundice like yellow-green tint within the dental hard tissue known as chlorodontia⁽²⁹⁻³¹⁾. Congenital erythropoietin porphyria (Günther's disease) is a rare, autosomal recessive disorder

of porphyrin metabolism, resulting in an increase in the formation and excretion of porphyrins. The porphyrin pigments have an affinity for calcium phosphate and are incorporated into teeth during dental formation and these cause a characteristic reddish-brown discoloration of the teeth, called erythrodonia. The affected tooth shows a red fluorescence under ultra-violet light^(28, 32, 33). Alkaptonuria, also known as phenylketonuria or ochronosis is an inborn error of metabolism of tyrosin and phenylalanine causing a build-up of homogentisic acid. This results in a brown discoloration of the permanent dentition⁽³⁴⁾.

Disturbance during development of a tooth: Enamel hypoplasia may result due to the disturbance of the developing tooth germ following trauma, infection or nutritional deficiency giving rise to localized or

generalized enamel defects^(35, 36). Periapical odontogenic infections of the primary teeth can disrupt normal amelogenesis of the underlying permanent successors and can cause localized enamel hypoplasia. Trauma to developing, yet unerupted, teeth can also disturb amelogenesis and may result in enamel hypoplasia, which is visualized as a localized opacity on the erupted tooth. Such hypoplastic lesions are referred to as Turner's hypoplasia⁽³⁷⁾.

Crown formation begins in utero; therefore, the potential for extensive intrinsic discoloration of the primary dentition may be present throughout pregnancy. Although rare, maternal rubella or cytomegalovirus infection, maternal vitamin D deficiency, drug intake during pregnancy and toxemia of pregnancy can lead to tooth discoloration, which generally manifests as a focal opaque band of enamel hypoplasia. Systemic postnatal infections (e.g., measles, chicken pox, streptococcal infections, scarlet fever) can also cause enamel hypoplasia. The band like discoloration on the tooth are visualized where the enamel layer has variable thickness and becomes

extrinsically stained after tooth eruption. Vitamins C and D, calcium, and phosphate are required for healthy tooth formation. Deficiencies can result in exposure-related or dose-related enamel hypoplasia⁽³⁸⁾.

Molar-incisor hypomineralization is an idiopathic condition characterized by severe hypomineralized enamel affecting incisors and permanent first molars. The enamel defects can vary from white to yellow to brownish areas and they always show a sharp demarcation between sound and affected enamel. The possible etiologies for this condition include environmental changes, infections during the early childhood, dioxin in breast milk and genetic factors^(39,40).

Genetic defects and hereditary diseases: Genetic defects in enamel or dentin formation include amelogenesis imperfecta⁽⁴¹⁾ (Figure 4), dentinogenesis imperfecta and dentinal dysplasia^(42, 43). These hereditary diseases can be associated with intrinsic tooth discoloration. Defects in enamel formation may also occur in a number of systemically involved clinical syndromes such as Vitamin D dependent rickets⁽⁴⁴⁾, Epidermolysis bullosa⁽⁴⁵⁾, Ehlers-

Table 2: Pre-eruptive causes of intrinsic discoloration

Factor responsible	Examples	Color
Metabolic Disorders	Hyperbilirubinemia	Yellow - Green
	Prophyria	Reddish brown
	Alkaptonuria	Brown
Disturbance of tooth Germ	<u>Localized</u> Turner Tooth	White to Yellow to Brownish
	<u>Generalized</u> Infection (maternal or childhood) Nutritional deficiency Molar Incisor Hypomineralization	
Genetic Disorder	Amelogenesis imperfecta	Yellow Brown
	Amelogenesis imperfecta	Blue brown
	dentin dysplasia	Yellow
	Systemic syndrome e.g.: Epidermolysis bullosa	Yellow
Medication	Tetracycline	Yellow, brown, blue or grayish
	Minocycline	Blue - green
	Ciprofloxacin	Greenish
	Fluoride supplements	Chalky white to brown/ black
Environmental	Endemic fluorosis	Chalky white to brown/ black

Table 3: Post-eruptive causes of intrinsic discoloration

Factors responsible	Examples	Colour
Dental conditions	Dental caries	Chalky white Yellowish brown Dark brown to black
	- Incipient - Active - Arrested	
	Tooth wear	Yellowish
Pulpal causes	Ageing	Yellowish
	Pulpal trauma with hemorrhage	Gray-brown
	Calcific metamorphosis	Yellowish to yellowish brown
Dental materials	Internal resorption	Pinkish
	Amalgam	Blue-gray
	Composite/GIC	Yellowish brown
	Intra canal medicaments e.g. Iodoform, Ledermix	Brownish gray
	Obturing materials & sealers	Grayish

Danlos Syndrome⁽⁴⁶⁾ and pseudohypoparathyroidism⁽⁴⁷⁾.

Medications: Tetracycline, a broad spectrum antibiotic, is known to cause intrinsic discoloration when prescribed during tooth development. Tetracycline staining results from systemic administration of the drug, which chelates with the calcium ions on the surface of the hydroxyl apatite crystals as a stable orthophosphate complex. Teeth affected by tetracycline have a yellowish or brown-gray appearance which is worse on eruption and diminishes with time (Figure 5). The affected teeth also fluoresce under ultraviolet light, giving off a bright yellow color^(48, 49).

Minocycline is a semi-synthetic derivative of tetracycline. Its prolonged ingestion can lead to green-gray or blue-gray intrinsic staining of the teeth. Unlike with other tetracyclines, minocycline causes staining during and after the complete formation and eruption of teeth. Four theories have been proposed to explain the possible mechanism of this side effect. The first is the extrinsic theory, where it is thought that minocycline attaches to the glycoprotein in acquired pellicle. It oxidizes on exposure to air or as a result of bacterial activity. This results in the degradation of the aromatic ring forming insoluble black complex. It is possible that the pigment is incorporated into the dentin by a demineralization/remineralization phenomenon. The second is the intrinsic theory, where the minocycline bound to the plasma proteins is deposited in collagen-rich tissues, such as the teeth. This then oxidizes slowly over time with exposure to light. The third possibility is that the drug chelates with iron to form an insoluble complex. The fourth suggestion is that minocycline could be deposited in dentin during secondary dentinogenesis and the process can be accelerated in bruxists^(16-18, 50).



Figure 1: Extrinsic yellowish brown stains due to the accumulation of plaque and calculus



Figure 2: Extrinsic reddish black stains in a patient with the habit of chewing pan

Ciprofloxacin, a quinolone given intravenously to infants at dosages of 10 to 40 mg/kg/day to treat infections with *Klebsiella*, has been associated with greenish discoloration of the teeth^(13, 51).

Environmental causes: Dental fluorosis is the most common cause of intrinsic tooth discoloration⁽⁵²⁾. The most important risk factor for fluorosis is the total amount of fluoride consumed from all sources during the critical period of teeth development. A daily fluoride intake of more than the optimum of 0.05-0.07 mg fluoride/kg body weight/day is thought to cause dental fluorosis⁽⁵³⁾. The sources of fluoride include naturally or artificially fluoridated drinking water, commercially formulated beverages, and oral healthcare products (e.g. toothpastes, mouth rinses, oral fluoride supplements)⁽⁵⁴⁾. Dental fluorosis is characterized by symmetrical patterns of enamel discoloration resulting from sub-surface hypomineralization due to the ingestion of excessive amounts of fluoride during the early maturation stage of enamel formation. The severity depends on when and for how long the overexposure to fluoride occurs and the individual physiologic response. The clinical appearance may vary based on the severity from areas of enamel flecking to diffuse opaque mottling superimposed on chalky white or dark brown/black areas⁽⁵⁵⁾ (Figure 6).

Post-eruptive causes for intrinsic discoloration (Table 3)

Dental conditions and caries: Tooth wear is the progressive loss of enamel and dentin due to attrition, abrasion and erosion. As the enamel thins the tooth becomes darker as the color of the dentin becomes more apparent. Once the dentin is exposed the potential for chromogens to enter the body of the tooth increases⁽³⁾.

The various stages of carious process can be recognized by the change in color as the disease progresses. The pathogenesis of dental caries begins with an incipient lesion confined to the enamel layer. Incipient carious lesions are associated with plaque accumulation and manifest as chalky white areas of discoloration secondary to demineralization. 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⁽⁵⁶⁾ (Figure 7).

The natural darkening and the yellowing of the teeth and the change in their light transmission properties that occur with age can be due to the combination of the factors involving both the enamel and dentin. The enamel undergoes both thinning and textural change, while the deposition of secondary and tertiary dentin and pulp stones all contribute to the darkening process of ageing^(57, 58).

Pulpal Pathology

Bacterial, mechanical or chemical irritation to the pulp may result in tissue necrosis and the release of disintegration by-products that might penetrate the tubules and discolor the surrounding dentin. Acute trauma to an erupted tooth can cause intrapulpal hemorrhage giving it a reddish tinge. This discoloration can change to gray-brown in a matter of days as the pulp becomes necrotic (Figure 8). Hemolysis of the red blood cells would follow and release the heme group to combine with the putrefying pulp tissue to form



Figure 3: Extrinsic black stains due to high concentration of iron in the water source



Figure 4: Generalized yellowish discoloration with rough enamel surface in amelogenesis imperfecta



Figure 5: Brown-gray discoloration due to tetracycline.

black iron sulfide⁽⁵⁹⁾.

Excessive formation of irregular dentin in the pulp chamber and along the canal walls may occur following certain traumatic injuries. This is known as calcific metamorphosis. As a result of this, the translucency of the crown gradually decreases, giving rise to yellowish or yellowish brown discoloration^(60, 61).

Root resorption following trauma often presents as a pink spot lesion at the cemento-enamel junction in an otherwise



Figure 6: White opaque and brown discoloration as a result of fluorosis



Figure 7: Yellowish brown discoloration in a patient with rampant caries



Figure 8: Grayish brown discoloration in the upper left central incisor due to pulp hemorrhagic products

symptomless tooth, known as the 'Pink tooth of Mummy'. The resorption may be internal, being of pulp origin or external of periodontal origin^(62, 63).

Dental materials

Dental restorations most commonly cause intrinsic discoloration⁽⁶⁴⁾. Amalgam restorations can generate corrosion products, leaving a blue-gray color in the tooth, especially in large cavity preparations with undermined

enamel known as amalgam blue⁽⁶⁵⁾. Open margins around composite or glass ionomer restoration may allow chemicals to enter between the restoration and the tooth structure and discolor the underlying dentine.

Several intracanal medicaments are liable to cause internal staining of the dentin. Phenols or iodoform based medicaments sealed in the root canal are in direct contact with dentin, allowing penetration and oxidation. Tetracyclines (e.g. Ledermix- triamcinolone acetonide and demethylchlortetracycline) used within the tooth for endodontic therapy may also cause dark gray-brown discoloration^(66, 67). Incomplete removal of obturating material and sealer remnants in the pulp chamber, mainly those containing metallic components, often result in dark discoloration^(68, 69).

Diagnosis

History

The patient's history of tooth discoloration provides useful information regarding the etiology^(2, 70). The history should include the following:

- § Dental history (previous dental treatment, oral hygiene practices, use of mouthwashes, amount and scheduling of fluoride intake, history of dental trauma)
- § Medical history (history of maternal or childhood diseases, use of medications)
- § Family history (genetic disorders)
- § Diet history (nutritional deficiencies, diet that can cause staining of the teeth)
- § Social history (occupational exposure to metals, use of tobacco)

Clinical Examination

The scratch test is usually used to distinguish between extrinsic and intrinsic discoloration⁽⁷⁰⁾. Discolored tooth surfaces are scratched with care by using a dental explorer, scaler, or similar sharp instrument to assess surface texture. Light scratching with a dental instrument removes weakly adherent plaque that causes extrinsic discoloration. If the discoloration requires removal with a sharp dental scaler, the discoloration is considered to be tenacious. Intrinsic discoloration cannot be removed by using the scratch test.

Extrinsic staining of a single tooth is unusual. The distribution is usually generalized. The stains are usually found on surfaces with poor tooth brush accessibility. Whereas in case of intrinsic discoloration distribution is either generalized to all teeth or localized to certain teeth or tooth surfaces. An intrinsic etiology usually exists when a single tooth is discolored. Teeth with extrinsic tooth discoloration usually demonstrate no signs of pulp disease, usually associated with intrinsic discoloration.

Management

The treatment of tooth discoloration consists of identifying the etiology and implementing the required therapy⁽⁷¹⁾. Scaling and polishing of the teeth using prophylactic paste applied with a rotating rubber cup may remove many extrinsic stains. For more stubborn extrinsic and intrinsic stain, various bleaching techniques may be attempted⁽²⁾. Bleaching can be performed externally, termed night guard bleaching or vital tooth bleaching, or intracorally



in root-filled teeth, called non-vital tooth bleaching^(52, 72).

Teeth discolored by dental caries or dental materials require the removal of the caries or restorative materials, followed by proper restoration of the tooth. Partial (e.g. laminate veneers) or full-coverage dental restorations may be used to treat generalized intrinsic tooth discoloration in which bleaching is not indicated or in which the esthetic results of bleaching fail to meet the patient's expectations⁷³⁻⁷⁵.

Conclusion

In the management of patients with discolored tooth, an understanding of the mechanism behind the discoloration is of relevance to the dental practitioner as it can be valuable in the decision-making process when considering how to treat the condition. An understanding of the pathological process involved can assist in explaining the cause to anxious or concerned patients/ parents.

References

1. Watts A, Addy M. Tooth discolouration and staining: a review of the literature. *Br Dent J* 2001;190:309-316.
2. Hattab FN, Qudeimat MA, al-Rimawi HS. Dental discoloration: an overview. *J Esthet Dent* 1999; 11:291-310.
3. Sulieman M. An overview of tooth discoloration: extrinsic, intrinsic and internalized stains. *Dent Update*. 2005; 32:463-471.
4. Dayan D, Heifferman A, Gorski M, Begleiter A. Tooth discoloration: extrinsic and intrinsic factors. *Quintessence Int* 1983; 14:195-199.
5. Vogel RI. Intrinsic and extrinsic discoloration of the dentition. *J Oral Med* 1975; 30:99-104.
6. Eriksen HM, Nordbř H. Extrinsic discoloration of teeth. *J Clin Periodontol* 1978; 5: 229-236.
7. Natto SA. Chemistry and mechanism of extrinsic and intrinsic discoloration. *J Am Dent Assoc* 1997; 128 Suppl: 6S- 10S.
8. Eriksen HM, Jemtland B, Finckenhagen HJ, Gjermo P. Evaluation of extrinsic tooth discoloration. *Acta Odontol Scand* 1979; 37:371-375.
9. Carranza FA, Newnian MG. Dental calculus. In: Carranza FA Jr., ed. *Clinical periodontology*. 8th Ed. Philadelphia: WB Saunders, 1996.
10. Mirbod SM, Ahing SI. Tobacco-associated lesions of the oral cavity: Part I. Nonmalignant lesions. *J Can Dent Assoc* 2000; 66:252-256.
11. Reichart PA, Lenz H, König H, Becker J, Mohr U. The black layer on the teeth of betel chewers: a light microscopic, microradiographic and electronmicroscopic study. *J Oral Pathol* 1985; 14:466-475.
12. Norton SA. Betel: consumption and consequences. *J Am Acad Dermatol* 1998; 38:81-88.
13. Tredwin CJ, Scully C, Bagan-Sebastian JV. Drug-induced disorders of teeth. *J Dent Res* 2005; 84:596-602.
14. Eriksen HM, Nordbř H, Kantanen H, Ellingsen JE. Chemical plaque control and extrinsic tooth discoloration. A review of possible mechanisms. *Clin Periodontol* 1985; 12:345-350.
15. Solheim H, Roksvaag P, Eriksen HM, Nordbř H. Oral retention and discoloration tendency from chlorhexidine mouth rinse. *Acta Odontol Scand* 1983; 41:193-196.
16. Good ML, Hussey DL. Minocycline: stain devil? *Br J Dermatol* 2003; 149:237-239.
17. McKenna BE, Lamey PJ, Kennedy JG, Bateson J. Minocycline-induced staining of the adult permanent dentition: a review of the literature and report of a case. *Dent Update* 1999; 26:160-162.
18. Patel K, Cheshire D, Vance A. Oral and systemic effects of prolonged minocycline therapy. *Br Dent J* 1998; 185:560-562.
19. Ayaslioglu E, Erkek E, Oba AA, Cebecioglu E. Doxycycline-induced staining of permanent adult dentition. *Aust Dent J* 2005; 50:273-275.
20. Lochary ME, Lockhart PB, Williams WT Jr. Doxycycline and staining of permanent teeth. *Pediatr Infect Dis J* 1998; 17:429-431.
21. Garcia-López M, Martinez-Blanco M, Martinez-Mir I, Palop V. Amoxicillin-clavulanic acid-related tooth discoloration in children. *Pediatr* 2001; 108:819.
22. Matson KL, Miller SE. Tooth discoloration after treatment with linezolid. *Pharmacotherapy* 2003; 23:682-685.
23. Parkinson CF, Parkinson SB. Effect of vitamin-iron preparation on tooth staining studied. *J Mo Dent Assoc* 1981; 61:34-37.
24. Nordbř H, Eriksen HM, Röllä G, Attramadal A, Solheim H. Iron staining of the acquired enamel pellicle after exposure to tannic acid or chlorhexidine: preliminary report. *Scand J Dent Res* 1982; 90:117-123.
25. Waerhaug M, Gjermo P, Röllä G, Johansen JR. Comparison of the effect of chlorhexidine and CuSO₄ on plaque formation and development of gingivitis. *J Clin Periodontol* 1984; 11:176-180.
26. Ellingsen JE, Eriksen HM, Röllä G. Extrinsic dental stain caused by stannous fluoride. *Scand J Dent Res* 1982; 90:9-13.
27. Pushpanjali K, Khanal SS, Niraula SR. The relationship of dental extrinsic stains with the concentration of trace elements in water sources in a district of Nepal. *Oral Health Prev Dent* 2004; 2:33-37.
28. Hayes PA, Full C, Pinkham J. The etiology and treatment of intrinsic discolorations. *J Can Dent Assoc* 1986;52:217-220
29. Guimarães LP, Silva TA. Green teeth associated with cholestasis caused by sepsis: a case report and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003; 95:446-451.
30. Atasu M, Genc A, Erçalik S. Enamel hypoplasia and essential staining of teeth from erythroblastosis fetalis. *J Clin Pediatr Dent* 1998; 22:249-252.
31. Amaral TH, Guerra Cde S, Bombonato-Prado KF, Garcia de Paula E Silva FW, de Queiroz AM. Tooth pigmentation caused by bilirubin: a case report and histological evaluation. *Spec Care Dentist* 2008; 28:254-257.
32. Trodahl JN, Schwartz S, Gorlin RJ. The pigmentation of dental tissues in erythropoietic (congenital) porphyria. *J Oral Pathol* 1972; 1:159-171.
33. Fayle SA, Pollard MA. Congenital erythropoietic porphyria--oral manifestations and dental treatment in childhood: a case report. *Quintessence Int* 1994; 25:551-554.
34. Siekert RG, Gibilisco JA. Discoloration of the teeth in alkaptonuria (ochronosis) and parkinsonism. *Oral Surg Oral Med Oral Pathol* 1970; 29:197-199.

35. Pindborg JJ. Aetiology of developmental enamel defects not related to fluorosis. *Int Dent J* 1982; 32:123-134.
36. 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.
37. Bhushan BA, Garg S, Sharma D, Jain M. Esthetic and endosurgical management of Turner's hypoplasia; a sequelae of trauma to developing tooth germ. *J Indian Soc Pedod Prev Dent*. 2008; 26 Suppl 3:S121-S124.
38. Seow WK. Enamel hypoplasia in the primary dentition: a review. *ASDC J Dent Child* 1991; 58:441-452.
39. Weerheijm KL. Molar incisor hypomineralization (MIH): clinical presentation, aetiology and management. *Dent Update* 2004; 31:9-12.
40. Takahashi K, Correia Ade S, Cunha RF. Molar incisor hypomineralization. *J Clin Pediatr Dent* 2009; 33:193-197.
41. Crawford PJ, Aldred M, Bloch-Zupan A. Amelogenesis imperfecta. *Orphanet J Rare Dis* 2007; 2:17.
42. Barron MJ, McDonnell ST, Mackie I, Dixon MJ. Hereditary dentine disorders: dentinogenesis imperfecta and dentine dysplasia. *Orphanet J Rare Dis* 2008; 3:31.
43. Kim JW, Simmer JP. Hereditary dentin defects. *J Dent Res* 2007; 86:392-399.
44. Zambrano M, Nikitakis NG, Sanchez-Quevedo MC, Sauk JJ, Sedano H, Rivera H. Oral and dental manifestations of vitamin D-dependent rickets type I: report of a pediatric case. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003; 95:705-709.
45. Brooks JK, Bare LC, Davidson J, Taylor LS, Wright JT. Junctional epidermolysis bullosa associated with hypoplastic enamel and pervasive failure of tooth eruption: Oral rehabilitation with use of an overdenture. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2008; 105:e24-28.
46. Klingberg G, Hagberg C, Norén JG, Nietzsche S. Aspects on dental hard tissues in primary teeth from patients with Ehlers-Danlos syndrome. *Int J Paediatr Dent* 2009; 19:282-290.
47. Lagarde A, Kerebel LM, Kerebel B. Structural and ultrastructural study of the teeth in a suspected case of pseudohypoparathyroidism. *J Biol Buccale* 1989; 17:109-114.
48. van der Bijl P, Pitigoi-Aron G. Tetracyclines and calcified tissues. *Ann Dent* 1995; 54:69-72.
49. Sánchez AR, Rogers RS 3rd, Sheridan PJ. Tetracycline and other tetracycline-derivative staining of the teeth and oral cavity. *Int J Dermatol* 2004; 43: 709-715.
50. McKenna BE, Lamey PJ, Kennedy JG, Bateson J. Minocycline-induced staining of the adult permanent dentition: a review of the literature and report of a case. *Dent Update* 1999; 26:160-162.
51. Lumbiganon P, Pengsaa K, Sookpranee T. Ciprofloxacin in neonates and its possible adverse effect on the teeth. *Pediatr Infect Dis J* 1991; 10:619-620.
52. Plotino G, Buono L, Grande NM, Pameijer CH, Somma F. Nonvital tooth bleaching: a review of the literature and clinical procedures. *J Endod* 2008; 34: 394-407.
53. Burt BA. The changing patterns of systemic fluoride intake. *J Dent Res* 1992; 71: 1228-1237.
54. Levy SM. An update on fluorides and fluorosis. *J Can Dent Assoc* 2003; 69:286-291.
55. Alvarez JA, Rezende KM, Marocho SM, Alves FB, Celiberti P, Ciamponi AL. Dental fluorosis: exposure, prevention and management. *Med Oral Patol Oral Cir Bucal* 2009; 14:E103-107.
56. Kleter GA. Discoloration of dental carious lesions. *Arch Oral Biol* 1998; 43:629-632.
57. Morley J. The esthetics of anterior tooth aging. *Curr Opin Cosmet Dent* 1997; 4:35-39.
58. Odioso LL, Reno EA. The impact of age on tooth colour. *J Ir Dent Assoc* 2001; 47:144-145.
59. Marin PD, Bartold PM, Heithersay GS. Tooth discoloration by blood: an in vitro histochemical study. *Endodent Traumatol* 1997; 13:132-138.
60. Gopikrishna V, Parneswaran A, Kandaswamy D. Criteria for management of calcific metamorphosis: review with a case report. *Indian J Dent Res* 2004; 15:54-57.
61. Amir FA, Gutmann JL, Witherspoon DE. Calcific metamorphosis: a challenge in endodontic diagnosis and treatment. *Quintessence Int* 2001; 32:447-455.
62. Silveira FF, Nunes E, Soares JA, Ferreira CL, Rotstein I. Double 'pink tooth' associated with extensive internal root resorption after orthodontic treatment: a case report. *Dent Traumatol* 2009; 25:e43-47.
63. Hiremath H, Yakub SS, Metgud S, Bhagwat SV, Kulkarni S. Invasive cervical resorption: a case report. *J Endod* 2007; 33:999-1003.
64. van der Burgt TP, Plasschaert AJ. Tooth discoloration induced by dental materials. *Oral Surg Oral Med Oral Pathol* 1985; 60:666-669.
65. Scholtanus JD, Ozcan M, Huysmans MC. Penetration of amalgam constituents into dentine. *J Dent* 2009; 37:366-373.
66. Kim ST, Abbott P. The effects of Ledermix paste as an intracanal medicament on the discolouration of teeth. *Aust Endod J* 2000; 26:86-87.
67. Kim ST, Abbott PV, McGinley P. The effects of Ledermix paste on discolouration of mature teeth. *Int Endod J* 2000; 33:227-232.
68. Partovi M, Al-Havvaz AH, Soleimani B. In vitro computer analysis of crown discolouration from commonly used endodontic sealers. *Aust Endod J* 2006; 32:116-119.
69. Parsons JR, Walton RE, Ricks-Williamson L. In vitro longitudinal assessment of coronal discoloration from endodontic sealers. *J Endod* 2001; 27:699-702.
70. Kerr AR (2008). Retrieved online Jan 9, 2009 from <http://emedicine.medscape.com/article/1076389-overview>.
71. Setien VJ, Roshan S, Nelson PW. Clinical management of discolored teeth. *Gen Dent* 2008; 56:294-300.
72. Sulieman MA. An overview of tooth-bleaching techniques: chemistry, safety and efficacy. *Periodontol* 2000 2008; 48:148-169.
73. Cutbirth ST. Indirect porcelain veneer technique for restoring intrinsically stained teeth. *J Esthet Dent* 1992; 4:190-196.
74. Crispin BJ. The full veneer as an alternative to the full crown. *Curr Opin Cosmet Dent* 1997; 4:6-10.
75. Walls AW, Steele JG, Wassell RW. Crowns and other extra-coronal restorations: porcelain laminate veneers. *Br Dent J* 2002; 193:73-76, 79-82.